CARDIOVASCULAR DISEASE RISK IN BLACK AND CAUCASIAN FEMALES: AN EASTERN CAPE SAMPLE

BY

SANDRA CLAUDIA REMSING

THESIS

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Department of Human Kinetics and Ergonomics

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ABSTRACT

The purpose of this study was to investigate cardiovascular disease risk in Black and Caucasian urban working females of the Makana region of the Eastern Cape. One-hundred and sixty six age-matched urban-working individuals (Black n = 88; Caucasian n = 78) voluntarily participated in this cross-sectional study. Cardiovascular disease risk was assessed in four categories: 1) obesity, classified as 'morphological risk', 2) blood pressure, classified as 'cardiovascular risk', 3) diet, physical activity, smoking, and alcohol consumption, classified as 'lifestyle risk', and 4) education and income, classified as 'socioeconomic factors'. Results showed that Black females were significantly *($p<0.01$)* heavier than Caucasian females and categorized as 'obese' according to BMI classifications (31.19 ± 8.09 kg.m^2 and 25.90 ±4.67 kg.m^2, respectively). Black females also presented with significantly *($p<0.01$)* higher waist circumferences and waist-to-stature ratios, further confirming the obesity prevalence in this group. When accounting for those who were on anti-hypertensive mediation, Black females additionally presented with significantly *($p<0.05$)* higher blood pressure, categorizing them as 'pre-hypertensive' (MAP = 98 ± 16 mmHg and 85 ± 11 mmHg, respectively). These results therefore placed Black females at increased cardiovascular disease risk compared to Caucasian females. With respect to lifestyle factors, Black females consumed significantly *($p<0.05$)* more kilojoules carbohydrates, and total sugar than Caucasian females, and expended more physical activity MET-minutes per week (2688.86 ± 1486.56 MET-minutes and 1828.27 ± 2238.10 MET-minutes, respectively). However, reduced validity of physical activity data as well as poor dietary recall limited the interpretations of these findings. In addition, despite significant differences between smoking and alcohol consumption between groups, the majority of both Black and Caucasian females reported being non-smokers and consumed alcohol minimally. Thus, these were concluded to be unlikely contributors to cardiovascular disease risk in these samples. Socioeconomic results nevertheless expectedly showed that Black females were significantly *($p<0.05$)* less educated, and subsequently earned lower incomes. It was thus concluded that this may be a likely contributor to higher cardiovascular risk in Black females. Future recommendations include the incorporation of exercise intensity measures as well as objective measures of habitual diet. This is in order to get a clearer understanding of the impact of these lifestyle factors on cardiovascular disease risk.

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CHAPTER I – INTRODUCTION

1.1 BACKGROUND

South Africa is comprised of a vast mix of people and cultures including not only a number of different racial groups, but also large socioeconomic disparities (Steyn *et al.,* 2006). This ranges from those few at the top end of the socio-economic scale, to the majority of South Africans who live below the poverty line (evidenced by a Gini coefficient of 63.4 in 2015, which is the highest among the BRICS countries) (The World Bank, 2016). As all of the BRICS countries (Brazil, Russia, India, China, and South Africa) are grouped together as countries in a similar stage of newly advanced economic development, the fact that the economic disparity is highest within South Africa highlights the unique context of the country, and emphasizes the need for individualized attention (The World Bank, 2016). Due to these unique set of circumstances, South Africans face a broad spectrum of diseases, comprising of communicable (infectious) diseases; non-communicable (lifestyle-related) diseases; and HIV/AIDS (Bradshaw & Steyn, 2001; Econoex, 2009). In addition, due to the high violent crime rate, South Africans are also vulnerable to violence-related injuries and trauma, which places them at an even greater health risk (Bradshaw *et al.,* 2006). Due to the combination of these four factors, the country suffers from what is termed the 'quadruple burden of disease' (Econex, 2009; Moodley *et al.,* 2013). Research has shown that the heaviest burden of disease falls on poor communities in urban areas, and this is often due to poor access to state health care and education (Mayosi *et al.,* 2009; Walter and Durandt, 2011). As the majority of those people living in these poor urban areas are black South Africans (Mayosi *et al.,* 2009), it is therefore black South Africans who bear the brunt of the quadruple burden of disease in the country (Mayosi *et al.,* 2009).

Historically, non-communicable diseases received little attention in developing countries; these diseases predominantly affected white urban males and were typically associated with 'affluence' characterized by sedentary lifestyles and poor eating habits (Ezzati *et al.,* 2005; Taubes, 2008). Evidence from almost fourty years ago indeed shows that white males were one of the most, if not the most, obese and at risk population for non-communicable diseases in the world (Ezzati *et al.,* 2005). However,

the health status of the world, and particularly in the developing countries within Africa, is rapidly changing (WHO, 2016).

Within most African countries, non-communicable diseases have emerged to become the second highest cause of death (Econex, 2009). This is second to HIV/AIDS which remains the primary cause of death in most African countries (WHO, 2007; WHO, 2011). Nevertheless, non-communicable diseases have risen rapidly in Africa.

Within South Africa, more people died due to non-communicable diseases (specifically cardiovascular disease) than due to HIV/AIDS or other infectious diseases in 2014, and the vast majority of these were Black South Africans (Econex, 2009; Zatu *et al.,* 2015). More specifically, Black South African women have emerged as the most at risk population for cardiovascular disease in the country, comprising of more than half of the cardiovascular disease deaths in South Africa in 2014 (Goedecke *et al.,* 2009). This recent and rapid change in the South African disease profile highlights the urgency with which cardiovascular disease risk requires attention, particularly in Black female South Africans.

A proposed explanation for the emergence of cardiovascular disease lies in the impact of rapid urbanization and industrialisation, which is initiated by the geographical transition from more traditional rural environments, to urban environments (Steyn *et al.,* 2006). What accompanies this geographical transition is an epidemiological and nutritional transition characterized by a change in cultural lifestyle behaviours such as changes in dietary and physical activity habits (Steyn *et al.,* 2006; Walter and Durandt, 2011). Some of these changes are attributed to inherent aspects of urbanized living such as readily accessible fast-food and public transport, making it easier for people to make poorer dietary choices and reducing their physical activity levels as they are using more public transport (Steyn *et al.,* 2006). However, many of these behavioural changes are also due to social factors such as the perceived 'higher status' associated with being able to afford fast-food, as well as the stigma towards being 'slim' in many African cultures, which is often associated with 'disease' or 'weakness' (Walter and Durandt, 2011). Subsequently, being overweight or obese is considered more socially acceptable, thus typically contributing to the growing rates of obesity and cardiovascular disease (Walter and Durandt, 2011). It therefore becomes important

to assess both lifestyle changes and socio-cultural practises and norms when assessing the health status of any population.

Nevertheless, some pressing questions still persists: what is causing and accelerating the incidence of cardiovascular disease in Black South African women? And more specifically, does cardiovascular disease affect Xhosa women of the Eastern Cape in a similar way to other racial groups? Many studies globally have looked at cardiovascular risk factors in Caucasian populations of all countries, but only a few have looked at Black South African populations (Alberti *et al.,* 2005). This is evidenced by the fact that population-specific cardiovascular risk factor 'cut-off' norms exist for Caucasian populations, but not for Black South Africans (Alberti *et al.,* 2005). Of those studies who have looked at Black South Africans, the majority of them have been conducted in the North West Province, the Western Cape, and Gauteng (Vorster *et al.,* 2002; Senekal *et al.,* 2003; Jennings *et al.,* 2008; Goedecke *et al.,* 2009), and these studies compared their findings to Caucasian cut-off norms. Not only does this question their validity, but it also highlights the large gap in the literature for the other provinces, including the Eastern Cape. It is useful to assess cardiovascular disease risk in other provinces in order to gain a deeper understanding of cardiovascular health in South Africa. In addition, gaining data that is representative of the entire country is beneficial in order to assess the relevance and reliability of 'cut-off' norms. Furthermore, there is a plethora of health-related research conducted on Caucasian women, and Caucasian women represent another urban population in South Africa. Therefore they represent an appropriate comparison group to Black urban South African women for any research into cardiovascular disease risk in urban female populations.

1.2 STATEMENT OF THE PROBLEM

As cardiovascular disease is rapidly increasing in South Africa, knowledge of the populations' disease risk profiles is required. Due to poverty and recent urbanisation, Black South African populations are vulnerable to developing cardiovascular disease. In particular, Black South African women have shown increased risk of cardiovascular disease in recent years. To date however, there is a paucity in the literature assessing cardiovascular disease risk in individuals residing in the Eastern Cape; one of the poorest and most poverty-stricken provinces in the country. In addition, most of the

research worldwide is conducted on Caucasian populations. Accordingly, the aim of this research is twofold: the first aim is to add new data to the body of literature on cardiovascular disease risk in black females within the Eastern Cape. Secondly, this research aims to assess and compare cardiovascular disease risk in urban black women and Caucasian women, thereby exploring the interaction between race, culture, and lifestyle factors, and their impact on cardiovascular disease risk.

1.3 RESEARCH HYPOTHESES

It is expected that cardiovascular disease risk will be different between Black females and Caucasian females. More specifically, it is expected that a higher prevalence of cardiovascular risk factors will be present in Black females compared to Caucasian females.

1.4 STATISTICAL HYPOTHESES

Null hypothesis 1:

There will be no difference in morphological characteristics between the two samples, as defined by a) Mass, b) BMI, c) waist circumference, d) waist-to-stature ratio e) body composition.

H_0: $\mu Black_{MORPH\ (a,b,c,d,\ e)} = \mu Caucasians_{MORPH\ (a,b,c,d,\ e)}$

H_a: $\mu Black_{MORPH\ (a,b,c,d,\ e)} \neq \mu Caucasians_{MORPH\ (a,b,c,d,\ e)}$

Null hypothesis 2:

There will be no difference in cardiovascular risk parameters (CV) between the two samples, as defined by f) blood pressure, g) fasting glucose levels, h) fasting insulin levles

H0: $\mu Black_{CV\ (f,g,h)} = \mu Caucasians_{CV\ (f,g,h)}$

Ha: $\mu Black_{CV\ (f,g,h)} \neq \mu Caucasians_{CV\ (f,g,h)}$

Null hypothesis 3:

The lifestyle-related factors will be similar between the two samples, as defined by i) dietary intake, j) physical activity levels, k) tobacco use, and l) alcohol consumption

H_0: $\mu Black_{LIFESTYLE\ (i,j,k,l)} = \mu Caucasians_{LIFESTYLE\ (i,j,k,l)}$

Ha: $\mu Black_{LIFESTYLE\ (i,j,k,l)} \neq \mu Caucasians_{LIFESTYLE\ (i,j,k,l)}$

Null hypothesis 4:

There will be no difference in the socio-economic status of the two samples, as defined by m) annual income, and n) education level

H_0: $\mu Black_{SOCIO\text{-}ECONOMIC\ STATUS\ (m,n)} = \mu Caucasians_{SOCIO\text{-}ECONOMIC\ STATUS\ (m,n)}$

Ha: $\mu Black_{SOCIO\text{-}ECONOMIC\ STATUS\ (m,n)} \neq \mu Caucasians_{SOCIO\text{-}ECONOMIC\ STATUS\ (m,n)}$

CHAPTER II – LITERATURE REVIEW

2.1 GLOBAL HEALTH PROFILE

Non-communicable diseases, also known as chronic diseases, are the leading cause of death worldwide (WHO, 2011). Twenty nine million deaths alone were attributed to non-communicable diseases in 2002 (Econex, 2009; Yach *et al.,* 2011) and they currently account for over 60% of deaths worldwide (WHO, 2011). Despite initially being associated with high income countries, the prevalence of non-communicable diseases is increasing rapidly in developing countries. This increase is to such an extent that currently, 80% of global chronic disease deaths occur in low and middle income countries (WHO, 2011). This is of particular relevance to a South African context as South Africa is classified as a middle income, or developing country.

There are four main diseases that are listed as non-communicable: cardiovascular disease, certain cancers, chronic obstructive respiratory disease, and type II diabetes, and typically occur as a result of lifestyle factors (Beaglehole *et al.,* 2011). These factors include physical inactivity, excessive alcohol and tobacco consumption, and unhealthy eating habits (WHO, 2011). In most cases, it is a combination of these lifestyle factors over a prolonged period of time that contribute to the development of these conditions (Crush *et al.,* 2011). Unlike communicable diseases, non-communicable diseases cannot be passed from one person to another- highlighting the profound impact that individual lifestyle factors have on the aetiology of these diseases (Yach *et al.,* 2011). Nevertheless, the risk of developing a non-communicable disease is amplified by certain non-modifiable risk factors such as age, sex, race, and family history. These factors will be discussed in detail at a later point in this chapter.

Cardiovascular disease led to the greatest number of deaths worldwide in 2002, followed by cancer, chronic obstructive respiratory disease, and type II diabetes (WHO, 2011). Since then, prevalence of all above listed diseases has increased, with a particularly large increase in prevalence of type II diabetes from 2% of the global population in 2002, to an estimated 9% in 2009 (Yach *et al.,* 2004; Bertram *et al.,* 2013). Although non-communicable diseases currently lead to 60% of deaths worldwide, it is predicted that if the present trend is maintained, non-communicable

diseases will account for 80% of the global burden of disease in 2020 (Boutayeb, 2006). It is therefore urgent and imperative to explore and develop efficient preventative strategies to halt the growing trend of non-communicable diseases through the control of risk factors. Specifically, this needs to be aimed at adjusting lifestyle behaviours which impact risk conditions, and thus affects overall health. This focus area will be discussed in greater detail at a later point in this chapter.

2.1.1 WHAT IS CARDIOVASCULAR DISEASE?

Cardiovascular disease is a general term that describes diseases of the heart or blood vessels (Naranjan *et al.,* 2012). More specifically, cardiovascular disease occurs when blood flow to the heart, brain, or rest of the body is reduced due to a blood clot (thrombosis), or a hardening and narrowing of the arteries (atherosclerosis) (Libby *et al.,* 2009). Four main types of cardiovascular disease exist and they are: coronary heart disease, aortic disease, stroke, and peripheral arterial disease (Libby and Theroux, 2005; Coffman and Eberhardt, 2003; Libby *et al.,* 2009; Naranjan *et al.,* 2012).

2.1.1.1 Coronary Heart disease and Aortic disease

Coronary heart disease and occurs when the flow of oxygen-rich blood to the heart is blocked or reduced by a build-up of plaque (cholesterol) in the coronary arteries (Libby & Theroux, 2005). As the coronary arteries are the two major blood vessels that supply the heart with blood, a narrowing of these arteries therefore restricts this blood flow substantially. If the arteries are partially blocked or hardened due to the plaque, this can lead to mild chest pain (angina) (Libby & Theroux, 2005). However, if the arteries become completely blocked however, the heart becomes starved of oxygen and an acute myocardial infarction (heart attack) occurs (Libby and Theroux, 2005). Coronary heart disease is the most prevalent form of cardiovascular disease and accounts for 25% of global deaths annually (WHO, 2011). A less common form of heart disease, aortic disease, occurs through the same mechanism as coronary heart disease only that the aorta is affected instead of the coronary arteries (Naranjan *et al.,* 2012).

2.1.1.2 Stroke and peripheral arterial disease

A stroke can occur when blood supply to the brain is obstructed and reduced (Wolf *et al.,* 1991). This deprives the brain of oxygen and nutrients, and subsequently causes brain cells to die. In 80% of cases, strokes are caused by a build-up of plaque which blocks the artery (ischemic stroke) while the remaining 20% occur as a result of leaking or bursting of a blood vessel (haemorrhagic stroke) (Wolf *et al.,* 1991). Peripheral arterial disease occurs through the same mechanics as a stroke, only that blood supply is obstructed to arteries in the limbs, usually the legs (Coffman & Eberhardt, 2003). As with a stroke, if the artery is completely blocked, nerve and muscles cells of the affected limb begin to die (Coffman & Eberhardt, 2003).

2.2 SOUTH AFRICAN HEALTH PROFILE

The South African health profile is unique as its people suffer a 'quadruple burden of disease' comprising acquired immune deficiency syndrome (AIDS), non-communicable diseases, communicable diseases, and death due to injury and violence (Bourne *et al.,* 2002; Bradshaw *et al.,* 2003; Econex, 2009; Tathiah *et al.,* 2013). Although it is common for developing countries to experience a double burden of disease resulting from the simultaneous occurrence of communicable diseases and non-communicable diseases, South Africa has the added burden of HIV/AIDS and injury due to violence (Econex, 2009). This places an increased demand on the already under-resourced public health services which are accessed by 80% of the population (Bradshaw *et al.,* 2005).

Despite the high prevalence of HIV/AIDS in South Africa, in 2014, more premature deaths resulted directly from non-communicable diseases than from HIV/AIDS (Figure 1) (WHO, 2016). In accordance with global trends, cardiovascular disease led to the greatest number of deaths in South Africa in 2014, and the majority of these were Black South Africans (Zatu *et al.,* 2014; WHO, 2016).

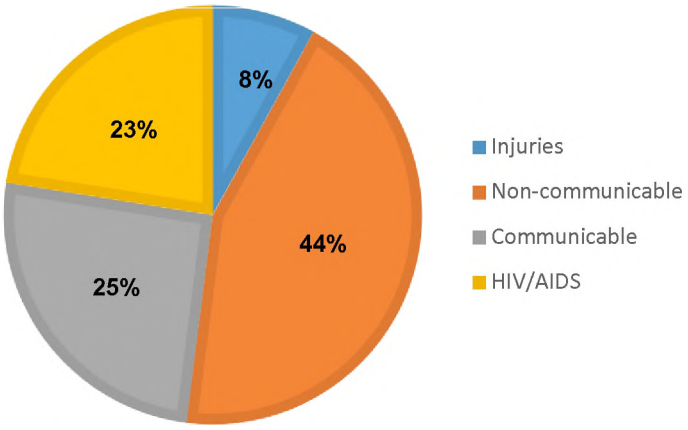

Figure 1 Estimated causes of death in South Africa, 2014 (WHO, 2016).

2.2.1 SOCIOECONOMIC STATUS AND CARDIOVASCULAR DISEASE RISK

Despite the dissolution of the Apartheid regime in 1994, the effects thereof are still echoed throughout South Africa (Talip *et al.,* 2003; Steyn *et al.,* 2005). This is evidenced by pervasive socioeconomic disparities- particularly affecting Black populations who are typically poorer and less educated than all other race groups (Bradshaw *et al.,* 2000). Subsequently, it is these poor Black populations that bear the brunt of the quadruple burden of disease (Bradshaw *et al.,* 2000; Vorster *et al.,* 2002).

Perhaps more pertinent, however, is the impact of socioeconomic status on cardiovascular disease risk in South Africa. There is a strong body of evidence highlighting the link between low socioeconomic status and increased cardiovascular disease risk, and this is attributed to three main factors: poor education, lack of access to sufficient healthcare, and *socioeconomic stress* (DHS, 2003; Steptoe *et al.,* 2003; Brydon *et al.,* 2006; Clark *et al.,* 2009).

Lack of education contributes to high cardiovascular disease risk through limited exposure to the dangers of unhealthy eating, lack of physical activity, smoking, and alcohol use from a young age[1] (DHS, 2003; WHO, 2016). This is supported by a vast body of research highlighting the protective effect of adequate education on the development of chronic diseases (DHS, 2003; Senekal *et al.,* 2003; Ehrlich *et al.,* 2004; Ehrlich *et al.,* 2005; Kruger *et al.,* 2005). This is of particular importance in a

[1] The impact of these lifestyle factors will be discussed in more detail later in this chapter, under 'modifiable risk factors for cardiovascular disease', pp. x.

South African context which is currently undergoing a nationwide 'education crisis'- invariably contributing to the high incidences of cardiovascular disease (Fleisch, 2008; Spaull, 2013).

In addition to poor education, those of low socioeconomic status are reliant on public healthcare; a vastly under-resourced and heavily burdened sector in South Africa, resulting in the lack adequate healthcare for the majority of poor populations (Bradshaw *et al.,* 2007). These health services are required to cater to a plethora of diseases, including infectious diseases, violence and trauma-related injuries, and HIV/AIDS- all of which are highly prevalent countrywide (Woolard, 2002). Thus, resource allocation is prioritized for these more immediate and urgent conditions, further limiting the care available to those with cardiovascular disease (Woolard, 2002). This therefore highlights the difficulties associated with relying on health statistics in South Africa, particularly where clinical care and subsequent diagnoses are limited.

Socioeconomic stress refers to the concomitant stress that occurs as a result low socioeconomic status (Steptoe *et al.,* 2003). This stress is attributed to poor living conditions, increased financial strain, as well as the above mentioned difficulties associated with lack of quality education and access to healthcare (Steptoe *et al.,* 2003). A plethora of research highlights the impact of socioeconomic stress on cardiovascular disease risk, further highlighting the association between low socioeconomic status and cardiovascular disease risk (Steptoe *et al.,* 2003; Brydon *et al.,* 2006; Clark *et al.,* 2009).

Therefore, in addition to both non-modifiable and modifiable risk factors which will discussed in detail below, it is imperative to also address socioeconomic status when assessing cardiovascular disease risk in certain populations.

2.3 NON-MODIFIABLE RISK FACTORS FOR CARDIOVASCULAR DISEASE

There are a number of non-modifiable risk factors that are associated with increased risk of developing cardiovascular disease. These include race, family history, age, and sex (DHS, 2003; Fodor & Tzerovska, 2004; WHO, 2007).

2.3.1 RACE AND FAMILY HISTORY

Globally, it is well documented that disproportionately higher rates of cardiovascular disease are evident in Black populations compared to Caucasian counterparts (Kurian and Cardarelli, 1995; WHO, 2007; Gebreab *et al.,* 2012). In addition, Hispanics and Native American Indians are also considered to be at greater risk than Caucasians (WHO, 2007). In 2009, overall death rate due to cardiovascular disease in the USA was 236.1 per 1000, however when looking at just African Americans, it was 387.0 for males and 267.9 for females (American Heart Association, 2016). Despite this statistic, it remains unclear whether this difference in prevalence is due to race alone, or to a combination of race and cultural specific lifestyle factors. This is an important consideration as lifestyle factors are considered as 'modifiable', therefore there needs to be a clear distinction between racial (genetic) and habitual factors (lifestyle) that contribute to cardiovascular disease risk. For example, 65% of Indian people living in India rely on rice as their staple food (WHO, 2011). This is due in part to availability, but also largely to cultural dietary practices that incorporate rice into almost every dish (WHO, 2011). If this is then considered in conjunction with the high prevalence of type II diabetes in Indian populations, it becomes premature to associate the disease prevalence purely with racial factors (genetics), while not considering the unique dietary practises (lifestyle) as a potential contributor. To the author's knowledge, no studies have attempted to unpack this relationship, and thus the interaction between race and cultural practices is largely underreported. Therefore in order to accept that certain races are more likely to develop cardiovascular disease than others, culturally specific lifestyle factors need to be assessed first.

Research has however demonstrated that certain cardiovascular disease risk factors have a genetic predisposition (Fodor and Tzerovska, 2004). In other words, if a genetic predisposition is inherited, cardiovascular disease is more likely to develop regardless of dietary habits and other lifestyle factors (Fodor and Tzerovska, 2004). In particular, this is evident with hypertension and hypercholesterolemia that runs in families (WHO, 2011). Despite genetic predispositions however, lifestyle factors can still reduce or exacerbate overall risk, and thus shouldn't be ignored (WHO, 2011).

In South Africa however, the prevalence of cardiovascular disease among different races is less clear. On one end, it has been suggested that Black South African males

and females are at less risk for developing cardiovascular disease than their Caucasian counterparts (Vorster *et al.,* 2005; Jennings *et al.,* 2008). One study highlighted that Black South Africans had lower levels of the hormone homocysteine compared to Caucasians, and this exerted a protective effect as high levels of homocysteine are associated with cardiovascular disease risk (Vorster *et al.,* 2005). Because baseline homocysteine levels are genetically determined, it was concluded that Black South Africans may have a genetic advantage over other populations with respect to cardiovascular disease risk (Vorster *et al.,* 2005). However, contradictory evidence states that the highest prevalence of stroke mortalities is evident among Black South Africans, in addition to Black South African women being the most obese population in the country (Tibazarwa *et al.,* 2009; Kandala *et al.,* 2014; Goedecke *et al.,* 2015). As a result, this evidence suggests that Black South Africans are at the highest risk for certain types of cardiovascular disease (Cappuccio, 1997). It is therefore imperative that more research on the general health of Black South African populations is conducted in order to gain deeper insight into the confounding findings to date.

Nevertheless, despite uncertainties regarding racial prevalence of disease risk, the health status of South Africa is changing (Bourne *et al.,* 2002; Econex, 2009). Due to rapid urbanization and the subsequent accessibility of commodities such as fast food, more Black South Africans are presenting with cardiovascular disease today than before (Bradshaw *et al.,* 2007; Econex, 2009; WHO, 2016). Additionally, insulin resistance and type II diabetes rates in South Africa have nearly doubled from 5.5% in 2000 to 9% in 2009, which highlights the ever increasing prevalence of chronic disease, which is reason for concern (WHO, 2011). Therefore, despite suggested genetic benefits, many Black South Africans are living with chronic diseases, and this places the impetus on exploring lifestyle factors for effective management and prevention.

2.3.2 AGE AND SEX

It is widely accepted that risk for cardiovascular disease increases with age (Dubnov *et al.,* 2003; Bray and Champagne, 2005). In particular, males over the age of 45 and females over the age of 55 are at high risk, with post-menopausal females posing the greatest risk (Carr, 2003). In females, it is suggested that the increase in risk after 55

years is associated with menopause which is characterized by the reduction in oestrogen concentrations and subsequent cessation of menses (Dubnov *et al.,* 2003). In normal concentrations, oestrogen plays a cardio-protective role in the body by promoting the growth of healthy vascular endothelium cells in blood vessels, as well as assisting in vasodilation and fluid regulation (Dubnov *et al.,* 2003). Additionally, the effects of reduced oestrogen concentrations result in redistribution of subcutaneous fat to the visceral area. As oestrogen levels drop, testosterone levels rise, which promotes central storage of fat. This places women at an even greater risk as visceral adiposity is one of the greatest risk factors for cardiovascular disease (Reckelhoff, 2001; Carr, 2003; Dubnov *et al.,* 2003). Therefore females in their productive years are largely protected from developing cardiovascular disease by this mechanism, whereas post-menopausal females whose oestrogen levels drop, are not (Wellman *et al.,* 1996). It is due to these risk factors that many women are prescribed exogenous hormone replacement therapy (HRT) in attempt to counteract the natural decline in oestrogen concentrations at menopause (Dubnov *et al.,* 2003).

For males, cardiovascular disease risk is attributed to firstly the absence of the cardio-protective effects of oestrogen, and secondly, to greater visceral adiposity (Dubnov *et al.,* 2003). As the male sex hormone testosterone promotes the storage of fat centrally, men < 45 years are at greater risk compared to females of the same age, as central adiposity is an independent risk factor the development of cardiovascular diseases (Reckelhoff, 2001). This explanation is supported by the fact that by the time females reach menopause, their risk of cardiovascular disease increases dramatically as their fat distribution becomes more centrally located (Reckelhoff, 2001; Dubnov *et al.,* 2003). It is however less clear why risk increases after the age of 45 years in males. The National Heart, Lung, and Blood Institute (2016) postulate that this is simply due to the ageing effect; however there is still a paucity in literature reporting on this.

2.4 MODIFIABLE RISK FACTORS FOR CARDIOVASCULAR DISEASE

As with the non-modifiable risk factors, there are also a number of modifiable-risk factors for cardiovascular disease. In order to effectively explore and develop management and preventative strategies, modifiable risk factors require particular focus as they are the ones that can be changed.

Apart from being modifiable, these factors also differ from non-modifiable risk factors as they consist of both modifiable 'conditions' as well as modifiable 'behaviours'. The conditions include hypertension, central obesity, insulin resistance/hyperglycaemia and dyslipidaemia, while the behaviours include physical activity levels, dietary habits, alcohol consumption, and smoking (Bazzano *et al.,* 2003; Pischon *et al.,* 2008). The key point however is that while 'conditions' and 'behaviours' are both modifiable factors, it is the behaviours that typically affect the conditions. In turn, the conditions affect the overall disease risk through the categorization of 'cardio-metabolic risk'. Figure 2 depicts this relationship below:

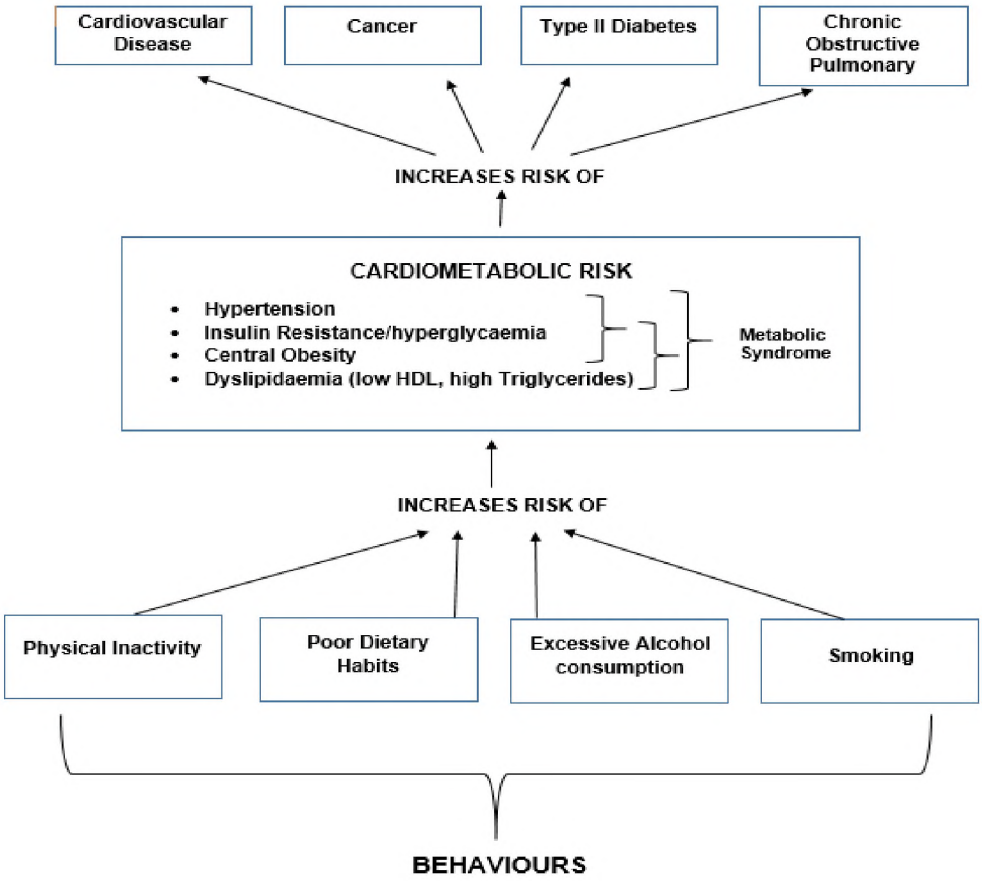

Figure 2: Diagram depicting the impact of modifiable behaviours on conditions and disease.

Research should therefore intervene at the behavioural level in order to effectively impact overall health (King *et al.,* 2007; Noakes *et al.,* 2013). In order to do so however, a detailed understanding of the modifiable health conditions associated with cardiovascular disease risk and how behaviour affects them, is required.

2.4.1 MODIFIABLE CONDITIONS

The following section unpacks the cardiovascular risk conditions in detail; however, due to the inherent relationship between risk behaviours and risk conditions, the mechanisms behind the development of these conditions will only be addressed later under 'modifiable behaviours'.

2.4.1.1 Hypertension

Hypertension is defined as the pathological elevation of blood pressure to a level ≥140/90 mm Hg, and is a major contributor to the development of cardiovascular disease (Galobardes *et al.,* 2003; Kemp *et al.,* 2011). In the majority of cases, hypertension contributes to mortality in the form of strokes, heart attacks, and kidney failure (WHO, 2011). More specifically, data from observational studies involving more than one million participants aged 40-89 years, indicated that death from both ischemic heart disease and strokes increased progressively and linearly as blood pressure increased (Kokkinos and Myers 2012). The authors stated in particular that for every 20 mm Hg systolic increase or 10 mm Hg diastolic increase in blood pressure, mortality from heart disease doubled. In addition, longitudinal data obtained from the Framingham Heart Study indicated that blood pressure levels between 130-139/85-89 mm Hg are associated with a more than twofold increase in risk of cardiovascular disease as compared with blood pressure levels below 120/80 mmHg (Simonneau *et al.*, 2013). However, the damage associated with hypertension can be mostly reversed if blood pressure is reduced back to normal levels (WHO, 2011). It is therefore imperative that hypertension management strategies are communicated and implemented in order to reduce cardiovascular disease risk.

Due to its prevalence, risk, and difficulty in management, hypertension is a global public health concern (Gasperin *et al.,* 2009). It affects approximately one billion people worldwide, and is expected to increase to 1.5 billion by 2025 (WHO, 2011). Kearney *et al.* (2005) indicated that the estimated number of hypertensive individuals in developing countries outweighed those in developed countries by almost twofold. However, contradictory global statistics indicate that one in three adults in American have hypertension, and the same is seen in South Africa and other developing countries (Kemp *et al.,* 2011). Nevertheless, despite the high prevalence worldwide, hypertension disproportionately affects black Africans, or those of black African

descent (Cappuccio, 1997). This is of particular importance in a South African context as hypertension is listed as the primary risk factor for cardiovascular disease in the local black populations- a finding likely linked to the high incidence of stroke within this group (Cappuccio, 1997; Kearney *et al.,* 2005; Kemp *et al.,* 2011).

In addition to being more prevalent in Black populations, hypertension is also more prevalent in females than in males (DHS, 2003; WHO, 2011). In the year 2000, 47000 premature deaths were attributed to hypertension in South Africa, and 66% of these were women (Maseko *et al.,* 2006; Norman *et al.,* 2007). The reason behind this disproportionate prevalence in females can only be speculated; but it has been suggested to be influenced by rural cultural norms whereby women are considered to be more attractive if they are overweight, and they are encouraged to adopt sedentary lifestyles (Walter and Durandt, 2011). This will be discussed in further detail under the 'modifiable behaviours' section.

Research has indicated that hypertension within Black populations may have a genetic link (Norman *et al.,* 2007), however country-wide statistics from the 1940s indicate that hypertension was largely non-existent within black South Africans at the time (Tibazarwa *et al.,* 2009). As a result, this draws attention to the impact of lifestyle factors as potential contributors to hypertension. Both the above mentioned cultural norms and the impact of lifestyle factors will be discussed in more detail under the 'modifiable behaviours' sections.

Interestingly, and likely influenced by the high prevalence of hypertension among black South Africans, the South African standardised blood pressure norms differ from norms indicated by the American Heart Association (2016). American norms state that a blood pressure between 120/80 mm Hg – 139/89 mm Hg is considered 'high-normal' or 'pre-hypertensive (WHO, 2011; American Heart Association, 2016). South African norms however indicate that blood pressure between 120/80 mm Hg - 129/84 mm Hg is still considered normal, while blood pressure between 130/85 mm Hg -139/89 mm Hg is considered 'pre-hypertensive' (South African Heart and Stroke Foundation, 2014). The distinction between the two norms is therefore in what is considered 'normal' versus 'pre-hypertensive', as they both indicate that a person is hypertensive from 140/90 mm Hg upwards. This is important when assessing an ethnically

homogenous sample of people as they require representative health norms for accurate representation of health status.

2.4.1.2 Obesity

Obesity is defined as a chronic condition where abnormal or excessive amounts of fat are accumulated and stored around the body (Rippe *et al.,* 1998). This condition leads to a plethora of health risks and implications, and is one of the greatest risk factors for cardiovascular disease and all-cause mortality (Després *et al.,* 2008; Goodpaster *et al.,* 2010; Tsatsoulis *et al.,* 2013). According to Goodpaster *et al.* (2010): "the high and sustained prevalence of obesity is among the most significant public health problems for the 21st century" pp. 9. As a result, it requires immediate and urgent attention.

The most widely used tool to classify overweight and obesity is body mass index (BMI); a ratio calculated between weight (kg) and height (m) which is then compared to established norms (James *et al.,* 2001). According to the World Health Organization criteria, obesity is diagnosed when BMI ≥ 30 $kg.m^{-2}$. Thereafter 'stages' of obesity, and associated level of risk, increases as BMI increases (Table 1).

Table 1: WHO classification of weight (WHO, 2004).

BMI ($kg.m^{-2}$)	**Classification**	**Risk**
< 18.5	Underweight	Low
18.5-24.9	Normal	Average
25.0-29.9	Overweight	Increased
30.0-34.9	Obese class I	High
35.0-39.9	Obese class II	Severe
≥ 40.0	Obese class III	Very severe

Despite the popularity of the BMI index, it is a crude measure as it cannot determine body composition; it can only determine total weight in relation to height (Nevill *et al.,* 2006; Després *et al.,* 2008). This becomes problematic when muscle mass cannot be accounted for, as a heavy muscle mass can translate into an 'overweight' or 'obese' BMI category, despite a low fat percentage. However, in the majority of individuals, it

can provide a basic insight into possible weight problems, and thus is commonly used as the first screening tool for obesity (Nevill *et al.,* 2006; Després *et al.,* 2008).

Despite the health risks associated with obesity in general, central obesity has been shown to have more adverse consequences than peripheral obesity (Després *et al.,* 2008; Tsatsoulis *et al.,* 2013). Central obesity is defined as the accumulation of fat around the abdomen, and is associated with high risk of hypertension, type II diabetes, and cardiovascular disease (Alberti *et al.,* 2005; Tsatsoulis *et al.,* 2013). It is primarily measured as waist-circumference (WC), but waist-to-stature ration (WSR) is an additional measure (Shields *et al.,* 2012). Waist circumference classifications do vary among different population groups, however the guidelines put forth by WHO based on several global studies provide a useful initial screen for risk profiles of individuals (Table 2).

Table 2: WHO classification of waist circumference (WHO, 2011).

Waist circumference (cm)		
	Risk	High Risk
Men	≥94	≥102
Women	≤80	≤88

The aetiology of obesity is multifactorial, consisting of a combination of genetic, metabolic, environmental, and behavioural factors contributing to its development and progression (Bray and Champagne, 2005). Nevertheless, it is more commonly thought that obesity occurs as a result of an energy imbalance, whereby energy consumed and stored exceeds energy expended (Potischman, and Freudenheim, 2003; King *et al.,* 2007; Taubes, 2008). In other words, it is widely accepted that obesity is strongly associated with poor diet and physical inactivity (Henderson, 2005). When looking at global statistics, this is supported by the fact that 75% of obesity is attributed to lifestyle factors, and only 25% is attributed to genetic and metabolic factors (Després *et al.,* 2008; Tsatsoulis *et al.,* 2013).

In South Africa, a noticeable prevalence of obesity can be seen in Black females; so much so that Black South African females are the most obese population in the country (Goedecke *et al.,* 2009; Tibazarwa *et al.,* 2009; Jackson, 2010). Country wide statistics

on overweight and obesity were first presented in the Department of Health Survey (DHS) carried out in 1998, and these indicated that 56% of females were overweight (across all races). Within the Eastern Cape however, it was shown that 25.7% of females were overweight; and a further 29.7% of females were obese. In a follow up survey carried out in 2003, notable increases in obesity rates were evident in the Eastern Cape when compare to the 1998 data. With 31.9% of females being classified as obese in 2003, the Eastern Cape presented with the highest obesity rates in the country (DHS, 2003). As these findings encompassed all races however, specific conclusions about Black South African females cannot be drawn. Nevertheless, these findings do highlight the prevalence of obesity in females and as the majority of the province is comprised of Black South Africans, it is likely that the majority of females making up this statistic would have been Black South African women (Stats SA, 2013).To support this conclusion, research from 2006 and 2010 indicate that Black South African women were not only significantly more obese than other populations, but that obesity prevalence in this group was increasing over time (van der Merwe and Pepper 2006; Jackson *et al.,* 2010).

2.4.1.2.1 'Metabolically healthy obese' South African women

Perhaps the most interesting and challenging factor coming to the forefront of research on South African populations is the observation of the 'metabolically health obese' phenotype evident in a number of Black South African women (Walker *et al.,* 2001; Weinsier *et al.,* 2001; Jennings *et al.,* 2008).

Jennings *et al.* (2008) highlight that while Black South African females were the most obese sub-population in the country, many of them expressed characteristics of metabolically healthy individuals, and thus were deemed the 'metabolically healthy obese'. Particularly when compared to their Caucasian counter parts, this study demonstrated that obesity in itself did not always correlate with metabolic distress in Black Africans; a direct contradiction of the commonly accepted association between obesity and cardiovascular disease risk (Alberti *et al.,* 2005; Tsatsoulis *et al.,* 2013). This study attributed the 'healthy obese' status of these black women to lower levels of visceral adiposity, which resulted in better glucose metabolism (Jennings *et al.,* 2008). Additionally, Weinsier *et al.* (2001) stated that the prevalence hypercholesterolemia, hypertension, and type II diabetes were low in many obese

Black South African women, and thus they exhibited a 'healthy obese' profile. However, this classification of 'metabolically healthy obese' Black South Africans needs to be interpreted with caution. All these studies have compared African data to European norms for cardiovascular risk, as African norms do not exist. As a result, it is possible that if the norms are adjusted specifically for African populations, the prevalence of cardiovascular disease risk would be different. Therefore further research assessing metabolic health profiles of Black South Africans is paramount to understanding the complexity associated race-specific disease risk.

2.4.1.3 Insulin Resistance and Type II Diabetes

In order to understand the mechanisms behind insulin resistance and type II diabetes, the role of insulin in the body needs to first be understood.

2.4.1.3.1 The role of insulin

Insulin is a peptide hormone produced by cells in the pancreas and it serves to regulate glucose metabolism in the body (Reaven, 1993; Ball *et al.,* 2004). Once broken down in the stomach, foods containing carbohydrates, as well as pure sugars, convert into glucose and are transported to the body's cells through the bloodstream (Ludwig, 2002). The cells however require the presence of insulin as they cannot receive the glucose and use it as energy without the presence of insulin (Ludwig, 2002). Thus shortly after food is ingested and blood glucose levels begin to elevate, the brain sends a message to the pancreas to release insulin into the blood stream in order to effectively transport the glucose in the blood to the body cells to be used as energy (Tsatsoulis *et al.,* 2013). Insulin achieves this by binding to the individual cells and generating a gateway for glucose to be diffused into the cell (Reaven, 1993; Noakes *et al.,* 2013; Després *et al.,* 2008).

A second role of insulin is to maintain adequate and safe blood glucose levels (Reaven, 1993). If more glucose is in the blood than is needed to provide cells with sufficient energy, insulin acts to remove the excess glucose and stores it in fat cells (Reaven, 1993). Without this action, excess blood glucose that is not transported into working cells for energy will remain in the blood stream which could lead to hyperglycaemia; a state in which blood glucose levels are elevated above normal levels (< 7 $mmol.l^{-1}$) (American Diabetes Association, 2014). If this state is prolonged,

damage to nerves, blood vessels, impaired vision and kidney failure can occur as blood flow to these organs is compromised due to the viscosity of the blood, and this can lead to cell death (Ball *et al.,* 2004). Thus insulin not only acts to transport energy in the form of glucose into the cells, but also acts as a protective mechanism against hyperglycaemia (Tsatsoulis *et al.,* 2013). The body therefore relies on sufficient and timely release of insulin from the pancreas in order to effectively manage energy transportation and homeostasis in the body (Ludwig, 2002; Ball *et al.,* 2004; Noakes *et al.,* 2013).

2.4.1.3.2 Insulin resistance

Insulin resistance occurs when insulin levels are excessively high (>10 mmol.l^{-1}) over a prolonged period of time, causing the body's sensitivity to the hormone to be reduced (Reaven, 1988; Ludwig, 2002; Després *et al.,* 2008; Noakes *et al.,* 2013; Tsatsoulis *et al.,* 2013). Insulin resistance is thus synonymous to reduced insulin sensitivity (Reaven, 1988). An insulin-resistant response occurs when the pancreas overproduces insulin in response to a rise in blood glucose (Tsatsoulis *et al.,* 2013). In most cases, the insulin production at this high level can continue to maintain blood glucose levels for a period of time, thus it is not classified as type II diabetes (Reaven, 1988; Ludwig, 2002). However, prolonged increased output of insulin can desensitize the cells to the effects of insulin over time, thus reducing the amount of glucose able to diffuse into the cells, and thereby increasing levels of circulating glucose in the blood (hyperglycaemia) (Reaven, 1988). In conjunction, over time, the pancreas becomes unable to keep up with the body's increased need for insulin thus further elevating concentrations of glucose in the blood (Ludwig, 2002). Consequently, insulin resistance is often referred to as 'pre-diabetes' as people who have insulin resistance often develop type II diabetes at a later stage (Reaven, 1988). Thus, effective management of insulin resistance is essential to preventing the development of type II diabetes (Reaven, 1988; Després *et al.,* 2008). Furthermore, insulin resistance is associated with a number of other health implications including elevated triglyceride levels, increased visceral adiposity, and elevated blood pressure, which further emphasizes the urgent need to explore effective management strategies (Després *et al.*, 2008).

2.4.1.3.3 Diabetes

Two forms of diabetes exist: type I diabetes and type II diabetes (Ludwig, 2002; Tsatsoulis *et al.,* 2013). Type I diabetes, also known as insulin-dependent diabetes, is commonly a hereditary condition in which the pancreas is incapable of producing adequate amounts of insulin (American Diabetes Association, 2014). However, individuals may develop type I diabetes later on in life due to exposure to certain viruses that permanently damage the pancreatic cells or pancreatic cancer (WHO, 2011). Nevertheless, as this is primarily a genetic disease that presents early on in life, there is a natural paucity in the literature with respect to its impact on cardiovascular disease. The majority of literature explores the impact of type II diabetes, an acquired type of diabetes, on cardiovascular health.

Type II diabetes, also referred to as 'diabetes mellitus' or 'non-insulin dependent diabetes', is the most common form of diabetes worldwide (Reaven, 2005; Noakes *et al.,* 2013). Unlike type I diabetes, type II diabetes is lifestyle-related whereby it develops over time due to overstimulation of the pancreas which initially causes insulin resistance (Noakes *et al.,* 2013). If the body adopts an insulin resistant state for a prolonged period of time, this typically causes the pancreas to fail to secrete necessary insulin levels, which causes a lack of energy to the cells and blood glucose levels rise (Ludwig, 2002; WHO, 2011; Tsatsoulis *et al.,* 2013). This represents the physiological state of a person with type II diabetes. Therefore in direct contrast to type I diabetes, type II diabetes is classified by excessive insulin levels, where type I is characterized by insufficient levels (American Diabetes Association, 2014). This is of importance as it is precisely this distinction that makes type II diabetes a huge risk for cardiovascular disease, as it is associated with the damaging effects of high insulin levels (Reaven, 1988; Reaven, 2005).

Among type II diabetic individuals, coronary heart disease and strokes account for 90% of all premature deaths (National Diabetes Data Group, 1995). As a result, type II diabetes was recognized as a coronary artery 'disease risk-equivalent' by the National Institute of Health in 1995. This classification suggests that the risk of heart disease in a person with type II diabetes is equal to that of a person without type II diabetes who has previously experienced a coronary event (Eckel *et al.,* 2005). Further statistics indicate that individuals with type II diabetes have a two-to-fourfold increase

in risk of developing coronary artery disease (WHO, 2011). Interestingly however, the risk is much greater in women than in men.

Results from the WHO MONICA Project (1988) assessed mortality rates in men and women with type II diabetes aged 25-64 and found that mortality was 38% higher for diabetic men and 86% higher for diabetic women one year after diagnosis (Evans *et al.,* 2001). More recent research shows similar findings as it is well documented that women with type II diabetes are at a greater risk for developing cardiovascular disease than their male counterparts (WHO, 2011; American Heart Association, 2015; National Heart, Lung, and Blood Institute, 2016). Although the exact mechanism by which type II diabetes places women at greater risk is not well understood, Tsatsoulis *et al.* (2009) state that: "The disparity between the incidence of coronary heart disease in age-matched premenopausal women and men suggests that endogenous sex hormones such as oestrogen and progesterone or both have a significant influence on vasculature… type II diabetes therefore abrogates the protective effects of oestrogens in premenopausal women" (pp. 36-37). Therefore, as oestrogen can no longer play a cardio-protective role in women with type II diabetes, they inevitably succumb to greater risk for cardiovascular disease (Tsatsoulis *et al.,* 2009).

2.4.1.4 Dyslipidaemia

When addressing the health implications associated with elevated cholesterol levels, the two most common conditions that are referred to are 'hypercholesterolemia' and 'dyslipidaemia' (Nelson *et al.,* 2014). In order to understand these conditions however, 'cholesterol', and the role it plays in the body, needs to first be defined.

Cholesterol is the broad name given to a form of steroidal-lipid that is responsible for a number of functions in the human body (American Heart Association, 2016). These functions include maintaining the permeability and fluidity of cell membranes, as well as being a precursor to synthesis of steroid-base hormones such as oestradiol and testosterone, and fat-soluble Vitamin D (WHO, 2011). The word itself is derived from the ancient Greek words 'chole'- (bile) and 'steros' (solid), to which the suffix –'ol' (alcohol) is added (Google dictionary).

Cholesterol itself is located within lipoproteins, which serve as transport molecules for cholesterol throughout the body (Grundy *et al.,* 1999). All lipoproteins comprise of

apolipoproteins, phospholipids, triglycerides, and cholesterol, however there are three distinct types of lipoproteins which vary according to the density (Superko & Krauss 2000; Sharma *et al.,* 2009). These types are: very low density lipoprotein (VLDL), low density lipoprotein (LDL) and high-density lipoprotein (HDL) (Sharma *et al.,* 2009). Although triglycerides exist within lipoproteins, they can also exist outside of them as 'serum triglycerides', which circulate in the blood and aid in fuelling the cells with energy (Superko & Krauss 2000; Sharma *et al.,* 2009). In normal quantities, all lipoproteins and triglycerides serve a fundamental role in maintaining homeostasis in the body; however it becomes detrimental to health when normal concentrations are disrupted (Superko and Krauss, 2000). In particular, high levels of total cholesterol, otherwise referred to as 'hypercholesterolemia', associated with increased risk of cardiovascular disease (Grundy *et al.,* 1999; Adiels *et al.,* 2008; Sharma *et al.,* 2009; Peer *et al.,* 2013; Goedecke *et al.,* 2015).

It is important to note however that when referring to cholesterol levels or more specifically, hypercholesteromemia, it is in fact the lipoprotein content (within which the cholesterol resides) that is being measured (Brunzell *et al.,* 2008). In other words, any elevation in VLDL, LDL, HDL, or triglyceride concentrations will contribute to an elevated 'total cholesterol' level (Brunzell *et al.,* 2008). However, a key limitation of measuring hypercholesterolemia is that it doesn't distinguish between which types of lipoproteins that are elevated, only that 'total cholesterol' (a crude and imprecise term), is elevated (Superko & Krauss, 2000). For example, normative values for total cholesterol are accepted as <5.2 mmol/l^{-1}; any values higher than this would exhibit a hypercholesteraemic state (Eckel *et al.,* 2005; Adiels *et al.,* 2008). This is inherently problematic however as not all types of lipoproteins are harmful when elevated; one is in fact protective (Castelli, 1996; Webber *et al.,* 2014).

It is well established throughout literature that high concentrations of both LDL cholesterol and triglycerides are positively correlated with increased risk for cardiovascular disease (Castelli, 1996; Grundy, 1998; Eckel *et al.,* 2005; Adiels *et al.,* 2008; Sharma *et al.,* 2009; Goedecke *et al.,* 2015). This is due to the small size and density of LDL particles which enables them to filter more rapidly into arterial wall. As a result, they often get lodged within the lining which develops into plaque build-up over time (Grundy, 1999). As the plaque builds, it can eventually cause a blockage in the artery which can lead to myocardial infarction (Libby and Theroux, 2005).

Alternatively, the blockages harden the lining of the artery which can lead to atherosclerosis (Grundy, 1999; Adiels *et al.,* 2008).

With respect to triglycerides, these are purely fat molecules that the body uses as fuel (Nordestgaard and Varbo, 2014). However, excess triglycerides lead to the synthesis of smaller, and denser LDL particles which increases their likelihood of getting lodged into the artery wall (Grundy, 1998; Nordestgaard and Varbo, 2014). It is therefore well accepted that in order to maintain cardiovascular health, LDL and serum triglyceride concentrations need to remain within safe levels (< 2.59 mmol/l^{-1} and < 1.7 mmol/l^{-1} respectively) (Castelli, 1996; Grundy, 1998; Eckel *et al.,* 2005; Adiels *et al.,* 2008; Sharma *et al.,* 2009; Kontush, 2014; Webber *et al.,* 2014).

In contrast to LDL and triglycerides, high concentrations of HDL serves a protective role from developing cardiovascular disease (Castelli, 1996; Tran-Dinh *et al.,* 2013; Kontush, 2014; Webber *et al.,* 2014). In a study conducted by Castelli (1996), the ratio of HDL to total cholesterol was found to be a greater indicator of heart disease than total cholesterol alone. In other words, this study showed that sex and age-matched individuals with a total cholesterol of 5.2 mmol/l^{-1}, and an HDL level of <1.04 mmol/l^{-1}, were found to be at the same risk as individuals with a higher total cholesterol of 6.7 mmol/l^{-1}, who also had higher HDL concentrations (Castelli, 1996). More recent studies have shown similar results, strengthening the finding of the protective effects of HDL on cardiovascular health (Tran-Dinh *et al.,* 2013; Kontush *et al.,* 2014; Webber *et al.,* 2014). Therefore, the use of total cholesterol as an indicator of health presents an unclear picture; it cannot ascertain the ratio of HDL, LDL and triglycerides and thus any conclusions drawn from it are innately limiting. Because of this, a more detailed assessment of cholesterol ascertaining the precise ratio of HDL, LDL, and triglycerides is typically used (Supoerko & Krauss, 2000). When this ratio is suboptimal, a state of 'dyslipidaemia' is reached (Adiels *et al.,* 2008; Sharma *et al.,* 2009).

Dyslipidaemia is determined using the 'atherogenic lipoprotein phenotype' (more commonly referred to as the 'lipid triad' or 'dyslipidaemic triad') which is characterized by the combination of high serum triglyceride levels (> 2 mmol/l^{-1}), high LDL levels (> 3.37 mmol/$^{l-1}$) and low HDL levels (< 1.04 mmol/$^{l-1}$) (Sharma *et al.,* 2009; Tran-Dihn *et al.,* 2013; Kontush, 2014; Webber *et al.,* 2014). Despite the former popularity of using hypercholesterolemia as an indicator of cardiovascular disease risk, the association

between dyslipidaemia and cardiovascular disease risk is undeniable, hence its inclusion in the metabolic syndrome criteria (Eckel *et al.,* 2005; Alberti *et al.,* 2005; Després *et al.,* 2008).

Worldwide, dyslipidaemia disproportionately affects Caucasians in comparison to those of black African ancestry (Goedecke *et al.,* 2015). Studies conducted in both the USA and South Africa have shown that Black women specifically exhibit lower total cholesterol, triglycerides, and LDL than Caucasian counterparts (Després *et al.,* 2000; Punyadeera *et al.,* 2001; Peer *et al.,* 2013; Goedecke *et al.,* 2015). Nevertheless, HDL concentrations are comparable between the two groups. These findings could explain why Black South African women have a lower lipid-attributed mortality rate compared to Caucasian counter parts (47 in 100 000 and 152 in 100 000 respectively) (Lambert *et al.,* 2007). One explanation is that black women have lower levels of visceral adiposity, which can therefore have a positive impact on cholesterol levels (Weinsier *et al.,* 2001; Jennings *et al.,* 2008). Nevertheless, it has also stated that black women present with more insulin resistance than white women (Eckel *et al.,* 2005). This begs the question of whether or not genetic polymorphisms may play a role in the more favourable lipid profiles in black females (Eckel *et al.,* 2005). Alternatively, cultural-specific lifestyle factors which may contribute to this dichotomy requires exploration.

2.4.2 THE METABOLIC SYNDROME AND CARDIO-METABOLIC RISK

Despite the increased risk of cardiovascular disease associated with any of the above modifiable conditions, what is more pertinent is the noticeable clustering of these conditions that often occur in a number of individuals (Reaven, 1999; Alberti *et al.,* 2005; Grundy *et al.,* 2005; Kahn *et al.,* 2005; Miranda *et al.,* 2005). This clustering of conditions is most commonly referred to as 'the metabolic syndrome', although a number of names such as syndrome X, dysmetabolic syndrome, and insulin resistant syndrome have been used (Reaven, 1988; Kahn *et al.,* 2005; Miranda *et al.,* 2005).

In his Banting Award Lecture in 1988, Gerald Reaven first described a syndrome characterized by the co-occurrence of a number of metabolic disturbances or conditions such as hypertension, insulin resistance, and dyslipidaemia (Reaven, 1988). He postulated that this syndrome was not only frighteningly common, but was paramount to identifying individuals at high risk of developing type II diabetes and cardiovascular disease (Reaven, 1988; Després *et al.,* 2008). In other words, he

hypothesized that the presence of this syndrome drastically increased a person's cardio-metabolic risk; a term used to predict the lifetime risk for developing cardiovascular disease[2] (Klein *et al.,* 2007). Reaven termed this syndrome 'syndrome X', although it was soon more commonly referred to as 'metabolic syndrome' as the diagnostic criteria evolved (Alberti *et al.,* 2005). Since then, there has been considerable evidence signifying an association between metabolic syndrome, type II diabetes and cardiovascular disease, and this led to attempts to produce standardised diagnostic criteria (Isomaa *et al.,* 2001; Alberti *et al.,* 2005; Eckel *et al.,* 2005; Kahn *et al.,* 2005; Miranda *et al.,* 2005; Després *et al* 2008).

In its infancy, the metabolic syndrome (or syndrome X as it was known then), was described by Reavan as a clustering of the following conditions: insulin resistance and/or hyperglycaemia; hypertension; and dyslipidaemia (particularly low HDL cholesterol and high VLDL triglycerides) (Reaven, 1988). In 1999 however, the World Health Organization revised these criteria to include obesity (particularly central obesity), as there was considerable evidence highlighting the intrinsic link between central obesity and metabolic disturbances; a finding that Reavan missed (Isomaa *et al.,* 2001; Alberti *et al.,* 2005; Kahn *et al.,* 2005). They additionally added that insulin resistance or its surrogates (impaired glucose tolerance or type II diabetes), required the presence of two additional conditions to be classified as metabolic syndrome, rather than all five conditions being present (Alberti *et al.,* 2005). The key however being that insulin resistance was the cornerstone of metabolic syndrome; a concept that Reaven himself initiated (Reaven, 1988; Alberti *et al.,* 2005; Reaven, 2005). In 2002 however, the US National Cholesterol Education Program: Adult Treatment Panel III took a less glucocentric approach: there was evidence to suggest that metabolic syndrome could be diagnosed without the presence of impaired glucose tolerance (Després *et al.,* 2008). Thus, they altered the diagnostic criteria accordingly and stated that any three of the five conditions need to be present to be diagnosed with metabolic syndrome; even in the absence of glucose-related conditions (Alberti *et al.,* 2005). These five conditions were: central obesity (WC of ≥ 94 cm men, ≥ 80

[2] 'Cardio-metabolic risk is determined the presence of a single condition, or a combination of the conditions including obesity, insulin resistance or type II diabetes, hypertension, and dyslipidaemia (Klein *et al.,* 2007; Després *et al.,* 2008). For example, a person presenting with hypertension alone has a higher cardio-metabolic risk than a healthy person without hypertension. Similarly, a person presenting with hypertension and insulin resistance has a higher cardiometabolic risk than a person with only hypertension (Després *et al.,* 2008). Cardio-metabolic is most severe in people with metabolic syndrome, although cardio-metabolic risk can still be evident in those without metabolic syndrome (Klein *et al.,* 2007).

cm females); hypertension (BP ≥ 130/85 mm Hg); high triglycerides (> 1.7 mmol.l^{-1}); low HDL cholesterol (> 1.03 mmol.l^{-1} men, < 1.29 mmol.l^{-1} women) and fasting hyperglycaemia (fasting plasma glucose ≥5.6 mmol.l^{-1}) (National Cholesterol Education program, 2002).

Due to the number of different definitions and diagnostic criteria however, this led to considerable confusion and lack of comparability and reproducibility between studies assessing prevalence of the metabolic syndrome (Alberti *et al.,* 2005). In particular, two critical problems have been identified: 1) the range of metabolic profiles evident in those with metabolic syndrome, and 2), the absence of racial consideration with respect to generalised norms (Alberti *et al.,* 2005; Eckel *et al.,* 2005; Després *et al* 2008)

2.4.2.1 Metabolic profiles

Using the 'three of five' framework, it is commonly accepted that if an individual presents with three or more of the above health conditions, they are diagnosed with metabolic syndrome (Balkau *et al.,* 2007). However, as there are a mosaic of combinations of three of the five criteria for metabolic syndrome, this makes it difficult to compare level of risk from person to person (Després *et al.,* 2008). For example, person 'A' might present with hypertension, insulin resistance, and low HDL-cholesterol concentrations, while person 'B' presents with central obesity, elevated triglyceride levels and hypertension. Both individuals can be classified as having metabolic syndrome, but their metabolic profiles are considerably different (Després *et al.,* 2008). How can one then compare their level of risk for cardiovascular disease or type II diabetes? Is one at a greater risk than the other? This is difficult to determine as the impact of each individual condition, and the interaction of all conditions, is not quantifiable. Therefore both individuals can be classified as 'high' risk candidates, but their levels of risk cannot be directly compared to one another (Després *et al.,* 2008).

This leads into the next limitation; the quantification of risk. The notion of quantifying 'risk' is inherently problematic as it lies on a continuum and is not absolute (Marquis *et al.,* 2005; Després *et al.,* 2008). Therefore, the fact that the five variables are not used as continuous variables but rather counted as 'present' or absent, compromises the quality of diagnosis of the metabolic syndrome by virtue of overlooking the 'degree'

of classification (Després *et al.,* 2008). In other words, person 'C', with the same 'present' conditions as person 'A' mentioned above, may have a higher blood pressure than person A (less favourable) but also a higher concentration of HDL-cholesterol (more favourable). Both patients have the same present conditions, but the degree and progression of the individual conditions vary, adding further complexity to the classification.

As a result, it is imperative that researchers understand the limitations of the current diagnostic tools for metabolic syndrome. The diagnostic tools can offer insight into metabolic health, but the inherent complexity associated with 'level of risk' must be approached with caution.

2.4.2.2 Racial consideration

Secondly, there is evidence to suggest that the cut-off values for the five conditions (central obesity, hypertension, fasting hyperglycaemia, HDL and triglyceride concentrations) are not representative to all ethnicities (Wang *et al.,* 1994; Gurrici *et al.,* 1998; Deurenberg-Yap *et al.,* 2002; Tan *et al.,* 2004). A study by Tan *et al.* (2004) assessed the metabolic health of 4723 Asian males and females of varying ages using the Adult Treatment Panel III criteria. Results showed that risk for type II diabetes was apparent at much lower levels of central obesity in Asians than in Europeans (Tan *et al.,* 2004). This was explained by the fact that Asians had a higher percentage body fat at a lower BMI compared to Caucasians (Deurenberg-Yap *et al.,* 2002). In light of this, the study concluded that the metabolic syndrome was largely underestimated in many Asian populations (Tan *et al.,* 2004). Further studies conducted in Singapore and China support the findings by Tan *et al.* (2004); they found that cardiovascular disease and type II diabetes incidences occurred at lower BMI values than in Caucasian counter parts (Gurrici *et al.,* 1998; Wang *et al.,* 1994). In addition, Gurrici *et al.* (1998) identified that for any given percentage of body fat, BMI was 3 kg/m^2 lower in Singaporeans. These studies are but a few that highlight the short-comings associated with applying 'blanket' cut-off values for metabolic syndrome to different ethnicities. Future studies should aim to ascertain level of risk within specific populations by taking into account their genetic differences and susceptibilities, in order to obtain more accurate diagnoses.

Within a South African context, this problem is just as prevalent. A number of studies have emphasized the need for cut-off values specific to Black African populations, as there is considerable evidence to suggest that metabolic profiles differ between races (Schutte and Olckers, 2007; Després *et al.,* 2008; Jennings *et al.,* 2008; Schutte *et al.,* 2014). Schutte and Olckers (2007) assessed and compared metabolic syndrome risk factors in 102 Black women and 115 Caucasian women (mean age of 31.3 ± 8.64 years). They found that 30.4% of Caucasian women had metabolic syndrome, compared to a 24.8% prevalence in Black women. However, when individual risk factors were assessed, the Black women were found to have higher odds ratios for having metabolic syndrome for HDL cholesterol, blood pressure, and fasting glucose levels than Caucasians. Therefore despite not meeting the 'three of five' criteria for metabolic syndrome as frequently as Caucasian women, these Black women were at similar risk of cardiovascular disease and all-cause mortality. In light of this, it was suggested that the waist-circumference cut-off values were inappropriately high for African women, and needed to be downwardly adjusted. This study therefore concluded that the percentage of Black women with metabolic syndrome is likely underrepresented in South Africa (Schutte and Olckers, 2007).

Després *et al.* (2008) similarly stated that the 88 cm cut-off value for waist-circumference in women was not applicable to all women across age and race. Here, the authors firstly stated that although pre-menopausal women stored fat differently to men (peripheral compared to central), post-menopausal women stored fat similarly to men (both central) (Reckelhoff, 2001; Carr, 2003; Dubnov *et al.,* 2003) As a result it was concluded that the waist-circumference cut-off value of ≥ 88 cm cannot be accurately applied to women of all ages, but is rather more applicable for pre-menopausal (Després *et al.,* 2008). Secondly, this study emphasized the need for different cut-off values for different races. This is due to the difference in fat distribution patterns among women of different races; for example, pre-menopausal Black African women tend to store fat more peripherally than age-matched Caucasian women (Després *et al.,* 2008). Therefore, it was concluded that this difference in fat storage likely makes their sensitivity to central adiposity, and thus its impact on health, different (Després *et al.,* 2008). Jennings *et al.* (2008) conducted a study assessing the prevalence of metabolic disturbances associated with obesity in Black South African women. It was found that a higher percentage of these women were termed

'metabolically health obese' as compared to Caucasian counterparts. This was largely attributed to differences in fat distribution between Black South African women and Caucasians, whereby more peripherally located fat stores in African women made them less susceptible to the dangers associated with centrally located fat. However, although this study highlighted the different metabolic profiles between Black South African and Caucasian women, it didn't question the need for race-specific norms which may lead to more accurate representation of risk in these sub-groups. It therefore becomes premature to state that obesity in Black South African women is less associated with metabolic distress as in Caucasian women, as this is only the case when looking at 'general', non-race specific norms. Further research in South African therefore needs to delve into the complexities associated with race specific norms, and endeavour to establish these prior to comparing disease prevalence between races.

2.5 MODIFIABLE BEHAVIOURS

2.5.1 Dietary habits

In addition to the quadruple burden of disease, South Africa suffers from another health epidemic known as the 'double burden of malnutrition' (Abuya *et al.,* 2012). This refers to a state in which a population experiences increasing levels of obesity, juxtaposed with a persistence of 'under-nutrition' (Abuya *et al.,* 2012). Under-nutrition or 'malnutrition' is typically a concern to those living in rural areas of South African, while over-nutrition has begun to increase rapidly in more urbanized areas (Steyn *et al.,* 2002; Stupar *et al.,* 2012). This increase has been so rapid in recent decades that over-nutrition has been termed 'the silent emergency' of the developing world (Crush *et al.,* 2011). This is attributed primarily to the nutrition transition. It is therefore essential to assess the nutritional habits of South Africans in order to gain a deeper understanding of their overall health profile.

2.5.1.1 The nutrition transition and a 'Westernized diet'.

The 'nutrition transition' is a term that describes the changes in habitual dietary patterns of a population as they transition from a predominantly rural lifestyle, to an urban industrialized one (Vorster *et al.,* 2002). Intrinsically linked to this is an 'epidemiological transition', which describes a shift from largely infectious diseases, to

chronic diseases associated with urbanization (Omran, 1998; Popkin and Gordon-Larsen, 2004). In South Africa, the traditional rural diet consisting of grains, starchy roots, legumes, vegetables, and fruit, has been abandoned in many urbanized areas for a diet high in energy-dense foods such as processed foods, animal-based foods, and foods with added fat and sugar (Popkin, 1994). As the 'traditional' diet is associated with a low prevalence of degenerative diseases, the adoption of the 'Westernized diet' has subsequently been suggested to have a negative impact on the health status of these South Africans (Popkin and Gordon-Larsen, 2004). In order to understand the influence of a Westernized diet on health, it is first necessary to understand what constitutes this diet.

Interestingly, despite the common use of the term, the definition and description of a Westernized diet is often incomplete or inaccurate. Typically, such a diet is described as one high in saturated fat, and low in carbohydrates and fibre (Steyn *et al.,* 2002; Vorster, 2002; Popkin and Gordon-Larsen, 2004). However, upon careful analysis, it appears that this is misleading. Firstly, one of the main components of a Westernized diet is fast food, where 'fast food' is defined as: "easily prepared and processed food such as burgers, pizzas, and fried chicken that are served at restaurants or fast food chains" (American Heart Association, 2016). Typically, this type of food is low in nutrients and low in fibre, while usually high in 'trans fats', rather than saturated fats (Mayo Clinic, 2016). Additionally, these foods have added sugars.

'Trans fats', otherwise known as trans-unsaturated fatty acids, are commonly produced industrially from vegetable fats through a chemical process of hydrogenation of oils, and is the most common form of fat used for deep-frying in fast food restaurants (Mayo clinic, 2016). As a result, the regular consumption of fast food normally leads to increased consumption ''trans fats'. Although saturated fat is a component of fast food, 'trans fats' often exceed the saturated fat percentage substantially (Mayo Clinic, 2016).

Secondly, a Westernized diet is described as one in which carbohydrates and fibre are generally replaced with foods derived from animals, such as meats, eggs, and diary (WHO, 2008; FBDG-SA, 2013). More specifically, it is widely accepted that over the past 100 years, the shift toward consumption of food derived from animals corresponds with the rapid increase in cardiovascular disease mortality globally (Norat

et al., 2005; Noakes *et al.,* 2013). Despite this, there is evidence to suggest that these perceived global dietary trends are based on incomplete and inaccurate data from the United States Department of Agriculture statistics (USDA) in the 1940s-1980s (Taubes, 2008). These statistics in fact show that from the end of World War II, the consumption of whole milk, cream, and meat in America was half of what it was in the years prior to the war (Taubes, 2008). Additionally, the consumption of vegetable fat during this time (1947–1976) nearly doubled (Taubes, 2008). Since then, global industrialization and urbanization has increased the consumption of fast food, and thus the 'Westernized diet' has in fact transformed into one high in trans fats, and refined sugars, rather than one high in animal fat and protein (Taubes, 2008). In a South African context, this is evidenced by the common shift from 'traditional foods' consisting of pap (porridge made from ground maize), amasi (sour milk), imifino (wild spinach and other wild leaves), and trotters (pigs feet), to a more 'Westernized diet' comprising of processed foods high in trans fats and sugar such as fried chicken, hamburgers, potato chips, pies, sweets, and cake (Stupar *et al.,* 2012).

The nutrition transition is generated by various aspects of economic globalization, urbanization with more sedentary lifestyles, changes in income, and influences of the modern food industry (Stupar *et al.,* 2012). In particular, there is an apparent association between economic status and dietary habits. Stupar *et al.,* (2012) interviewed a number of focus groups in urbanized Black South African schools, and found that there is a superior social status attached to being able to eat at fast food places, particularly fried chicken outlets. In a school setting, this notion of 'status' extends to being able to afford 'junk' food from a tuckshop, rather than having to rely on packed food from home which is perceived as 'poor' (Stupar *et al.,* 2012).

2.5.2 Physical inactivity

The association between physical inactivity and cardiovascular disease has been widely researched and acknowledged as a growing global concern (Blair, 2009; Walter and Durandt, 2011). Blair (2009) specifically states that physical inactivity is not only a concern, but is the biggest public health concern of the 21st century. In 2014, physical inactivity was identified as the fourth leading risk factor for developing chronic diseases, preceded only by tobacco use, hypertension, and high blood glucose levels (Bull and Bauman, 2011; WHO, 2011). Despite being fourth on the list however, the

actual impact of physical inactivity cannot be directly compared to hypertension and high blood glucose levels as physical inactivity is a behaviour, while hypertension and high blood glucose levels are conditions. Since behaviours typically impact conditions, it can therefore be argued that it is *due* to behaviours such as physical inactivity and tobacco use that conditions such as hypertension and high blood glucose levels arise in the first place, which then lead to increased risk of all-cause mortality. Additionally, physical inactivity is an intangible factor with respect to mortality; it is not possible to determine death directly due to physical inactivity. Rather, physical inactivity could lead to a number of health conditions which give rise to disease, which then results in premature death. Therefore statements such as '3.2 million premature deaths were attributed to physical inactivity alone in 2014' by the World Health Organization (2016) are equally misguided as they are presumptuous since a), the deaths cannot be tangibly attributed to physical activity alone, and b), the causes of death in this particular statistic negate other external factors that could have contributed to ill health such as poor diet and/or genetic factors. It therefore remains pertinent to distinguish behaviours from conditions in order to assess and compare risk. Nevertheless, what is clear, is that physical inactivity is a worldwide problem, contributing to many health complications; thus attempts to increase levels of physical activity worldwide is pertinent to improving overall health (Blair *et al.,* 2004; Bradshaw *et al.,* 2007; Garber *et al.,* 2011).

The 1995 American College of Sports Medicine (ACSM) guidelines recommended that adults should accumulate at least 30-minutes of moderate-intensity cardiorespiratory exercise on most, but preferably all days of the week to reap the health benefits associated with exercise (Blair *et al.,* 2004; Garber *et al.,* 2011). These health benefits include, but are not limited to, improved glucose sensitivity, lowered blood pressure, improved lipid profile, reduced risk of colon and breast cancer, reduced levels of depression, and weight loss (Bradshaw *et al.,* 2007). However, in 2008, data showed that 34% of women and 28% of men worldwide did not meet these recommendations (WHO, 2010). Since then, prevalence of physical inactivity or 'sedentariness' has only increased, predominantly in low to middle-income countries (WHO, 2010).

As with the nutrition transition, the decrease in physical activity levels is equally attributed to rapid urbanization and industrialization, as this has led to the mechanisation of many previously manual jobs, and an increase in sedentary

behaviour during many other occupational and domestic activities (Bradshaw *et al.,* 2007; WHO, 2010). Additionally, 'passive' modes of transport have also been associated with declining activity levels, although this is more prevalent in first world countries (Bradshaw *et al.,* 2007).

However, one pressing question is evident. As there is considerable evidence highlighting the association between increased physical activity, improved over-all health, and weight-control, it becomes somewhat perplexing that obesity has become a global epidemic. In other words, if increased physical activity is the solution to combating obesity, why are people not engaging in physical activity? On possible answer to this question lies in the critical analysis of the 'energy-in, energy-out' model of weight-management.

2.5.2.1 Energy-in, energy-out model

The 'energy-in, energy-out', which is the most commonly accepted model for explaining the aetiology of obesity, works on the premise that obesity occurs as a result of a surplus of energy-in over time. This is due to the intake of more energy (calories) than are expended through exercise or activities of daily living (ADL) (King *et al.,* 2007; Taubes 2008). As a result, the body has no means of 'using up' the surplus of energy that is taken in, and thus stores it body fat (Tsatsoulis *et al.,* 2013). The solution to obesity is therefore to expend more energy than is taken in for weight loss to occur. Alternatively, if weight is to be maintained, one must simply expend as much energy as is consumed (Taubes, 2008). Based on this model, the worldwide epidemic of obesity would be easily rectified if people ate less and exercised more. Nevertheless, despite physical activity promotion and education, obesity rates are increasing, and there are 700 million obese people in the world (van Zyl *et al.,* 2012). King *et al.* (2007) provide one possible explanation to this.

In their paper, King *et al.* (2007) explore the traditional 'energy-in, energy-out' model in greater detail and conclude that this mechanism of understanding is outdated. They highlight the metabolic and behavioural compensatory responses associated with an increase in energy expenditure, in response to an energy imbalance (King *et al.,* 2007). Due to this imbalance, and the fact that the human body is always looking to achieve and maintain homeostasis, this energy imbalance is usually compensated for by an increase in energy intake, and/or an increase in sedentary time (King *et al.,*

2007). This points to a much more nuanced and intricate understanding of obesity development, and highlights the need to address many different lifestyle factors and their interactions with each other in order to gain a deeper understanding into the aetiology of this disease.

2.5.2.2 Physical inactivity in South Africa

Within South Africa, black women are identified as the most physically inactive, as well as the most obese (Walter and Durandt, 2011). Despite controversy around whether these women are typically more metabolically healthy than Caucasians, there is still a plethora of evidence suggesting that they are still a high risk group for chronic diseases, particularly cardiovascular disease and type II diabetes (Vorster *et al.,* 2002; Bradshaw *et al.,* 2007; Walter and Durandt, 2011). Although there is evidence to suggest that the high obesity rates within this population group may be attributed to genetics in part (Jennings *et al.,* 2008), other evidence highlights cultural beliefs and norms as the primary contributor to the epidemic (Vorster *et al.,* 2002; Steyn *et al.,* 2004; Bradshaw *et al.,* 2007; Walter and Durandt, 2011).

Walter and Durandt (2011) assessed the socio-cultural barriers to physical activity among black isiXhosa speaking urban professional women, and found that the biggest barrier towards physical activity was the perceived 'unattractiveness' attached to weight loss by both the women themselves, and their partners. In this study, the participants who were interviewed stated that being overweight or 'fat' symbolized beauty, affluence, good health, and a negative HIV status in the isiXhosa culture (Jennings, 2004; Walter and Durandt, 2011). As a result, many women felt that engaging in exercise would lead to inevitable weight loss, and thus preferred to remain sedentary (Walter and Durandt, 2011). Another study by Jackson (2010) that assessed cardiovascular risk in urban, Black working males and females, found that perceived prevalence of obesity in females was significantly lower than actual rates of obesity. In this sample, 93.2% of the females were classified as obese, while only 44.9% perceived this to be the case (Jackson, 2010).

In addition to cultural beliefs, research highlights the low rates of physical activity in populations of lower socioeconomic status; a finding attributed to the financial costs associated with partaking in formalized sports such as membership fees, equipment, and gear (Clark *et al.,* 2009). However, Black males are considerably more active than

Black females on average, suggesting that it is more likely cultural beliefs that influence the prevalence of sedentariness in Black female populations than low socioeconomic status (Walter & Durandt, 2011).

2.5.3 SMOKING

Smoking of tobacco products is a well-established risk factor for the development of cardiovascular disease, and is the leading cause of premature mortality globally (Fodor & Tzerovska, 2004, WHO, 2016). Specifically, it is associated with 71% of all lung cancers, 42% of chronic respiratory disease, and nearly 10% of cardiovascular disease (Shisana *et al.,* 2014). Presently, it is estimated that tobacco accounts for six million deaths per year, and this is expected to increase to 10 million deaths in 2050 (Groenewald *et al.,* 2007; Shisana *et al.,* 2014).

Of particular relevance is that one of the major lifestyle changes associated with urbanization is smoking- the prevalence of which is increasing in low and middle-income countries, particularly in urban areas (Pearson, 1999; Boutayehb, 2006). Research shows a smoking prevalence of nearly 50% in developing countries- a figure also expected to increase by 3.4% per year (Boutayeb, 2006). This trend is evident in South Africa and contributes to the increasing burden of chronic diseases, especially among the Black populations (John *et al.,* 2004).

In 1992, the first national statistics became available, which reported that 32% of all South Africans smoked (Groenewald *et al.,* 2007). It further stated that smoking was more prevalent in males of all population groups (52% of males smoked versus 17% of females) but that smoking rates among Black individuals were higher than those among Caucasians (Sitas *et al.,* 2004). However, notably lower smoking rates were reported for Black females, only 10% of whom reportedly smoked, in comparison to 27% of Caucasian and 59% of Coloured females (Sitas *et al.,* 2004).

In 1998, smoking prevalence across the country was assessed in the national DHS- which provided the first available data for the Eastern Cape (DHS, 1998). Although this data was neither race nor sex specific, it reported that 50% of men within the Eastern Cape smoked, compared to 11% of women. For males, this represented the third highest prevalence rate in the country, while for females, this was the third lowest prevalence rate countrywide (DHS, 1998).

In 2003 however, national statistics reported that smoking prevalence had decreased across the board (DHS, 2003; Groenwald *et al.,* 2007). This was attributed to tobacco control legislation and rapidly increasing taxes making cigarettes simultaneously less desirable, and more expensive (DHS, 2003; van Walbeek, 2005). Between 1998 and 2003, aggregate cigarette consumption decreased by more than a third, with a per capita cigarette consumption reduction off nearly 50% (van Walbeek, 2005). The highest decrease was evident in poorer Black populations, while decreases among wealthier Caucasian populations were less (van Walbeek, 2005). Nevertheless, the majority of this decrease was evident in male populations; females among different race groups showed little change. This is evidenced by the fact that between 1998 and 2003, smoking prevalence in Black and Caucasian female populations remained at 5.1% and 26.7%, respectively (DHS, 2003).

2.5.4 Alcohol consumption

Regular consumption of alcohol can have positive psychosocial and health effects (Cushman 2001; Bradshaw *et al.,* 2007). Several studies have shown that the regular consumption of moderate amounts of alcohol is associated with reduced blood pressure and decreased overall mortality (Castelnuovo *et al.,* 2002; Yusuf *et al.,* 2004). However, excessive alcohol consumption has many adverse effects and is associated with liver cirrhosis and certain cancers, as well as hypertension (WHO, 2000; Bradshaw *et al.,* 2007).

Historically, different patterns of alcohol consumption have been evident in different population groups (Bradshaw *et al.,* 2007). Traditionally, rural Black individuals were known to consume alcoholic drinks made from plants, fruits, and grains, while drinks such as wine and beer were typically consumed by Caucasian individuals (Bradshaw *et al.,* 2007). Due to recent urbanization however, traditional plant-based alcoholic drinks have been replaced with western alcoholic drinks; a transition believed to be partly attributed to the prominence of viticulture in South Africa.

Viticulture has become an integral part of the South African way of life, and is thought to have had a notable impact on drinking behaviours (Walker *et al.,* 2002). The supply of crude wine as part of Black farm workers' wages is thought to have contributed to the culture of heavy drinking that currently exists within the country. This heavy

drinking culture is so prominent that alcohol consumption has been termed 'the country's most abused drug' (Walker *et al.,* 2002).

In 1998, nationwide results on alcohol consumption indicated that 28% of South Africans, accounting for 8.3 million individuals consumed alcohol (DHS, 1998). Within this percentage, prevalence rates were reported to be highest in Caucasian men (71%) and women (51%) and lowest in Black women (12%). Eastern Cape specific findings showed that 47.4% of males and 16.2% of females consumed alcohol (DHS, 1998). In 2003, follow-up results indicated that alcohol consumption had decreased; findings mirrored across all provinces including the Eastern Cape. Interestingly however, reasons behind this decrease were not explored. These findings showed that of women in the Eastern Cape, 89.5% reported abstaining from alcohol within the last 12 months, and only 1.1% reported being harmful or hazardous drinkers; a finding markedly lower than that of males, where 57.6% reported abstaining from alcohol within the last 12 months, and 4.8% reported being harmful or hazardous consumers (DHS, 2003). When comparing results across different race groups, of the women who reported abstaining from alcohol within the last 12 months, 61.3% of these were Black females, and 30.1% were Caucasian (DHS, 2003). These findings are mirrored across other studies, indicating that lifetime abstinence from alcohol use was most common in Black female populations, although the reasons behind this are not thoroughly explored (WHO, 2000; Martinez *et al.,* 2011). However, due to the fact that these statistics are based on self-reports, it is likely that these were underestimated (Schneider *et al.,* 2007).

CHAPTER III - METHODOLODY AND PROTOCOL

3.1 RESEARCH AIM

The aim of this research was twofold: the first aim was to add new data to the body of literature on cardiovascular disease risk in Black and Caucasian females within the Eastern Cape. Secondly, this research aimed to compare the cardiovascular disease risk in urban Black and Caucasian women, by exploring the interaction between race, socioeconomic, and lifestyle factors.

3.2 RESEARCH DESIGN

This research was conducted as a 'cross-sectional design', otherwise referred to as a 'comparative descriptive design'. Herein, the study populations were assessed at one point in time with the aim of determining the prevalence of the outcome of interest (cardiovascular disease risk). The key to this type of design was that the environment within which the population exists remained the same; the researcher merely observed, measured, described, and reported on the prevalence of pre-selected variables naturally existing in the group. Thereafter, inferences on health status and subsequent proposed intervention-areas were further explored if/where necessary.

3.2.1 DEPENDANT AND INDEPENDENT VARIABLES

Various factors such as morphological characteristics, cardiovascular-risk parameters (CV), lifestyle-related factors, and socioeconomic status were assessed in order to provide an indication of the risk for cardiovascular disease. Each of these categories represented a dependant variable of interest for this study: obesity- representing morphological risk; hypertension and blood glucose and insulin concentrations- representing cardiovascular risk parameters (CV); diet, physical activity levels, smoking, and alcohol use- representing lifestyle-related factors; and education and net income- representing socio-economic status. According to these criteria, cardiovascular disease risk profiles of Black and Caucasian women forming an urban working population were compared; thus race and demographic location (urban) represented the independent variables of interest. Both dependent and independent variables are represented below (Table 3).

Table 3: Dependent and independent variables.

	RISK			
URBAN WORKING POPULATION	**MORPHOLOGICAL**	**CARDIOVASCULAR**	**LIFESTYLE**	**SOCIO-ECONOMIC STATUS**
Black	MASS, BMI, WC, WSR,	BLOOD PRESSURE	DIET, PA, SMOKING, ALCOHOL	EDUCATION, NET MONTHLY INCOME
Caucasian				

Where: BMI = Body Mass Index; WC = waist-circumference; WSR: waist-to-stature ratio; PA = physical activity

3.3 WORLD HEALTH ORGANIZATION (WHO) STEP-WISE APPROACH

To assess the cardiovascular risk of these population groups, the WHO STEP-wise approach (STEPS) was used. This is a methodological tool which has been recommended for the assessment and surveillance of chronic disease risk, and is more specifically acclaimed as the gold standard entry level assessment tool for low and middle income countries (Strong and Bonita, 2004; Riley *et al.,* 2016). The key objectives of STEPS are to gather comprehensive information on chronic disease risk factors thereby enabling appropriate implementation of programmes and interventions, and to collect standardised risk factor data to enable comparisons between populations/countries (Riley *et al.,* 2016). As a result, the use of this tool was particularly advantageous as it allowed for comparisons between different racial groups. Additionally, this approach takes into account financial and logistical constraints that often hinder the implementation of objective measures, which was a further benefit within the context of this research. Therefore STEPS was chosen as the most comprehensive, accessible, and valid tool to use in order to achieve the aims of this research.

According to Bonita *et al.* (2001), a risk factor (within the context of chronic disease) is defined as any attribute, characteristic, or exposure of an individual, which increases the likelihood of developing a chronic disease. Using this definition, the STEP-wise approach proposes that risk be assessed according to the following three steps: Step 1, questionnaire-based information; Step 2, standardized physical measurements

(objective measures); and Step 3, blood samples for biochemical analyses (Bonita *et al.,* 2004).

3.3.1 THE USE OF STEPS IN THE CURRENT STUDY

Within the current study, Steps 1, 2 and 3 was utilized in the assessment of each risk factor. However, step 3 was adjusted slightly in accordance with the research aims. These changes, and the justifications behind them, are addressed below. A summary of the techniques used within the STEPS framework is presented:

Table 4: STEPs framework for the assessment of selected cardiovascular disease

RISK	STEP 1: Questionnaires	STEP 2: Physical measurements	STEP 3: Blood samples
Diet	• Three-day dietary recall. • Selected questions on habitual intake (Adapted from Adult Health Questionnaire, 2003)		
Physical activity	• Adapted Global Physical Activity Questionnaire (GPAQv2).		
Tobacco use	• Adapted WHO STEP-wise surveillance questionnaire		
Alcohol use and dependency	• Standard questionnaire for the monitoring of alcohol consumption and related hard (WHO STEP-wise)		
Obesity		• Stature, mass and waist circumference (from which BMI and waist-to-stature ratio will be calculated). • Bioelectrical impedance analysis (BIA)	
Hypertension	• Self-reporting of hypertension	• Measured blood pressure	
Type II Diabetes	• Self-reporting of type II diabetes		• Fasting glucose and fasting insulin concentrations (HOMA-1)
Hypercholesterolemia	• N/A	• NA	• NA
Socio-economic status	• WHO STEP-wise questionnaire on 'expanded demographic information'.		

The two sub-sections that were altered for the purpose of this study were the replacement of fasting glucose concentrations alone for fasting glucose and insulin concentrations, and the absence of hypercholesterolemia measures.

3.3.1.1 Blood glucose concentrations

Recent literature suggests that Black African populations do not typically present with elevated blood glucose levels as frequently as in other populations (Goedecke *et al.,* 2009). The proposed explanation for this lies in a genetic predisposition to hyperinsulinemia within Black African populations, whereby excessive levels of insulin are secreted for a prolonged period of time in order to maintain safe blood glucose levels (Goedecke *et al.,* 2009). As a result, blood glucose measures alone often depict an inaccurate picture of glucose metabolism in these populations, thereby compromising the validity of the measure. For this reason, it was therefore decided that the relationship between fasting glucose and fasting insulin levels would be a more valid measure to take when assessing the biochemical health of Black Africa populations. Therefore both fasting glucose and fasting insulin concentrations were chosen to be measured in this study.

3.3.1.2 Hypercholesterolemia

Evidence exits to show that hypercholesterolemia is becoming an outdated and inaccurate indicator of cardiovascular disease risk (Brunzell *et al.,* 2008). Despite its popularity in the past, recent evidence suggests that a more accurate measure is one that includes the ratio of HDL, LDL and triglyceride concentrations to total cholesterol concentrations (Sharma *et al.,* 2009; Goedecke *et al.,* 2015). Because of this, measures of total cholesterol only (which indicates the presence or absence or hypercholesterolemia) were excluded from this study as it is unethical to subject participants to a test which is shown to be inconclusive (Brunzell *et al.,* 2008). Although it would be beneficial to measures HDL, LDL and triglyceride concentrations within the context of this study, it is an expensive test to administer and thus was not used due to financial constraints.

3.4 ETHICS

Ethical clearance was obtained from Rhodes University Human Kinetics and Ergonomics Department Ethical Standards Committee for research involving human participants (Appendix 1).

3.5 MORPHOLOGICAL RISK

3.5.1 OBESITY

Various measurements exist pertaining to the assessment of obesity. Most commonly, these include stature, body mass (from which body mass index is calculated), and waist circumference (which in conjunction with stature, is used to calculate waist-to-stature ratio). Despite the ease of use of the above measures, alone, they often provide an incomplete and rather crude account of obesity risk (Neville *et al.,* 2006; Daniels, 2009). Therefore, a more comprehensive assessment requires measures of body composition in addition. These include bioelectrical impedance analyses, skinfolds analysis, and hydrostatic weighing.

3.5.1.1 Body Mass Index (BMI)

The use of BMI is a well-known measurement is assessment of fatness in relation to stature (Daniels, 2009). Despite its biggest limitation of not being able to distinguish between fat mass and lean body mass, it is still accepted as one of the standard measures of obesity, particularly when used to assess sedentary populations (Flegal *et al.,* 2013). One of the main benefits of using BMI is that it provides a quick and easy indication of body fat percentage, which is particularly applicable in field based research. By using both body mass and stature values, BMI therefore provides some indication of the linearity of 'fatness' in relation to height (Baumgartner and Jackson, 1998).

The BMI classification system proposed by WHO (2004) provide guidelines for identifying and classifying overweight and obese individuals (Table 5).

Table 5: WHO classification of body mass index (WHO, 2004).

BMI ($kg.m^{-2}$)	Classification	CVD Risk
< 18.5	Underweight	Low
18.5-24.9	Normal	Average
25.0-29.9	Overweight	Increased
30.0-34.9	Obese class I	High
35.0-39.9	Obese class II	Severe
≥ 40.0	Obese class III	Very severe

3.5.1.2 Waist Circumference (WC)

Despite the health risks associated with obesity in general, central obesity has been shown to have more adverse consequences than peripheral obesity (Després *et al.,* 2008; Tsatsoulis *et al.,* 2013). Central obesity is defined as the accumulation of fat around the abdomen, and is associated with high risk of hypertension, type II diabetes, and cardiovascular disease (Alberti *et al.,* 2005; Tsatsoulis *et al.,* 2013). It is primarily measured as waist-circumference (WC), but additional measures include waist-to-hip ratio (WHR) and waist-to-stature ratio (WSR) (Shields *et al.,* 2012). Waist circumference classifications do vary among different population groups, however the guidelines put forth by WHO based on several global studies provide a useful initial screen for risk profiles of individuals (Table 6).

Table 6: WHO classification of waist circumference (WHO, 2004).

Waist circumference (cm)		
	Risk	High Risk
Males	≥94	≥102
Females	≤80	≤88

Due to the controversy in the literature with respect to race-specific waist circumference norms, and more specifically, the lack thereof, the above classifications serve as mere guidelines. Nevertheless, as waist circumference is accepted as a primary indicator of cardiovascular disease risk, it is an important measure to take in

the context of this study. In order to account for possible misrepresentation of obesity within the Black female population however, an additional measure of waist-to-stature ratio was done.

3.5.1.3 Waist-to-Stature Ratio (WSR)

Waist-to-stature ratio, otherwise known as 'waist-to-height ratio', is a particularly useful measurement when looking at populations who are typically shorter or taller than the standard height averages worldwide (Goedecke *et al.,* 2009). In the case of Black African women, the average height is 8 -10 cm shorter than the average height of Caucasian females (Goedecke *et al.,* 2009). This is one of the reasons that the use of standardised waist circumference norms may not accurately reflect obesity in certain populations, as the differences in average height are not accounted for. For example, a blanket waist-circumference norm of ≥88 cm (indicative of high risk for cardiovascular disease in a woman) may be of *greater* risk to a Black African woman who is typically shorter than an age-matched Caucasian woman. In other words, the shorter woman would have a higher waist circumference proportionately to the taller woman, therefore putting her at a higher risk. It is for this reason that waist-to-stature ratio was a particularly useful and applicable ratio to use in the context of this study.

3.5.1.4 Body Composition: Bioelectrical impedance analysis

There are three widely accepted and commonly used methods for determining body composition, namely bioelectrical impedance analysis, skinfolds analysis, and hydrostatic weighing (Pecoraro *et al.,* 2003). For the purpose of this study, bioelectrical impedance analysis was chosen for reasons discussed below.

Bioelectrical impedance analysis (BIA) is defined as the opposition of a conductor to the flow of an alternating current and involves the application of a 50kHz current through the body via electrodes which are place on the hands and feet (Dehghan and Merchant, 2008). This technique introduces a painless current (800μA) into the body at a frequency of 50 kHz in order to determine the electrical impedance of body tissues. As lean body tissue is rich in water and electrolytes, it poses minimal impedance. In contrast, adipose tissue high in fat poses high impedance and therefore increases impedance to a maximum (Dehghan and Merchant, 2008). Due to this, lean body

mass and fat mass can be calculated from the difference in conductivity though the use of the following formula:

$V = p \times S^2/R$

Where:

V = conductive volume (assumed to represent total body water or fat free mass)

P = specific resistivity of the conductor

S = stature (an estimate of length of the conductor)

R = whole body resistance (measured with four surface electrodes placed on the wrist and ankle)

(Taken from Saladino, 2014)

Bioelectrical impedance analysis is a non-invasive and relatively inexpensive technique for the measurements of body composition. Additionally, due to its portability, it is ideal for field based research (Kyle *et al.,* 2004). Although skinfolds analysis is also portable measure and is considered more accurate in normal to overweight populations, this technique is invasive and provides inaccurate results in obese populations (Goedecke *et al.,* 2009). This is due to the fact that skinfold callipers can only open to a set wideness that is often too narrow to grip the largest areas of fat at certain sites in obese populations (Amaral *et al.,* 2011). As the prevalence of obesity in Black African female populations, it is likely that a large proportion of the participants will be obese, and thus the use of this technique was rejected.

Nevertheless, despite the practical benefits, bioelectrical impedance analysis is not without short-comings, as it is still considered the least accurate method of the above mentioned three (Savastano *et al.,* 2010). The compromised accuracy here is typically due to the effects that body water content have on the measurements, whereby body composition before and after water ingestion can often produce different values of lean and fat body mass (Savastano *et al.,* 2010). However, by ensuring that all participants are measured after having not eaten or drunk for 10 hours prior, the possible interaction effect of this confounding variable is largely diminished. Bioelectrical

impedance analysis is also a widely used and established method for determining body composition, and was therefore an acceptable selection for use in this study.

3.6 CARDIOVASCULAR RISKS

3.6.1 HYPERTENSION

Accurate and precise measurement of blood pressure is paramount in classifying and identifying hypertension-related cardiovascular disease risk (Pickering *et al.,* 2005). The auscultatory technique, which involves a trained observer using a mercury sphygmomanometer and the Korotkoff sound technique, represents an accurate 'gold-standard' blood pressure measurement technique (Ogedegbe and Pickering, 2010). Although this is a manual technique which is subject to human error, it is still considered accurate and precise in comparison to automated blood pressure machines, providing that the user is properly trained and experienced in using the equipment (Ogedegbe and Pickering, 2010). In addition, the use of manual blood pressure measurement also considerably reduces the risk of equipment malfunction or error; an inherent limitation of automated blood pressure machines.

South African blood pressure norms differ from American standardized norms in that a blood pressure <129/84 mm Hg is still considered 'normal', whereby this is considered as 'pre-hypertensive' according to American norms (South African Heart and Stroke Foundation, 2014). Nevertheless, the globally-accepted cut-off value for Stage 1 hypertension is >140/90 mm Hg, and will thus be used as the basis of classification for hypertension. Any pre-hypertensive readings will be classified according to the South African norms due to the demographic of the sample groups in this study.

Table 7: Hypertension classification (South Africa Heart and Stroke Foundation, 2016).

SYSTOLIC BP (mmHg)	DIASTOLIC BP (mmHg)	CLASSIFICATION
120-129	80-84	Normal
130-139	85-89	Pre-hypertensive
140-159	90-99	Stage 1 hypertension
≥160	≥100	Stage 2 hypertension

BP indicates 'blood pressure'; (mmHg) indicates millilitres of Mercury.

3.6.2 INSULIN SENSITIVITY

Insulin resistance (characterized by impaired insulin sensitivity and hyperinsulinemia) is a key factor contributing to type II diabetes in Black African populations (Goedecke *et al.,* 2010).

The standard technique for the assessment of insulin sensitivity is the hyperinsulinemic euglycemic clamp (Keskin *et al.,* 2005). This technique, often used in combination with the hyperglyceric clamp, determines the adequacy of compensatory β-cell hypersensitivity by periodically introducing small concentrations of glucose and insulin into the blood at regular pre-determined intervals over a set time duration (Keskin *et al.,* 2005). Although typically considered the gold-standard technique, it is also complex and laborious technique that is too invasive for general epidemiological studies, as well as for any field study evaluating a large number of participants (Keskin *et al.,* 2005; Minamino *et al.,* 2009). In light of this, an established and widely used surrogate measure of insulin resistance, the Homeostasis Model Assessment Index (HOMA-I) will be used for this study.

The HOMA-I approach has been widely used in clinical research to assess insulin sensitivity, and works by applying a set formula to fasting glucose and insulin levels in order to determine and index indicating level of insulin sensitivity (or resistance) (Keskin *et al.,* 2005; Minamino *et al.,* 2009). Rather than looking purely at fasting insulin and fasting glucose levels in isolation, the HOMA-I approach uses the product of the two and then divides it by a constant (40.5 if mg/dL or 22.5 if mmol/l). This calculation compensates for fasting hyperglycaemia and generates an index for insulin sensitivity (Keskin *et al.,* 2005). The index classification is presented below in Table 8.

Table 8: HOMA-I Classification for insulin sensitivity (Keskin *et al.*, 2005).

HOMA INDEX	
Normal insulin function (high sensitivity)	< 2.0
Borderline (compromised insulin sensitivity)	2.1 - 2.5
Moderate (insulin resistance)	2.6 - 3.0
High (sever insulin resistance)	>3.0

An example of HOMA-I application:

Fasting glucose: 5.2 mmol/l

Fasting insulin: 12.2 µU/ml[3]

(5.2 x 12.2) / 22.5

= 2.82 (Moderate insulin resistance)

3.7 LIFESTYLE RISKS

3.7.1 DIET

Habitual diet, and in particular, an unhealthy habitual diet, remains one of the strongest predictors of cardiovascular disease risk factors such as obesity, hypertension, and type II diabetes (Bouyateb, 2006; Steyn and Nel, 2006). Various techniques exist for the assessment of habitual diet, the most common of which being food frequency questionnaires, a 24-hour dietary recall, and a three-day dietary recall (Nel and Steyn, 2002). The primary difference between these methods is that the food frequency questionnaires require participants to recall and foods and beverages consumed over

[3] µU/ml = µIU/ml = mIU/L

a longer period of time (days, weeks, or months), whereas the 24-hour dietary recall and the three-day dietary recall method (as the names suggest), requires consumption to be recalled over a shorter period, either 24 hours or 72 consecutive hours.

Despite the inherent limitation of only recording consumption over a 24-hour to 72-hour period, these methods are beneficial when compared to food frequency questionnaires in that they are quick, simply, and an easy for participants to adhere to (De Keyzer *et al.,* 2015). For the purpose of this study, a three-day (72-hour) dietary recall was used as it was more likely to provide an accurate representation of habitual diet than a once-off 24-hour dietary recall. These recalls were administered so that participants include two weekdays and one weekend day in order to get as accurate a representation of a normal full week as possible.

Studies have also shown that the three-day dietary recall method to compare well with the results of food frequency questionnaires, particularly when repeated measures are used, and was therefore chosen as an appropriate alternative for this study (De Keyzer *et al.,* 2015; Thompson *et al.,* 2015). Due to logistical and time constraints, repeated measures were not possible for this study, however, in addition to carrying out a 24-hour food recall, additional questions regarding the habitual intake of various foods and beverages will be carried out. These questions are validated and based on similar questions in the Adult Health Questionnaire used in the 2003 Demographic Health Survey (DHS, 2003). This is added to strengthen the reliability of the dietary intake data so to ensure that dietary recollection is as accurate as possible.

3.7.2 PHYSICAL ACTIVITY

The association between physical activity and cardiovascular risk is well established, and the lack thereof is an ever-increasing global concern (Blair, 2009; WHO, 2010; Walter and Durandt, 2011) The regular participation in physical activity, recommended at 30 minutes of moderate intensity exercise, five days a week, is associated with a substantial reduction in cardiovascular disease risk (Whitt-Glover *et al.,* 2012). In contrast, individuals who take part in little to no physical activity have increased cardiovascular disease risk (Kohl *et al.,* 2012).

There are various methods pertaining to the measurement of physical activity, both objective and subjective. Previously, exercise was more strictly defined as 'planned,

structured exercise sessions' (such as sports or jogging) with the specific aim of improving physical health (Lambert *et al.,* 2007). Thus, objective measures such as calorimetry, the Double Labelled Water (DLW) technique, pedometers, and accelerometers were typically used to assess energy expenditure during these activities (Lambert *et al.,* 2007). More recently however, it has been recognized that physical activity is not exclusive to structured exercise; it occurs in many other domains of life such as work and travel. Thus, self-reported measures of time spent engaging in these activities have become useful in the assessment of total physical activity levels (Lambert *et al.,* 2007). Physical activity questionnaires have therefore become increasingly popular tools to use, as they have the added advantages of being inexpensive, and easy to apply and administer within a field-based setting (Pitta *et al.,* 2006).

Various validated physical activity questionnaires have been developed, such as the Minnesota Leisure Time Physical Activity Questionnaire (MLTPAQ); the Zutphen Physical Activity Questionnaire (ZPAC); the Stanford Seven-Day Physical Activity Questionnaire; the International Physical Activity Questionnaire (IPAQ); the Global Physical Activity Questionnaire (QPAQ), and more. Of these, the GPAQ was developed by the World Health Organization as an instrument to assess physical activity patterns in developing countries, and was used as part of a larger project aimed at monitoring risks for chronic diseases in developing countries (Armstrong and Bull, 2006). Research was carried out testing the reliability and validity of the GPAQ in a large number of countries, particular those with low levels of education (including South Africa). From this, the final GPAQ version 2 instrument (GPAQv2) was developed and was deemed a suitable physical activity surveillance instrument in developing countries (Armstrong and Bull, 2006). The GPAQv2 has since been incorporated within the WHO STEP-wise approach model and was thus chosen as an acceptable method for this study.

The GPAQv2 comprises of 16 questions grouped to capture physical activity undertaken in different behavioural domains namely work, transport, and leisure (Singh and Purohit, 2011). The main outcome variables from GPAQv2 analysis are a categorical variable of total physical activity (low, moderate, or high); and a continuous variable of total physical activity within each domain (reported as median METmins.week^{-1}) (Armstrong and Bull, 2006).

3.7.3 ALCOHOL CONSUMPTION

Alcohol abuse is widely accepted as a contributing risk factor to the development of cardiovascular disease (Chopra *et al.,* 2002; Schneider *et al.,* 2007). For this reason, it is imperative to assess alcohol consumption in order to compare and relate consumption to cardiovascular disease risk factors. The WHO STEP-wise questionnaire is a comprehensive and appropriate tool for the assessment of habitual alcohol intake, and encompasses the frequency of alcohol use as well as past and present consumption practises (Bonita *et al.,* 2001). Moreover, the alcohol consumption in the latest National Demographic and Health Survey (2003) was assessed using questions based on the WHO STEP-wise questionnaire, thus validating the applicability of this test on a South African population (DHS, 2003).

3.7.4 TOBACCO USE

As with alcohol consumption, the habitual use of tobacco is linked to a high risk of cardiovascular disease and therefore requires assessment (Bonow *et al.,* 2002; DHS, 2003; Myer *et al.,* 2004; WHO, 2011).

In 1998, the first Demographic and Health Survey was carried out in South Africa and this provided a platform for the assessment of tobacco use within different population groups and within different provinces (DHS, 1998). The tobacco-related questions in this survey were based on and derived from the WHO guidelines for controlling and monitoring of the tobacco epidemic (Bradshaw and Steyn, 2001; Swart and Panday, 2010). In the 2003 Demographic Health Survey however, these initial questions were adapted to form a more rigorous assessment based on the WHO STEP-wise programme. To date, the tobacco-use questionnaire within the STEP-wise approach is still commonly used and widely accepted for assessing chronic disease risk and was thus chosen for this study.

3.8 SOCIO-ECONOMIC STATUS

Defined as a measure of an individual's social position in society based on education, income, and occupation, socio-economic status is well documented factor affecting cardiovascular disease risk (Winkleby *et al.,* 1992; Stringhini *et al.,* 2013; Kivimäki *et al.,* 2014). More specifically, poor socio-economic status has been linked to increased risk of cardiovascular disease, and this is attributed in large to lack of education

surrounding health lifestyle choices, and financial constraints limiting access to adequate health care (Stringhini *et al.,* 2013; Kivimäki *et al.,* 2014).This is of particular relevance to a South African context where socio-economic disparity is so high (evidenced, in part, by a Gini Coefficient of 63.4 in 2015), and where the majority of those from poor socio-economic backgrounds are Black African populations (The World Bank, 2016). It is therefore essential to assess the influence of socio-economic status on health in Black South African populations as it is such central feature to their demographic.

The WHO STEP-wise programme contains a section pertaining to the demographic information of individuals, and includes socio-economic questions based on employment, income level, and education level. As this programme is a widely accepted and used tool for developing countries, it was therefore used in study.

3.9 EXPERIMENTAL PROCUDRES: EQUIPMENT AND MEASURMENTS

Due to factors not within the control of the primary researcher, glucose and insulin measures, as well as body fat distribution measures were not able to be done in the study as initially planned. Subsequently, methods pertaining to these measures were removed from the procedures section of this chapter below. These limitations are addressed in further detail under 'Limitations to the study', pg. 63).

3.9.1 ANTHROPOMETRIC AND MORPHOLOGICAL RISK

3.9.1.1 Stature

Stature was be measured to the nearest millimetre (mm) using a stadiometer (Harpenden Stadiometer, Chasmors Ltd, London). Before measurement commences, participants were required to remove their shoes and stand upright facing forwards while allowing three points of their body to be in contact with the back of the stadiometer, namely the back of the head, gluteus maximus, and calcaneus. The measurement was taken from the floor to the vertex in the mid-sagittal plane.

3.9.1.2 Body Mass

Body mass was measured to the nearest 0.1 kg using a calibrated electronic scale (Toledo® Trek Scale Co, Model 1842, Cleveland). Participants were required to

remove their shoes and any heavy clothing or jackets in order to obtain accurate results.

3.9.1.3 Body Mass Index (BMI)

BMI ($kg.m^2$) was calculated from stature and body mass values using the following formula: body mass (kg) / stature (m) ^{2}

3.9.1.4 Waist Circumference (WC)

Iliac waist circumference was measured using a standard dressmaker's tape measure and was recorded to the nearest millimetre (mm). This measurement was performed with the participant standing upright, at the end of normal expiration, and after having removed any heavy outer garments (Janssen *et al.,* 2004).

3.9.1.5 Waist-to-stature Ratio (WSR)

The waist-to-stature ratio was calculated by dividing waist circumference (cm) by stature (cm). This calculation elicited a ratio which was then used as a classification system for cardiovascular disease risk, based on the Ashwell Shape Chart© (Ashwell, 2011).

Table 9: Waist-to-stature ratio.

INDEX VALUE	CLASSIFICATION	COLOUR REGION
<0.4	Low ('take care')	Brown
0.4 – 0.49	Normal ('ok')	Green
0.5 – 0.59	Moderate ('take care')	Yellow
>0.6	High ('action')	Red

The data above is taken from the Ashwell Shape Chart (Figure 3), demonstrating the cut-offs visually.

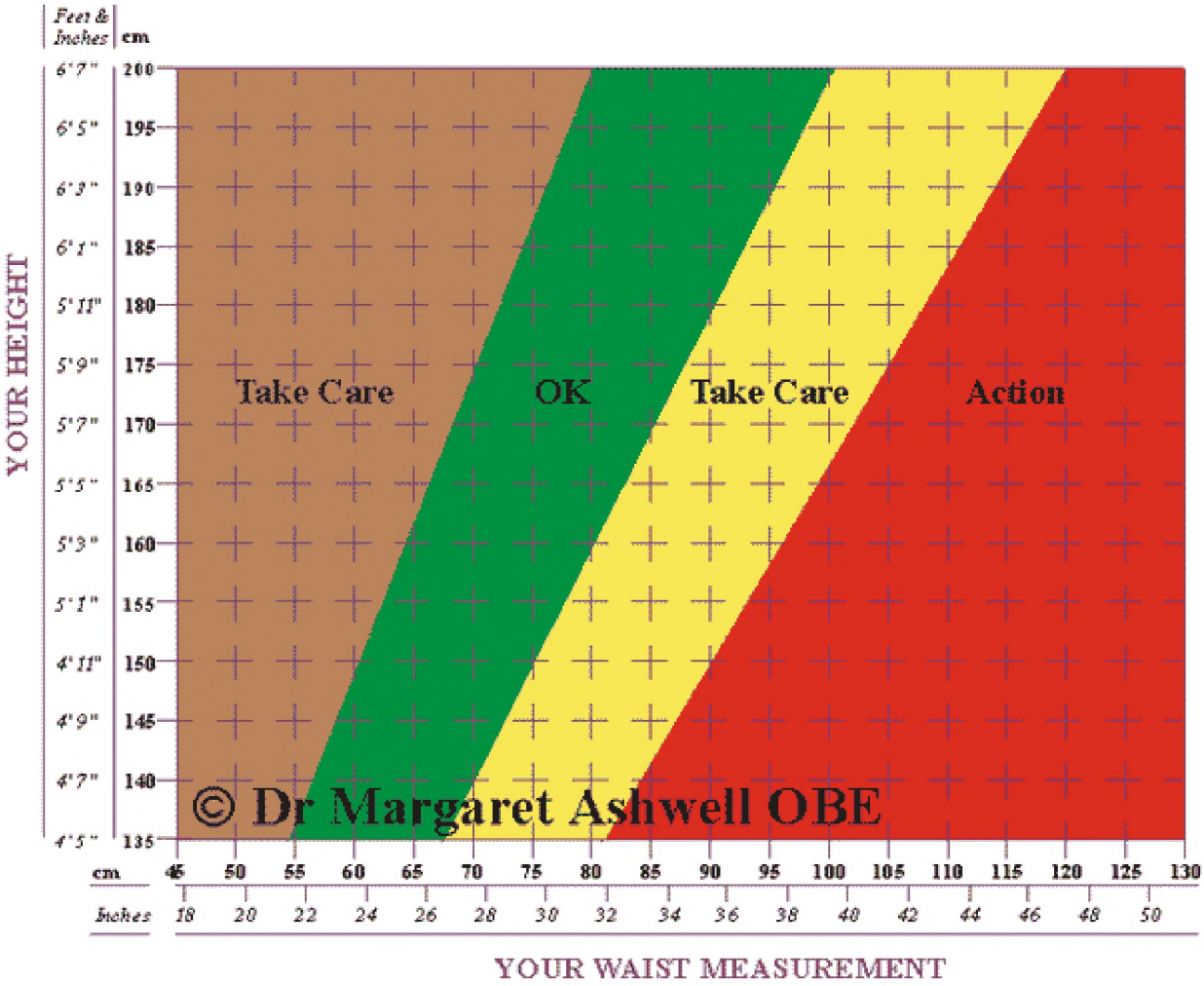

Figure 3: The Ashwell Shape Chart based on waist-to-stature ratio.

3.9.2 CARDIOVASCULAR RISK

3.9.2.1 Blood Pressure

Blood pressure (mm Hg) was measured in triplicate by the principle researcher using a manual sphygmomanometer (Baumanometer® Sphygmomanometer, W.A. Baum Co., Inc., New York) with two minutes separating the three measurements. Participants were required to rest quietly in a seated position for five minutes prior to the measurement. The blood pressure measurements were taken according to the auscultatory method (Chobanian *et al.,* 2003), by applying a sphygmomanometer cuff of appropriate size to the left brachial artery. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded at the first and fifth Korotkoff sounds and the two closest readings will be averaged and recorded.

3.9.2.2 Medical Conditions Questionnaire

In addition to the physical measurement of blood pressure, glucose and insulin levels, self-reported hypertension and insulin resistance or type II diabetes were also recorded. This was done by means of structured questions in which the individuals were asked if they have ever been told by a health professional that they were hypertensive/had high blood pressure, and/or if they have ever been told they have insulin resistance or type II diabetes.

3.9.3 LIFESTYLE RELATED RISKS

3.9.3.1 Diet

3.9.3.1.1 Three-day food recall

A three-day food recall was used in order to assess the habitual dietary habits of the participants over a consecutive 72-hour period. This method required participants to record everything they consumed (food and liquid) over a period of 72 hours, which was filled in on a blank template provided (Appendix 9). Thereafter, an interview was carried out during which the participants were required to verbally confirm specific quantities and types of foods consumed. Visual aids of various portion-size references were also included to assist with understanding, explanation, and accuracy of food quantities recorded (Appendix 11).

3.9.3.1.2 Dietary Intake Questionnaire

Individuals will also be asked various additional questions regarding the type of food stuffs typically consumed, and how often these food stuffs are consumed. These question are included to gain a deeper understanding of the types of foodstuffs habitually consume, and thus serves to strengthen the accuracy of dietary assessment. The dietary intake questionnaire is taken from the WHO STEP-wise programme.

3.9.3.1.3 Food Finder

Dietary intake and macronutrient composition for each participant as analysed using the Medical Research Council (MRC) FoodFinder 3™ software programme for Windows® (Microsoft Corporation). This contains a South African Food Composition

Database (SAFOODS) which is the official database on the nutrient composition of foods eaten in South Africa.

3.9.3.2 Physical Activity

3.9.3.2.1 Global Physical Activity Questionnaire version 2 (GPAQv2)

Individuals were asked various questions on their habitual physical activity levels using the GPAQv2. This questionnaire included questions related to activity at work, activity involved in getting to and from places, and activity related to recreational activities. This therefore aimed to provide an overall indication of physical activity, taking into account the different lifestyle domains, as well as duration and intensity of physical activity.

Physical activity levels were also categorised according to a scoring system in the volume of activity is computed by weighting each type of activity by its energy requirements. This was computed using METs (multiples of the resting metabolic rate), to derive a score in MET-minutes: calculated by multiplying a MET score by minutes performed. 1 MET-minute represent 3.5 ml of oxygen per kg of body weight per minute (GPAQv2). Data was therefore collected as a continuous measure and reported as median MET-minutes for walking (W); moderate-intensity activities (M); and vigorous-intensity activities (V), within each domain (work, transport, and leisure). Physical activity was calculated using the following MET values:

Table 10: Domain-specific MET classification (GPAQv2).

DOMAIN	MET VALUE
Work	Moderate MET value = 4.0
	Vigorous MET value = 8.0
Transport	Walking MET value = 4.0
Leisure	Moderate MET value = 4.0
	Vigorous MET value = 8.0

Physical activity levels were defined according to the definition proposed by the International Physical Activity Questionnaire (IPAQ) Scoring Committee, which defines overall reported physical activity levels as follows:

Table 11: Classification of levels of physical activity (adapted from IPAQ classification of physical activity).

CATEGORY	CRITERIA
1. Low/inactive or insufficiently active	< 600 MET-minutes/week
2. Moderate/Minimally active	600– 3000 MET-minutes/week
3. High/Sufficiently active	> 3000 MET-minutes/week

3.9.3.3 Tobacco use

3.9.3.3.1 STEP-wise questionnaire

The WHO STEP-wise questionnaire contains questions pertaining to past and current smoking habits, as well as exposure to environmental tobacco smoke at home and at work, and exposure to dust and fumes. Participants were asked these questions in an interview format and the primary research and/or research assistants filled in their answers.

Smokers were classified as individuals who report that they currently smoke either occasionally or daily, and were further classified as light smokers (1 – 14 tobacco equivalents per day) or heavy smokers (≥ 15 tobacco equivalents per day) where applicable (Bradshaw and Steyn, 2001). One tobacco equivalent is defined as one manufactured cigarette; one hand-rolled cigarette; one pipe; one cigar; one cheroot; or one cigarillo (Bradshaw and Steyn, 2001).

3.9.3.4 Alcohol consumption

3.9.3.4.1 Adapted questions for the assessment of alcohol use (WHO STEP-wise)

Alcohol use was assessed by means of a set of questions based on those used in the DHS (2003), and adapted from the WHO STEP-wise programme. Through the use of these questions, information was obtained regarding the frequency of consumption, as well as the quantities of alcohol consumed each day during a typical week. Individuals were considered 'risky drinkers' if the consumed over three drinks per day (in the case of females) (Schneider *et al.,* 2007).

3.10 EXPERIMENTAL PROCEDURES

3.10.1 PHASE 1

As the central aim of this project was to assess cardiovascular risk in Black and Caucasian females within the Eastern Cape, the priority was to find enough participants residing in the greater urban area of Grahamstown who were both willing and able to participate in the study. As this was a comparative-descriptive study design which was non-repeated, the aim was get as many participants as possible. Looking at similar studies conducted in South Africa, the majority of these are large scale studies involving 900 to 5000 + participants (Vorster *et al.,* 2002; Steyn *et al.,* 2005; Motala *et al.,* 2008; Motala *et al.,* 2011; Murphey *et al.,* 2014). However, three studies have included 200 to 300 participants in total, with no fewer than 100 participants per comparative group (Schutte and Olckers, 2007; Jennings *et al.,* 2008; Jackson *et al.,* 2010). The nature of this study was such that the sample group would be on a smaller scale, therefore guidelines from smaller scale studies were used. A sample size of 200 to 300 participants, with approximately equal numbers of Black and Caucasian women, was therefore chosen for this study. This was to ensure that the study was statistically viable, while also practically achievable with respect to availability of eligible participants, and time.

Therefore, in order to gather as many participants as possible, the initial stage of this study involved contacting local businesses and institutions within the Grahamstown area. Where possible, meetings were set up with managers and supervisors where the aims of the project, the requirements for participation, as well as the benefits of participating were communicated (Appendix 4). A mother-tongue Xhosa speaking individual who assisted in the data-collection phase of this study was present at the meetings in order to provide translation if necessary. Here, any questions that the supervisors had were answered and clarified. The supervisors were then be asked to distribute the project information to their employees.

The second stage involved an information session with interested employees (as gathered by the supervisors) where the research project aims and procedures were explained in detail by the primary researcher, as well as by the isiXhosa translator where necessary. Participants were also told that if they wished, they would be allowed to bring some food and drink with them to the testing session which they could

consume directly after the physical measurements have been taken. This was to optimize their comfort, as they would have fasted for 10 hours prior. Participants were then recruited on a voluntary basis, and were required to sign a consent form after thoroughly reading the information letter and completing the pre-screening questionnaire. Where necessary, follow-up meetings were held where logistics with regards to when testing commencement were discussed.

Once all interested participants had signed-up and have selected a day and time slot for testing, this marked the transition into phase 2.

3.10.2 PHASE 2

On arrival, each participant was introduced to the researcher, research assistants, nursing sister, and translator, and were then assigned their participant code. Thereafter, participants were verbally reminded of the purpose of the study, as well as the testing procedures they were about to undergo. They were also assured that if at any point they wished to pull out from the study, they were welcome to do so. As all participants were female, only female research assistants were conducting the measurements to ensure that the participants felt as comfortable as possible. Furthermore, each participant was measured in a different area of the department to ensure their privacy.

Once the participants felt ready to begin, the researcher and/or research assistants began with the physical measurements. While the physical measures were being taken, the participants were given verbal feedback about the values they obtained, and any questions they had about these values were answered. After the physical measures were taken, the participants were given a few minutes to relax before commencing the questionnaire portion of the study.

The questionnaires on self-reported CVD risk, tobacco use and alcohol consumption, socio-economic status, dietary habits, and physical activity habits were then administered and completed. Although there was only one translator present, the translator was called when/if needed by the research assistants and could therefore assist with more than one individual's testing session when necessary. Once the questionnaires were completed, the participants were thanked for their time and reminded of their participant codes, which would be used for feedback purposes.

3.11 DATA ANALYSES

Statistical analyses were conducted using STATISTICA version 13 (Statistica©, Statsoft Inc). Descriptive statistics were carried out initially in order to obtain mean and standard deviation responses for all variables. Independent T-tests were then conducted on all variables to determine any significant differences between Black and Caucasian females. Cohen's d formula was then used to determine the effect size of all variables. Correlational analyses were also carried out to assess for any significant relationships between different variables. All statistical hypothesis tests were set at a 5% level of significance.

3.12 LIMITATIONS TO THE STUDY

Various limitations of this research project reduce the accuracy and applicability of the data obtained. The following factors should be taken into consideration:

Due to time and financial constraints, testing a large number of individuals was not possible. The sample size in the current study was relatively small, and was therefore not encompassing and entirely representative of the Makana district of the Eastern Cape. Therefore, conclusions drawn from this sample specifically may not be indicative of trends for the whole province, or country as a whole.

Due to the small geographical size of the Makana region, selecting a narrow age range would have substantially reduced the number of participants viable for participation. Therefore, a broader age range encompassing women of adult reproductive age (18-50 years) was chosen. However, due to the notable effect of age on cardiovascular disease risk, this was a limitation as the age effect was not accounted for.

The use of self-reporting questionnaires required participants to answer as honestly as possible. Every effort was made to emphasize the need for honesty and accurate reporting, and participants were reminded that their responses would remain confidential, however, this could not have been objectively controlled. Thus, it is possible that this may have affected the accuracy of the data. Despite this limitation however, it is accepted that physical activity questionnaires and other self-report instruments such as food recall diaries remain the most practical when assessing larger samples in epidemiological studies (Richardson *et al.,* 2001).

Of the 166 participants in the study, only 95 participants returned completed food recall diaries (46 Caucasian and 49 Black). This greatly reduced the accuracy of the dietary results, particularly with respect to correlations between dietary data and other factors. As a result, correlations with dietary data were excluded from analyses.

Language barriers between the researcher and participants may have hindered the accuracy of the results obtained. The primary researcher and research assistants were English mother tongue speakers, while the Black participants were all Xhosa mother tongue speakers. Despite ensuring that all participants were able to read and understand basic English, the extent of English language proficiency was not ascertained, therefore likely impacting the accuracy of data obtained. Nevertheless, the use of a Xhosa-speaking interpreter was made, who was present and assisted during all testing sessions. Questionnaires were also translated into Xhosa in an attempt to overcome language barriers.

Due to financial constraints, glucose and insulin concentrations were not able to be assessed in this study. Thus, insight into insulin resistance and type II diabetes prevalence in this study was not ascertained.

Due to equipment malfunction, it was not possible to conduct Bioelectrical Impedance on participants in this study. Therefore body fat percentage was not measured.

CHAPTER IV – RESULTS

Risk factors have been grouped into three categories: 1) obesity, classified as 'morphological risk'; 2) hypertension classified as 'cardiovascular (CV) risk'; and 3) diet, physical activity, smoking and alcohol use, classified under 'lifestyle-related risks'.

4.1 DEMOGRAPHIC INFORMATION

Table 12: General demographic and socioeconomic characteristics of participants.

	Caucasian	Black
Age (years)	33.88 (± 12.19)	39.67 (± 11.79)
Gross monthly income (ZAR)	R17242.31 (± 10834.93)**	R4710.61 (± 2671.84)**
Education (years)	17.05 (± 2.80)**	11.66 (± 2.10)**

(**) denotes significant difference ($p < 0.01$)

In addition to being urban working individuals, participants were age-matched with no significant difference *($p>0.05$)* between groups (Table 12). However, there were significant differences *($p< 0.01$)* with both education levels and gross monthly income between groups. Caucasian women in this sample reported a gross monthly income 3.66 times higher than the Black women *($d=1.69$).* Despite this, as well as a larger standard deviation within the Caucasian cohort however, coefficients of variation (62.84% and 56.72% respectively) indicate that the disparity in earnings within both groups is similar.

Caucasian women in this sample are also significantly *($p<0.01$)* more educated than the Black females, having spent an average of 17.05 (± 2.80) years in formal education as compared to 11.66 years (± 2.10) *($d=2.21$)* (Table 12).

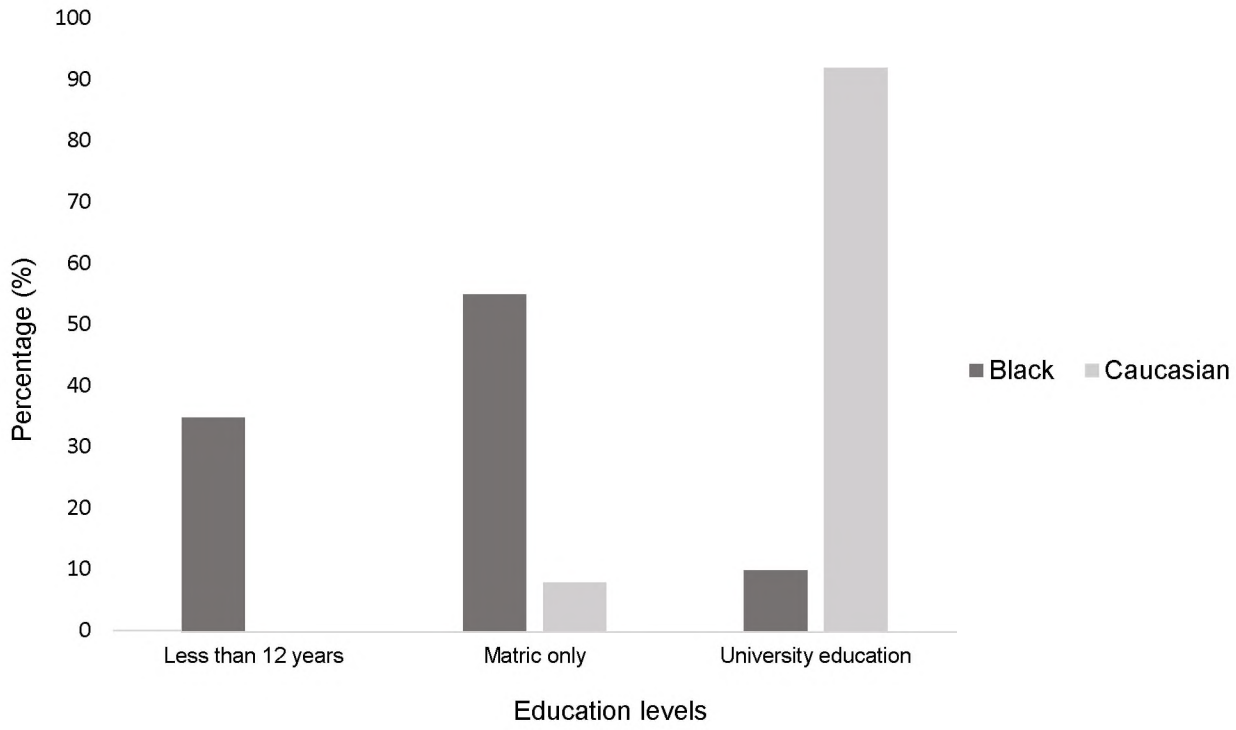

Figure 4: Levels of completed education (%).

Of the Caucasian females, 92% completed tertiary education with the average number of years spent in formal education being 17.05 years (± 2.80). This equates to approximately 12 years of schooling and five years of Tertiary education, with no participant having completed less than 12 years of formalized schooling. Black women, on the other hand, spent an average of 11.66 (± 2.10) years at school, with only 65% completing 12 years of formalized schooling. Of this 65%, only 10% went on to study at University. The remaining 36% did not complete high school.

4.2 MORPHOLOGICAL RISK

4.2.1 OBESITY

4.2.1.1 Stature, mass, Body Mass Index (BMI)

With the exception of stature, Black women demonstrated significantly *($p<0.01$)* higher values for Mass, BMI, waist circumference, and waist-to-stature ratio than Caucasian females (Table 13).

Table 13: Mean (±SD) anthropometric and morphological characteristics of participants.

	Black	Caucasian
Stature (cm)	156.74 (± 6.18)	164.55 (± 6.36)**
Mass (kg)	84.16 (± 21.62)	70.15 (± 13.44)**
BMI (kg.m^2)	34.19 (± 8.07)	25.90 (± 4.67)**
WC (cm)	95.34 (± 16.77)	79.84 (± 10.81)**
WSR	0.61 (± 0.10)	0.48 (± 0.09)**

(**) denotes significant difference ($p < 0.01$)

Black women were significantly *($p<0.01$)* heavier than their Caucasian counterparts *($d=0.95$)* and subsequently had significantly higher BMI values *($d=1.35$).* The BMI classification categorizes the Caucasian women as 'overweight' while the Black women are classified as 'Obese class I (Figure 5).

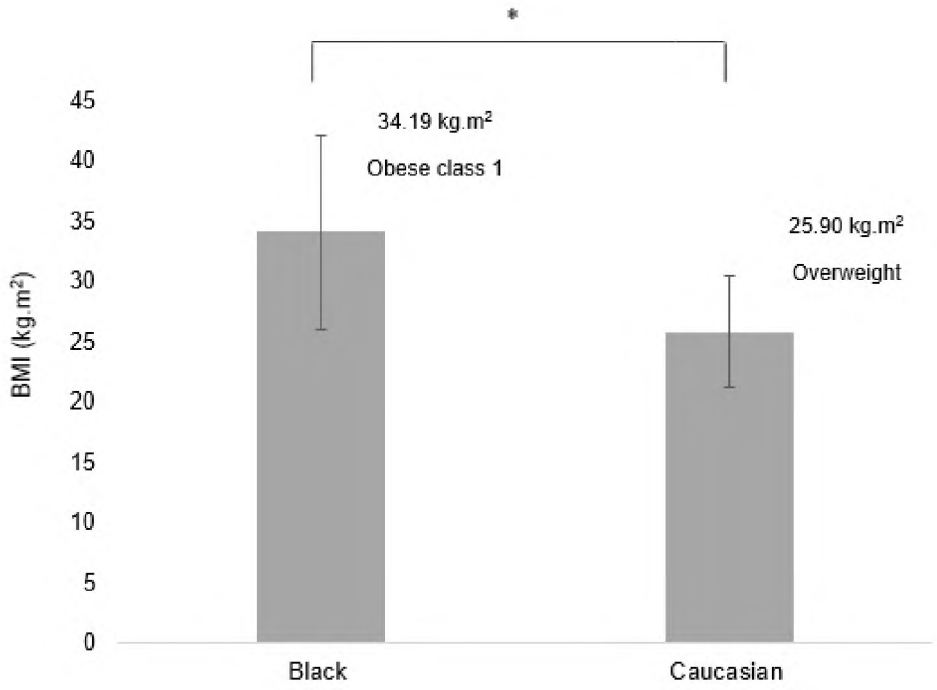

Figure 5: Mean (±SD) BMI classification.

Noteworthy is that both groups lie close to the extremes of their respective BMI classification brackets. Black women, with a mean BMI of 34.19 kg.m^2, fall just short of the 'Obese II' classification which begins at 35.00 kg.m^2. Similarly the Caucasian women, with a mean BMI of 25.90 kg.m^2, are just short of the 'normal' category which ends at 24.99 kg.m^2.

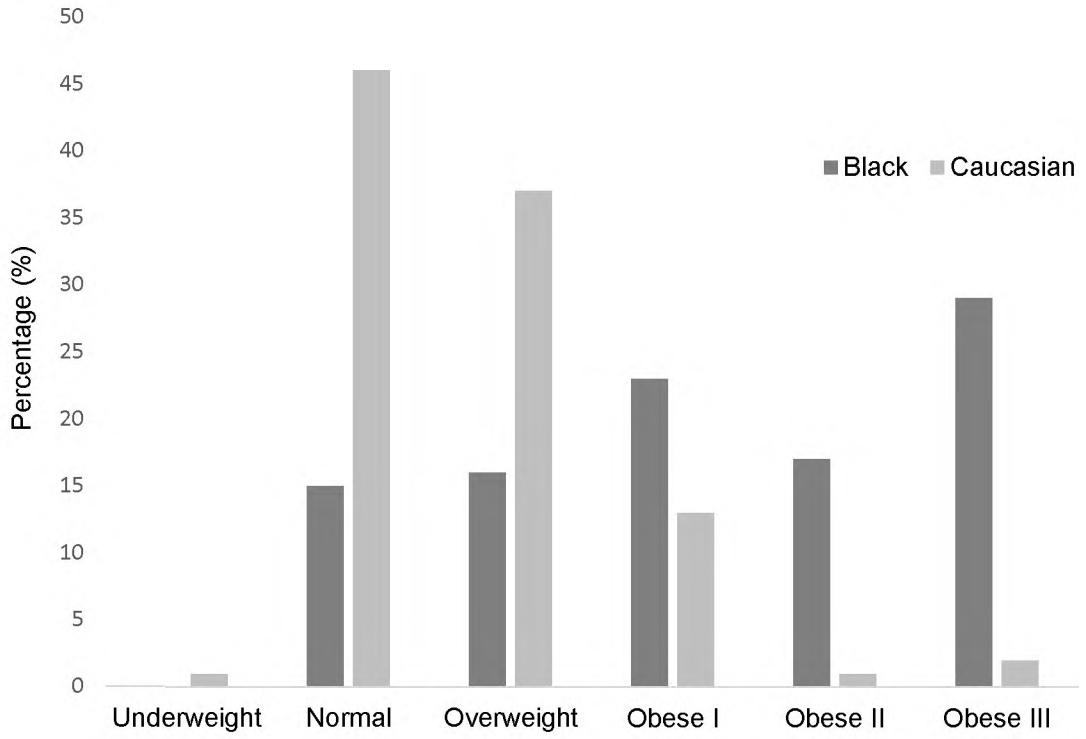

Figure 6: Percentage of participants falling within different BMI classifications.

Figure 6 represents the distribution of participants falling within the six different BMI classification categories. In the Caucasian group, the majority of participants fell within the 'normal' category, followed by 'overweight' and 'obese I'. The Black group, however, had the majority of participants fall within the 'obese III' category, followed by 'obese I', and 'obese II'. This highlights the inherent limitations of relying on mean data when individuals vary so greatly.

4.2.1.2 Waist circumference (WC) and waist-to-stature ratio (WSR)

Black women had significantly *(p<0.01)* larger waist circumferences than the Caucasian women, with a mean of 95.34 cm (± 16.77) compared to 79.84 cm (±10.81) *(d=1.09).*

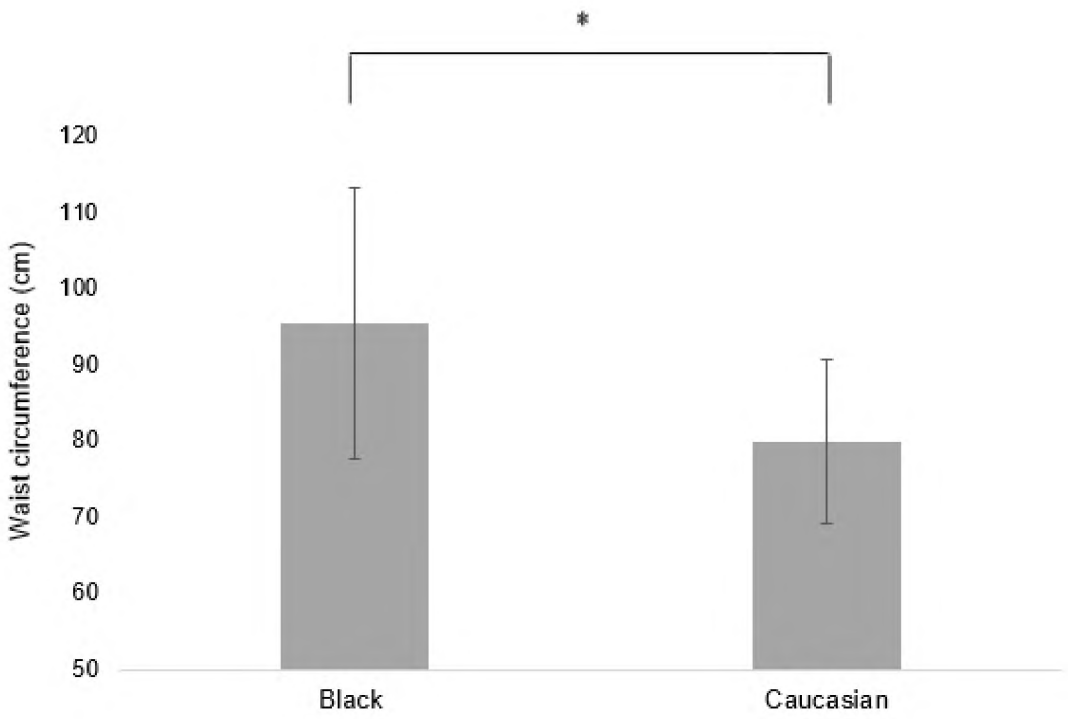

Figure 7: Mean (±SD) waist circumference of both groups.

With respect to waist circumference (WC) 'cut-offs', the Caucasian group are categorized as 'not at risk' while the Black women are classified as being at 'high' risk for cardiovascular disease. As with the relationship between weight and BMI, this measure similarly contributes to the significantly *(p<0.05)* higher WSR in the Black South African group (0.61) as compared to the Caucasian group (0.48). The Caucasian women are classified as 'healthy' (0.42 – 0.48) while the Black women are classified as being at 'high risk' for cardiovascular disease.

Both waist circumference and waist-to-stature results indicate that the majority of weight is centrally distributed in the Black group.

4.3 CARDIOVASCULAR RISK

4.3.1 BLOOD PRESSURE

Systolic (SBP), Diastolic (DBP) and Mean Arterial Blood pressure (MAP) measures were significantly *($p<0.05$)* higher in the Black compared to the Caucasian group (*$d = 0.40$; $d = 0.50$; $d = 0.41$*, respectively). Nevertheless, with respect to blood pressure classifications, both groups presented with measures within the 'normal' range (Table 14).

Table 14: Mean (±SD) blood pressure measures.

	Black (n = 88)	**Caucasian (n = 79)**
SBP (mm Hg)	122 (± 18)*	116 (± 12)*
DBP (mm Hg)	77 (± 16)*	70 (± 11)*
MAP (mm Hg)	92 (± 16)*	86 (± 11)*

(*) denotes significant difference ($p < 0.05$)

When isolating cases of 'known hypertension', 18% of the Black women reported previously known hypertension diagnoses, compared to 6% in the Caucasian cohort. Of these, all reported taking blood pressure lowering medication during the duration of this study. As a result, total mean blood pressure measures must be considered in this context.

The effect of participants taking hypertension medication during this study is noticeable, and not surprising as the efficacy of hypertension medication is well documented (Gu *et al.,* 2012).

Table 15: Mean (±SD) blood pressure measures.

	Black ALL (n = 88)	Black (B-WHD) (n = 72)	Caucasian ALL (n = 79)	Caucasian (C-WHD) (n = 74)
SBP (mm Hg)	122 (± 18)*	130 (± 15)**	116 (± 12)*	115 (± 12)**
DBP (mm Hg)	77 (± 16)*	83 (± 14)*	70 (± 11)*	69 (± 11)
MAP (mm Hg)	92 (± 16)*	98 (±14)*	86 (± 11)*	85 (± 11)

(**) denotes significant difference ($p < 0.01$); (*) denotes significant difference ($p<0.05$); WHD = without hypertension diagnosis

Table 15 shows that among participants without hypertension diagnoses (WHD), the Black group again present with significantly *($p<0.01$)* higher measures compared to the Caucasian group. In addition, the B-WHD group also present with significantly *($p<0.05$)* higher measures compared to the entire (n = 88) Black group, placing this sub-sample at increased cardiovascular disease risk (SBP *d=0.92*; DBP *d=1.21*; MAP *d=1.08*). The Caucasian group, however, show no significant difference when comparing the entire groups' data to the reduced sample (C- WHD) (Figure 7). According to established South African norms, the B-WHD group classifies as 'pre-hypertensive' while the C-WHD group remain 'normal'.

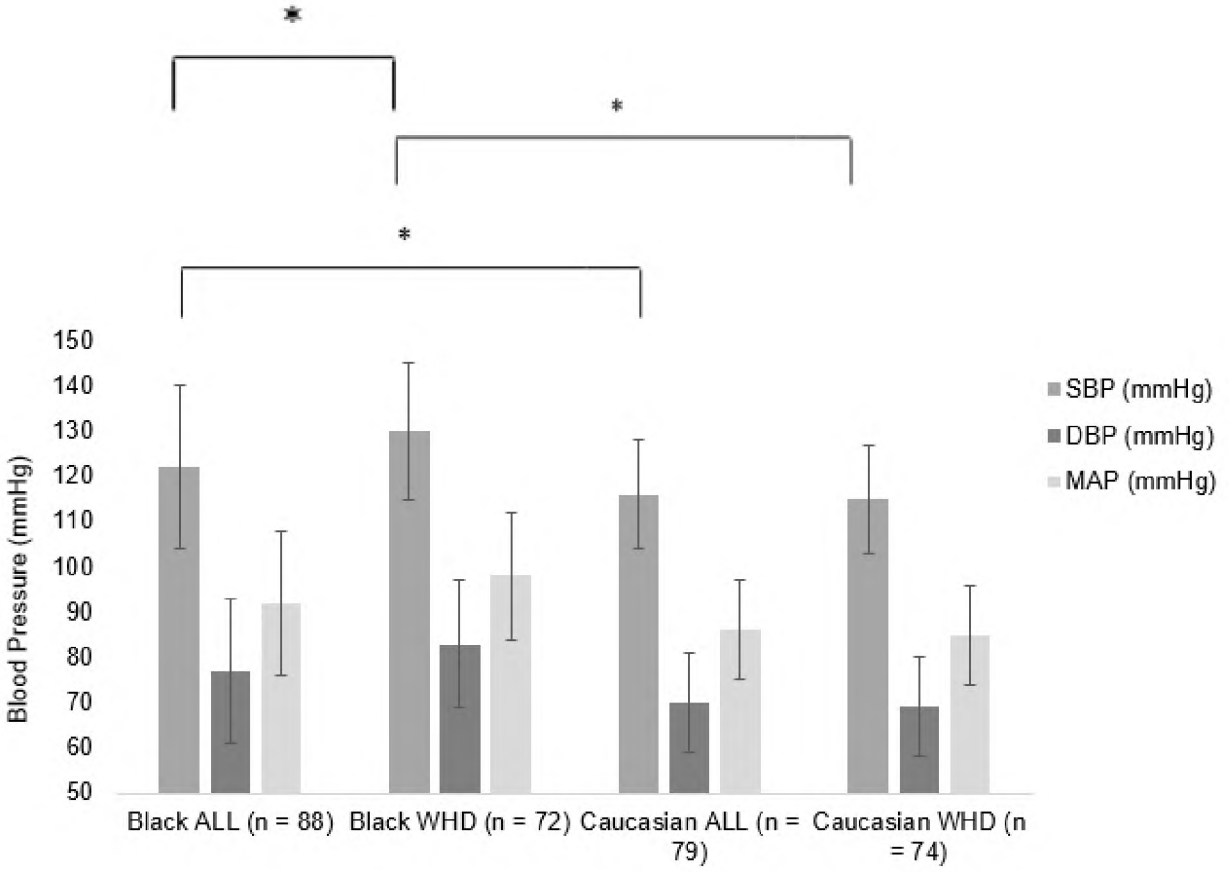

Figure 8: Mean (±SD) blood pressure of participants with and without previous hypertension diagnoses.

4.4 LIFESTYLE-RELATED CHARACTERISTICS

Black females presented with significantly higher *($p<0.05$)* self-reported physical activity levels and total energy intake per day (KJ) compared to the Caucasian sample. Caucasian females however, presented with significantly higher *($p<0.05$)* self-reported cases of alcohol use and smoking (Table 16).

Table 16: Self-reported lifestyle-related risks

Measure	Caucasian Females	Black Females
Active MET-minutes/week	1828.27 ± 2238.10	2688.86 ± 1486.56*
Diet: Total energy intake (KJ/day)	6401.19 ± 1796.47	7243.60 ± 1977.44*
Alcohol consumption (g/day)	10.42 ± 19.21	0.92 ± 3.90**
Current smokers (%)	14	3*

(*) denotes significant difference ($p < 0.05$); (**) denotes significant difference ($p<0.01$); kJ = kilojoules

4.4.1 PHYSICAL ACTIVITY

4.4.1.1 Total active MET-minutes/week

Black females were significantly *($p<0.05$)* more active than the Caucasian females, taking part in a mean total of 2688.86 (±1486.56) MET-minutes of activity per week as compared to the Caucasian mean total of 1828.27 (±2238.10) MET-minutes per week *($d=0.46$).* However the high standard deviations, particularly in the Caucasian group, indicate large variation in baseline physical activity levels between groups.

According to the GPAQ physical activity classifications, 'sufficient' physical activity is defined as expending ≥3000 MET-minutes/week. Within the Black group, 45% reported being sufficiently active, while only 23% of the Caucasian group met this criterion. In contrast, 41% of the Black group reported being 'minimally' active (600 – <3000 MET-minutes/week), while 47% of Caucasian women reported being minimally active. Insufficient physical activity, classified as <600 MET-minutes/week, was higher in the Caucasian cohort (29% versus 13%) (Figure 8).

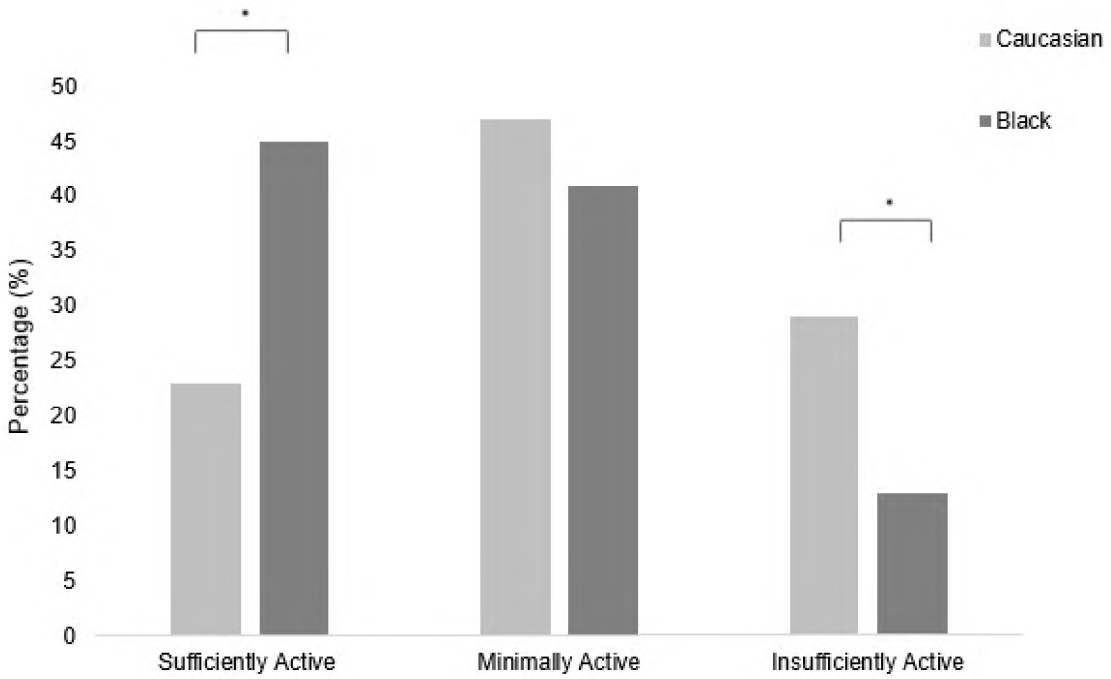

Figure 8: Percentage of participants falling in GPAQ categories

Figure 8 represents the distribution of participants falling within the GPAQ activity classification categories. In the Caucasian group, the majority of participants fell within the 'minimally active' group, while in the Black group, the majority fell within the 'sufficiently active' group, closely followed by the 'minimally active' group'. Between groups, there was a significantly *($p<0.05$)* greater percentage of 'sufficiently active' participants in the Black group (45%) compared to the Caucasian group (23%). Similarly, there was a significantly *($p<0.05$)* greater percentage of 'insufficiently active' participants in the Caucasian group (29%) compared to the Black group (13%). There was no significant difference found between the percentages of 'minimally active' participants in both groups.

4.4.1.2 Transport-related physical activity

Of the total mean active MET-minutes/week, the Black group reported a significantly *($p < 0.01$)* higher percentage attributed to walking to and from work compared to the Caucasian group *($d=0.69$).* In the Black group, 15% of total activity was attributed to

walking to and from work (415.68 ± 588.21) while only 5% was attributed to walking to and from work in the Caucasian group (95.13 ± 271.28).

Within the Black group, 45% of participants reported walking for more than 10 minutes continuously to get to and from work every day, while 55% reported using motorized transport. In the Caucasian group, 15% reported walking to and from work every day while 85% reported driving to and from work (Figure 9).

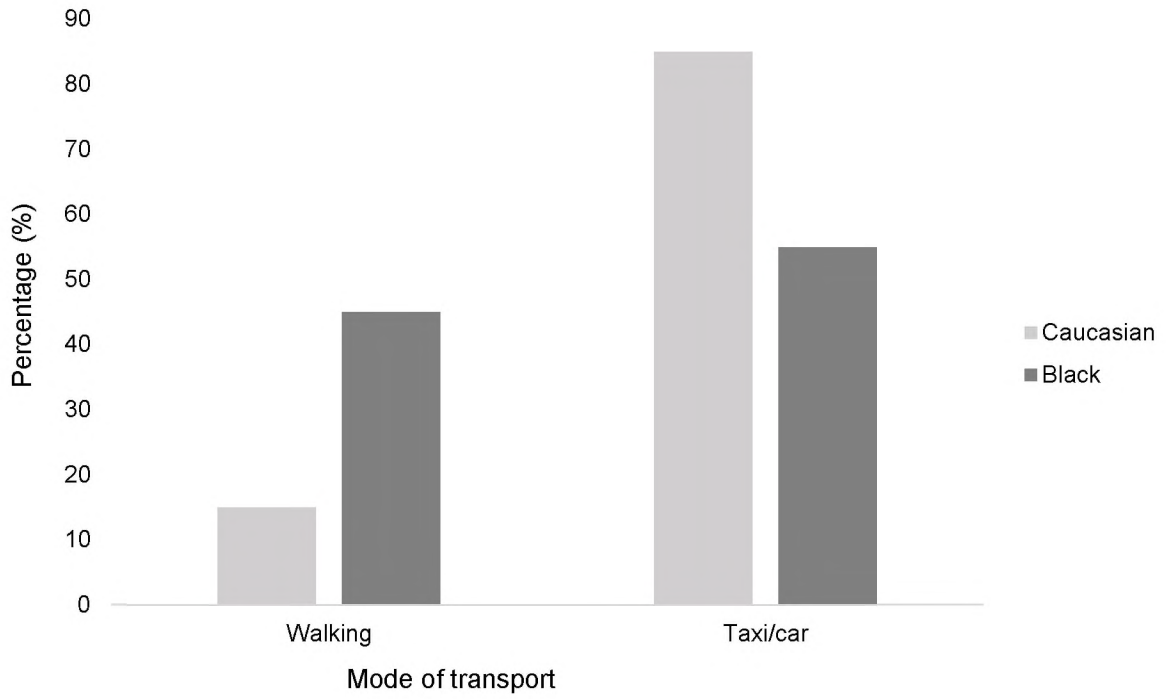

Figure 9: Adoption of transport modes.

Despite the preference for motorised transport in both groups, a significantly *(p<0.01)* larger percentage of total active MET-minutes/week were attributed to walking in the Black group compared to the Caucasian group (45% and 15%, respectively).

4.4.1.3 Seated MET-minutes

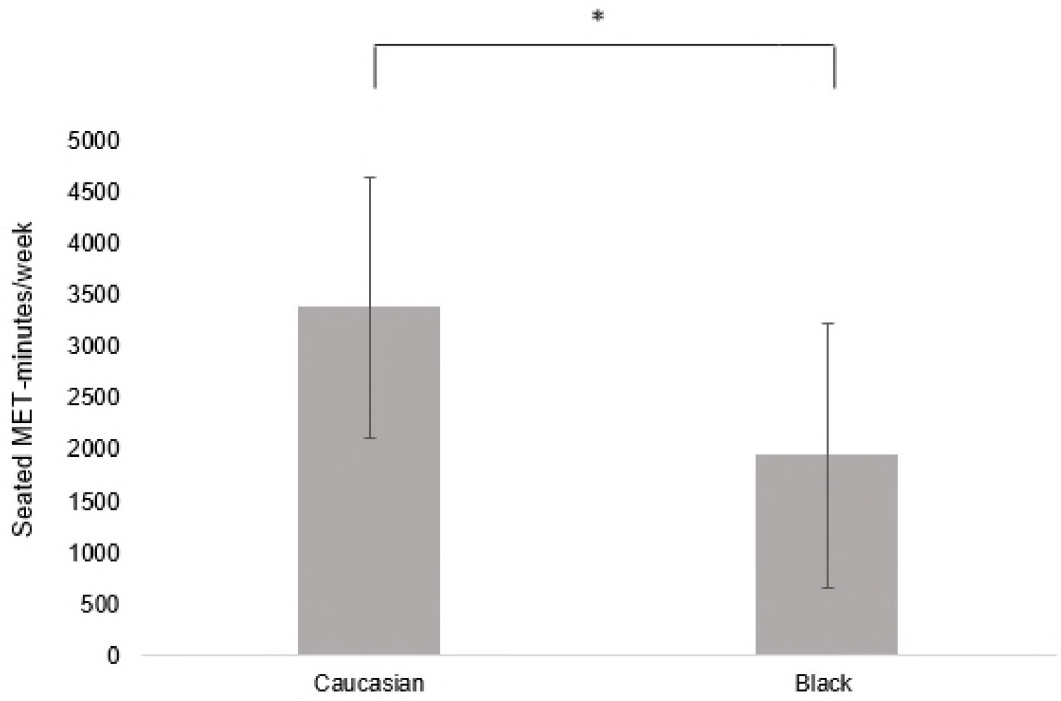

Figure 10: Mean total seated MET-minutes/week.

Caucasian females spent significantly *(p<0.05*) more time sitting than the Black females, reporting a mean total of 3384.05 (±1264.12) seated MET-minutes/week versus 1952.05 (±1286.73) seated MET-minutes/week *(d=1.12)* (Figure 10). Large standard deviations however, indicate variance within both samples.

4.5 DIET

Black females consumed significantly *(p<0.05)* more kilojoules (7243.60 ± 1977.44) per day than the Caucasian females (6401.19 ± 1796.47) *(d=0.45)* (Figure 9).

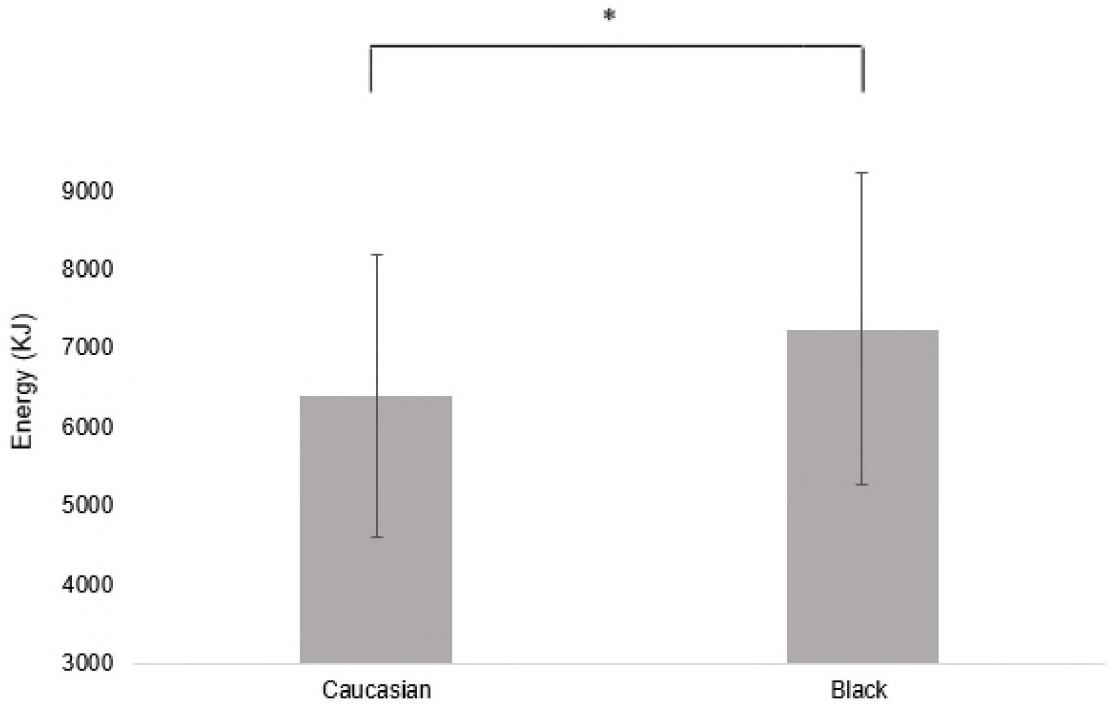

Figure 11: Mean (±SD) total energy intake (kJ) per day.

Figure 11 shows the significant difference in mean kJ intake between groups per day. Both groups reported consuming less than the standard recommendation for females of 8,700kJ (or 2000cal) per day.

When analysing macronutrient composition, carbohydrates (CHO) were shown to contribute the bulk of total dietary intake in both groups (Figure 12).

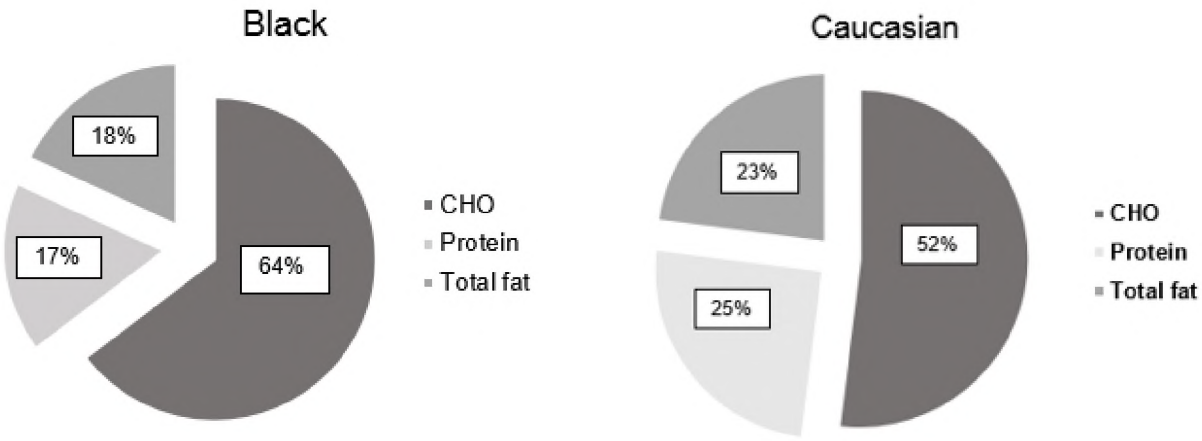

Figure 12: Macronutrient composition.

Figure 12 shows that for both groups, over half of the daily macronutrient intake was composed of carbohydrates. However in the Black group, the carbohydrate intake approximates two-thirds of total intake, while in the Caucasian group it approximates half of total intake (64% and 52%, respectively). The remaining intake was divided almost equally between protein and fat in the case of both cohorts.

In addition to consuming significantly more total kilojoules and carbohydrates (d=1.05), Black females consumed significantly *(p<0.05)* more polyunsaturated fat (*d=0.70*) and total sugar (*d=0.55*), while Caucasian females consumed significantly *(p<0.05)* more total protein (*d=0.46*) and trans fats (*d=0.52*) (Table 17).

Table 17: Mean (±SD) daily consumption of selected dietary components.

	Caucasian	Black
TOTAL PROTEIN (g)	69.42 ± 29.65	58.45 ± 17.30*
CARBOHYDRATES (CHO) (g)	141.82 ± 55.60	214.93 ± 80.32**
TOTAL FAT (g)	64.26 ± 22.65	61.73 ± 21.24
Saturated fat (g)	22.23 ± 9.03	18.44 ± 8.10
Monounsaturated fat (g)	21.38 ± 9.48	19.08 ± 7.11
Polyunsaturated fat (g)	12.35 ± 6.75	17.49 ± 7.95*
Trans fat (g)	1.61 ± 1.28	1.06 ± 0.80*
TOTAL SUGAR (g)	35.02 ± 15.00	46.72 ± 24.14*

(*) denotes significant difference ($p<0.05$); (**) denotes significant difference ($p<0.01$).

These differences in polyunsaturated and saturated fat consumption between groups is likely associated with the types of cooking fats predominantly used within each group (Figure 13).

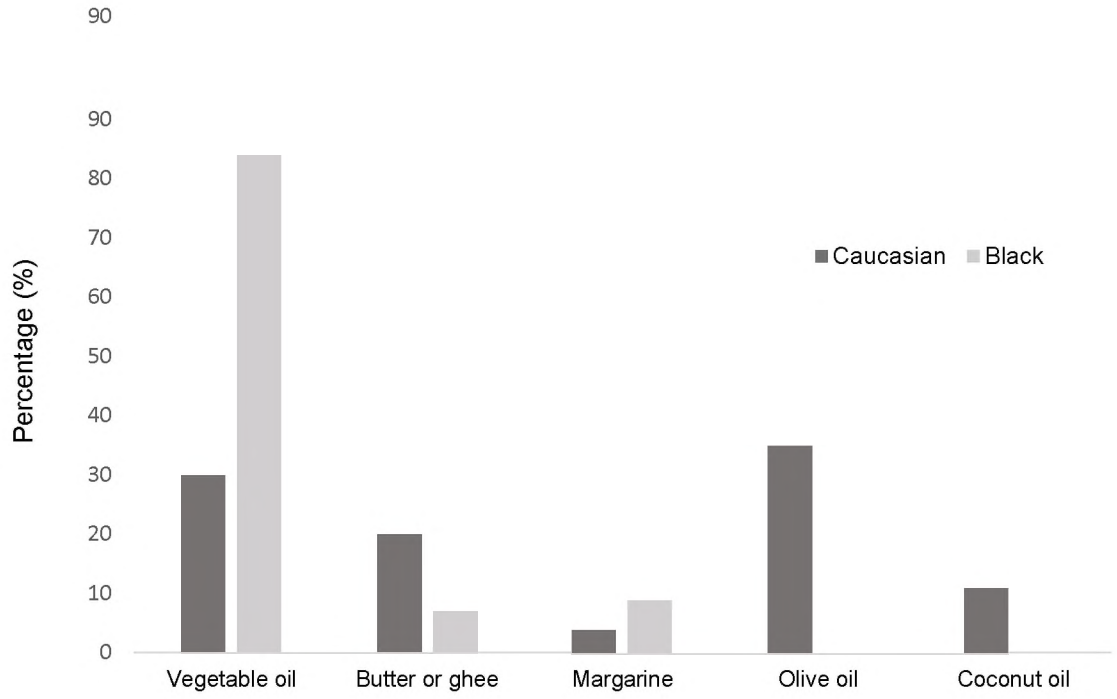

Figure 13: Cooking fat preferences.

Figure 13 shows that the majority of the Black females reported using vegetable oil as their primary cooking fat (85%). This is followed by 8% who reported using margarine and 6% who reported using butter. In the Caucasian sample however, more of a distribution is evident. The majority of participants in this group reported using olive oil as their primary cooking fat (35%), and this is closely followed by the use of vegetable oil (30%). The remaining participants reported using butter (20%), coconut oil (11%), and only 4% used margarine.

Table 18: Classification of cooking fats according to chemical structure.

	Polyunsaturated fats		**Monounsaturated fats**	**Saturated fats**	
	Vegetable oil	Margarine	Olive oil	Butter and ghee	Coconut oil
Caucasian	34%		35%	31%	
Black	93%		0%	7%	

When classifying these five household cooking fats as 'monounsaturated fats', 'polyunsaturated fats', and 'saturated fats', it is evident that Black women used polyunsaturated fats almost exclusively (93%), while the Caucasian women did not show such a distinct tendency (Table 18).

4.6 SMOKING AND ALCOHOL CONSUMPTION

4.6.1 SMOKING

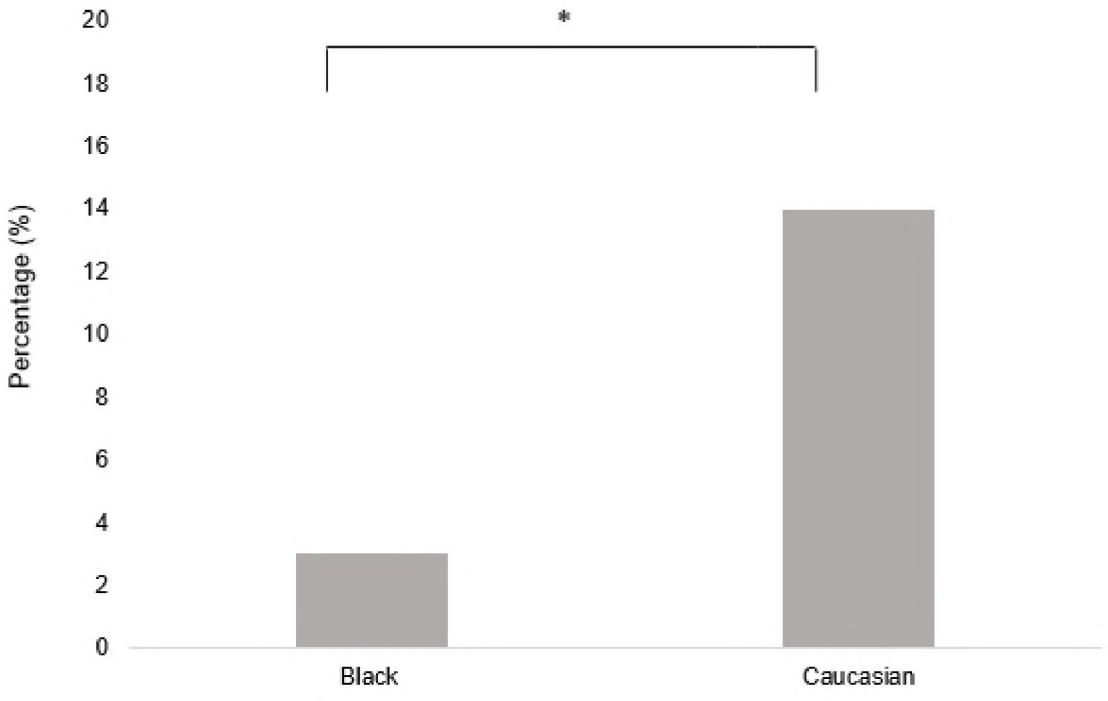

Figure 14: Percentage of participants in both groups who currently smoke.

Significantly *(p<0.05)* more Caucasian females reported being smokers (14%) than Black females (3%). Nevertheless, the majority of the participants in this study were non-smokers.

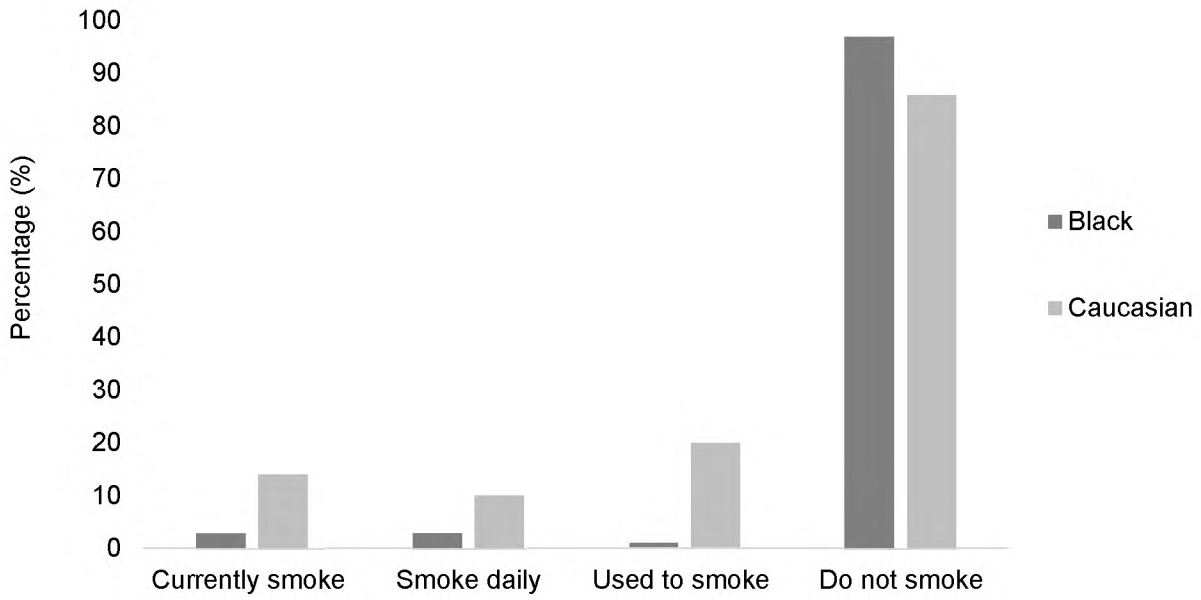

Figure 15: Past and current smoking habits.

Figure 15 shows that while there were more current and past smokers in the Caucasian group, the majority of both groups reported being non-smokers. As a result, it is unlikely the smoking is a prevalent contributor cardiovascular disease risk in these sample groups.

4.6.2 ALCOHOL CONSUMPTION

Alcohol consumption was significantly *($p<0.05$)* higher in the Caucasian sample (10.46g ± 19.21) compared to the Black sample (0.92g ± 3.90) (*$d=0.73$*). (Figure 16).

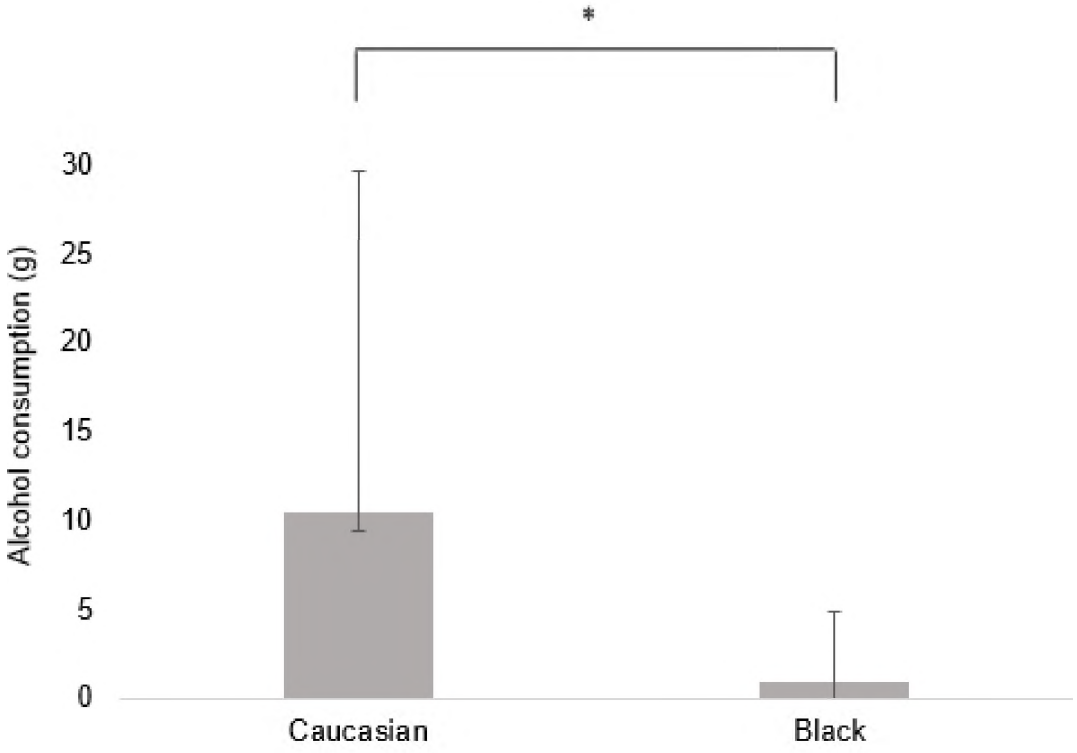

Figure 16: Mean (±SD) alcohol consumption (g) per day.

Figure 16 shows that the Caucasian sample consumed significantly *($p<0.05$)* more alcohol (g) per day, however the standard deviations indicate large variance within and between both samples.

In addition to consuming more alcohol per day, the majority (91%) of the Caucasian sample reported consuming alcohol within the last 12 months while surprisingly, in the Black sample, the majority (51%) reported having never consumed any alcoholic beverage (Figure 17).

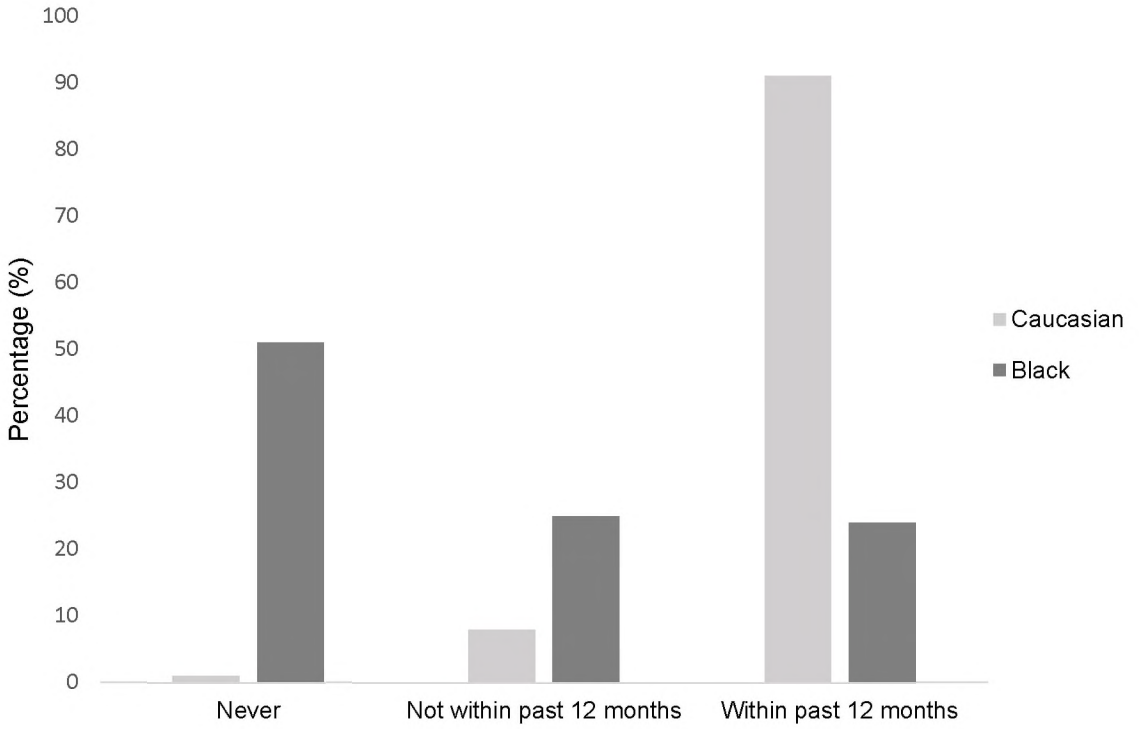

Figure 17: Frequency of alcohol consumption over the past 12 months.

The majority of Caucasian women reported consuming alcohol within the last 12 months (91%). Conversely, the majority of Black females reported never having consumed alcohol (51%).

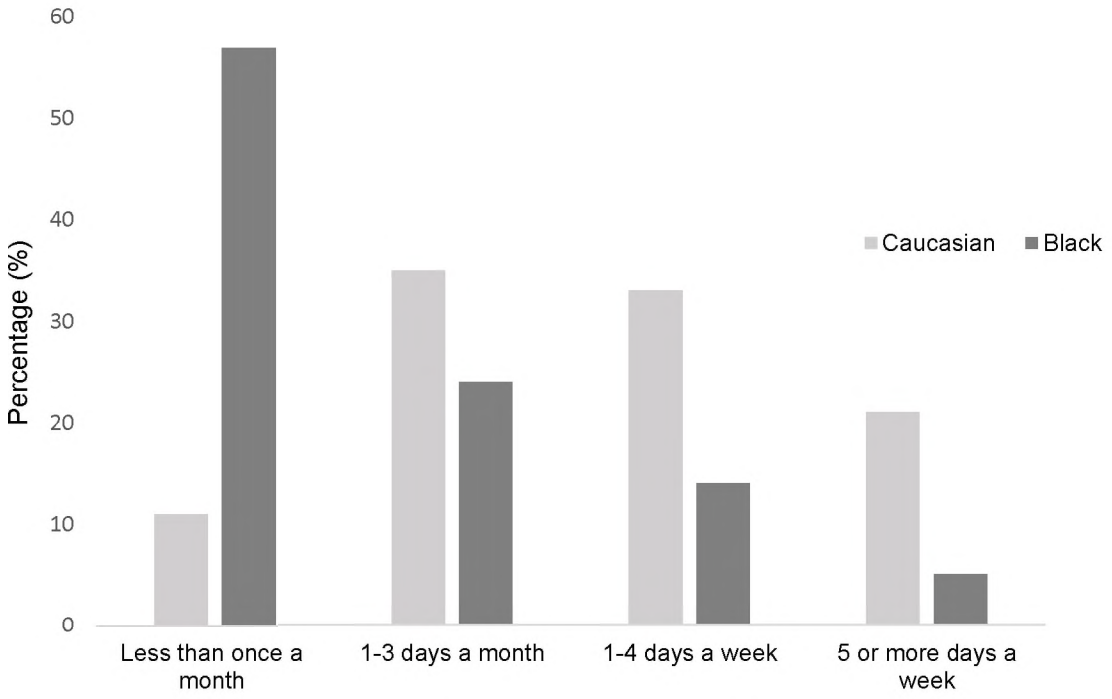

Figure 18: Frequency of alcohol consumption.

When assessing the alcohol consumption of those participants who had consumed within the past 12 months, the majority of the Black women (57%) reported consuming alcohol 'less than once a month' while the Caucasian women reported mostly consuming between '1-3 days a month' (35%) and '1-4 days a week' (33%) (Figure 18).

4.7 CORRELATIONS

4.7.1 SOCIOECONOMIC FACTORS

The strength of relationships was determined using the correlation coefficient (r): r < .40 = weak; r .40 - .60 = moderate; r > .60 = strong.

Table 19: Correlations between socio-economic variables and CVD risk conditions.

	Body mass		**BMI**		**WC**		**WSR**		**MAP**		**Total active MET-minutes**	
	C	**B**	**C**	**B**	**C**	**B**	**C**	**B**	**C**	**B**	**C**	**B**
Education (years)	r= -0.15 r^2= 0.02	r= -0.13 r^2= 0.17	r= -0.17 r^2= 0.29	r= -0.18 r^2= 0.32	r= -0.13 r^2= 0.17	r= -0.20 r^2= 0.04	r= -0.13 r^2= 0.17	r= -0.24 r^2= 0.58	r = -0.08 r^2= 0.01	r = -0.24 r^2= 0.58	r = -0.07 r^2= 0.01	r = -0.35 r^2= 0.12
Income (R/month)	r= -0.13 r^2= 0.17	r= -0.09 r^2= 0.01	r= -0.16 r^2= 0.03	r= -0.08 r^2= 0.01	r= -0.01 r^2= 0.00	r= -0.03 r^2= 0.00	r= -0.02 r^2= 0.00	r= -0.02 r^2= 0.00	r= 0.11 r^2= 0.01	r= -0.01 r^2= 0.00	r= -0.01 r^2= 0.00	r= 0.03 r^2= 0.00

Red indicates significance ($p<0.05$); C = Caucasian; B = Black; BMI = Body Mass Index; WC = Waist Circumference; WSR = waist-to-stature ratio; MAP = mean arterial pressure.

Negative relationships were found between education level and each of the risk conditions outlined in Table 19. However, these relationships are weak with the only significances being with education and WSR, MAP, and total active MET-minutes in the Black group. In addition, low r^2 values indicate that the predictability of one variable from another were poor. Weak negative relationships were also found between monthly income and risk conditions, however none were significant.

4.7.2 PHYSICAL MEASURES

Table 20: Correlations between cardiovascular risk conditions (Caucasian; Black).

	Mass		BMI		WC		WSR		MAP	
Mass			r=0.91	r^2=0.83	r=0.85	r^2=0.72	r=0.63	r^2=0.40	r=0.41	r^2=0.17
BMI	r=0.95	r^2=0.90			r=0.85	r^2=0.72	r=0.87	r^2=0.76	r=0.36	r^2=0.13
WC	r=0.91	r^2=0.83	r=0.92	r^2=0.85			r=0.96	r^2=0.92	r=0.39	r^2=0.15
WSR	r=0.84	r^2=0.71	r=0.92	r^2=0.85	r=0.97	r^2=0.94			r=0.22	r^2=0.05
MAP	r=0.43	r^2=0.18	r=0.44	r^2=0.19	r=0.43	r^2=0.18	r=0.42	r^2=0.18		

(*) denotes significance ($p<0.05$); BMI = Body Mass Index; WC = Waist Circumference; WSR = Waist-to-stature ratio; MAP = Mean Arterial Blood Pressure

Table 20 shows significant positive correlations between all risk conditions except for WSR and MAP in the Caucasian group. In addition, the relationship between all risk conditions except for MAP, were strong ($r>0.6$). This supports the evidence highlighting the prevalence of co-occurring risk conditions, leading to increased risk of metabolic syndrome.

4.7.3 LIFESTYLE

4.7.3.1 Physical Activity

Table 21: Correlations between physical activity variables and CVD risk conditions.

	Body mass		BMI		WC		WSR		MAP	
	C	**B**	**C**	**B**	**C**	**B**	**C**	**B**	**C**	**B**
Total active MET-minutes	r= -0.17 r^2= 0.03	r= -0.03 r^2= 0.00	r= -0.12 r^2= 0.01	r= -0.01 r^2= 0.00	r= -0.24 r^2= 0.58	r= 0.02 r^2= 0.00	r= -0.19 r^2= 0.04	r= 0.04 r^2= 0.00	r = -0.11 r^2= 0.01	r = 0.29 r^2= 0.08
Seated MET-minutes	r= -0.03 r^2= 0.00	r= -0.12 r^2= 0.01	r= -0.05 r^2= 0.00	r= -0.15 r^2= 0.01	r= -0.05 r^2= 0.00	r= -0.16 r^2= 0.03	r= -0.01 r^2= 0.00	r= -0.18 r^2= 0.03	r= -0.20 r^2= 0.04	r= -0.38 r^2= 0.14

Red indicates significance ($p<0.05$); C = Caucasian; B = Black; BMI = Body Mass Index; WC = waist circumference; WSR = waist-to-stature ratio; MAP = mean arterial pressure.

No strong relationships were evident between physical activity variables and CVD risk conditions, despite significance in some (Table 21). In the Black group, significance was found between total active MET-minutes and MAP, as well as seated MET-minutes and MAP. This was not mirrored in the Caucasian group where there was no significance.

4.7.3.2 Diet

Due to the limitations with respect to dietary data recall, correlations using dietary variables were excluded from analysis.

CHAPTER V - DISCUSSION

Following the format of Chapter IV, this Chapter discusses the results according to the following three categories: 1) obesity, classified as 'morphological risk'; 2) hypertension classified as 'cardiovascular (CV) risk'; and 3) diet, physical activity, smoking and alcohol consumption classified as 'lifestyle-related risks'. Owing to the interlinking of various risk factors and the need for combined discussion, however, the order changes in some instances to allow for an integrated and coherent understanding of the results.

5.1 SOCIOECONOMIC STATUS

Socio-economic results pertaining to education levels and gross monthly income showed large disparities between groups; Black women were significantly less educated, and earned less (Table 12). These findings were supported by the effects sizes, indicating the large difference between groups (*d=1.69* and *d=2.21*, respectively). With respect to education, this is a largely expected finding as the prevailing lack of funding and resources allocated to public education in the Eastern Cape disproportionately affects the poorest populations (DHS, 2003; WHO, 2016).

Looking at disparities in income, this is largely attributed to the type of work the participants did. Within the Black sample, most individuals were domestic workers, kitchen assistants, or cleaners working as Rhodes University. This type of work is classified as 'basic skills' work which does not require any specific training or education after school, and subsequently yields low income (DHS, 2003). This coincides with the simultaneous low education levels of this group. In the Caucasian group the type of work was more varied, however the majority of women held graduate jobs in the business sector. Due to the education levels required to attain these types of jobs, they typically yielded a higher income.

5.2 MORPHOLOGICAL RISK

5.1.2 OBESITY

5.1.2.1 Mass and Body Mass Index (BMI)

Black women were significantly *(p<0.01)* heavier than the Caucasian women resulting in significantly *(p<0.01)* higher BMI values classifying them as obese (34.19 kg/m^2 and 25.90 kg/m^2. Similarly, effect sizes showed a large difference between groups for both measures (*d=0.95* and *d=1.35* respectively). This finding is supported by Goedecke *et al.* (2009) and van der Merwe and Pepper (2006) who similarly found that Black South African females were significantly heavier and had higher BMIs than their Caucasian counterparts. In addition, extensive research suggests that Black South African women are the most obese population in South African- a finding attributed to both low socio-economic status and sociocultural beliefs surrounding the perceived desirableness of excess body fat (van der Merwe & Pepper, 2006; Goedecke *et al.,* 2009; Tibazarwa *et al.,* 2009; Jackson *et al.,* 2010; Walter & Durandt, 2011 Goedecke *et al.,* 2015; South African Heart and Stroke Foundation, 2016).

Literature from 2006 found that 31-34% of Black South African women and 18-24% of Caucasian women were obese countrywide (BMI>25.00 kg/m^2) (van der Merwe & Pepper, 2006). The current study, however, found that 69% of Black women and only 16% of Caucasian women were obese. This discrepancy is likely owing to the fact that the current study was localized to the Makana region of the Eastern Cape, while the former study was conducted across all provinces.

This is evidenced, in part, by region-specific data from the 2003 Demographic Health Survey (DHS) indicate that obesity rates vary considerably between provinces (Table 22).

Table 22: Provinces ranked according to obesity prevalence in adult South African women (DHS, 2003).

Provinces	Obesity Prevalence
Eastern Cape	31.9%
Western Cape	30.3%
Gauteng	30.1%
Mpumalanga	28.0%
Free State	26.2%
Kwazulu Natal	24.5%
North West	24.4%
Northern Cape	24.2%
Limpopo	21.8%

Table 22 shows that the Eastern Cape had the highest percentage of obese women, followed closely by the Western Cape and Gauteng. In-line with previous findings, the 2003 DHS also indicated that Black women accounted for the majority of obese women across all provinces (55.0%).

As highlighted in Table 22, obesity prevalence in Black women in the Eastern Cape was approximately 31.9% in 2003. More recent data, however, shows a much higher percentage: a study conducted on Black males and females in the Makana region of the Eastern Cape found that 81% of the female population were obese (Jackson *et al.,* 2010) while the current study found that 69% of females in the same region were obese. However, these studies focused on a small sub-sample of the Eastern Cape population, thus the findings are likely not representative across the entire province. Nevertheless, both studies show an obesity prevalence markedly higher than provincial data, suggesting that this specific sub-sample of Black females are more obese than the rest of the province. In part, this may be attributed to socioeconomic factors as it is well documented that a negative relationship between socioeconomic status and obesity levels exists (Vorster *et al.,* 2002; Kruger *et al.,* 2005; DHS, 2003; WHO, 2016). However, the current study showed no significance in the relationships between socioeconomic factors and obesity markers, despite the relationships being negative (Table 19). A further consideration is that the majority of Black females in

both studies were employed by an institution which provided meals for their employees. Although the nutrient composition of these meals was not evaluated, it is possible that these meals were high in energy and low in nutritional value, leading to overconsumption and subsequent weight gain in this group. A deeper look into diet as well as other contributing lifestyle factors will be addressed later on in this chapter.

To the author's knowledge, limited research on obesity prevalence in South African Caucasian women exits. Nationwide data from 2003, however, showed that the mean BMI of Caucasian South African women was 25.00 kg/m^2, and 14% were obese (DHS, 2003). Conversely, one regional study in the North West province found that the mean BMI of Caucasian women was 28.50 kg/m^2, and 30.4% were obese (Schutte & Olkers, 2006). The current study, however, mirrored the results of the 2003 DHS study where mean BMI was 25.90 kg/m^2 and 16% of Caucasian women were obese. As Caucasian female populations in South African typically do not present with high obesity prevalence, this may account for the lack of research in this area.

5.2.1.2 Waist Circumference and Waist-to-Stature ratio

Black women had significantly *($p<0.01$)* larger waist circumferences than Caucasian women (95.34 cm and 79.84 cm), with a large between-group effect size *($d=1.09$)*.This is indicative of central obesity (WC > 80 cm) and a 'high' risk classification for cardiovascular disease (WC ≥ 88 cm) in the Black women (Figure 7). The Caucasian women, however, presented with no central obesity and a 'low' risk classification for cardiovascular disease.

As with body mass and BMI results, this finding is supported by data from 2003 showing that Black South African women had larger waist circumferences than Caucasian South African women countrywide (84.6 cm and 80.2 cm, respectively) (DHS, 2003). DHS data also showed that 34.7% of women in the Eastern Cape exceeded the 88 cm cut-off for 'high' CVD risk, and that Black women accounted for two thirds of this percentage (66.3%).

The current findings, nevertheless, appear to be in direct conflict with studies by Weinsier *et al.* (2001) and Jennings *et al.* (2008) who found that Caucasian women had higher levels of visceral (central) adiposity compared to Black women. The Weinsier *et al.* (2001) study, however, was conducted on 46 America participants (23

of Black African descent and 23 Caucasian).It is, thus, not directly comparable to the current study. In addition, contrary follow-on research by Goedecke *et al.* (2015) found that Black South African women had significantly *($p<0.05$)* larger waist circumferences than Caucasian females. Upon further analysis, however, body fat distribution results showed that the Black women had less visceral adipose tissue and more subcutaneous adipose tissue than Caucasian females (Goedecke *et al.,* 2015). This, therefore, placed the Caucasian women at higher CVD risk *despite* their lower relative waist circumference measures. Thus, although the current findings appear to be contradictory at first glance, the waist circumference findings are similar albeit the different interpretation of associated risk. The Goedecke *et al.* (2015) study subsequently concluded that waist circumference measures alone, while useful surrogates, were not accurate in determining fat mass distribution and thus should be interpreted with caution.

Waist-to-stature ratio (WSR) was used in this study in addition to waist circumference measures. This was due to the proposed limitations associated with waist circumference measures alone, particularly when comparing populations with varying statures. As with waist circumference, waist-to-stature results showed that Black women were classified as at 'high risk' for cardiovascular disease (0.61) while the Caucasian women were classified as 'normal' or healthy' (0.48). These ratios were calculated according to the Ashwell Shape Chart® outlined in Chapter 3, pp. 57. Despite the limitations associated with waist circumference measures alone, in this study, both the waist circumference and waist-to-stature results indicated high cardiovascular disease risk in the Black women compared to the Caucasian women.

5.3 CARDIOVASCULAR RISK

5.3.1 BLOOD PRESSURE

Black women presented with significantly *($p<0.05$)* higher systolic, diastolic, and mean arterial blood pressure than the Caucasian women. Both groups, nevertheless, presented with 'normal' mean blood pressure readings (Black women: 122/77 mm Hg and Caucasian women: 116/70 mm Hg). In addition, the effect size was moderate (*$d=0.40$*; *$d=0.50$*; *$d=0.41$*, respectively). This finding supports nationwide data from 2003 where Black South African women had a mean blood pressure of 121/75 mm Hg and Caucasian South African women, 120/75 mm Hg (DHS, 2003). The current

findings are, however, contradictory to the bulk of previous literature highlighting the disproportionately high prevalence of hypertension in Black females (Cappuccio, 1997; Forrester, 2004; Kearney *et al.,* 2005; Norman *et al.,* 2007; Walter & Durandt, 2011; Crymble *et al.,* 2014; South African Heart and Stroke Foundation, 2014).

A more in-depth analysis into the current study revealed a likely explanation. Isolating the cases of 'known hypertension' revealed that 18% of Black women reported previously known hypertension diagnoses, compared to 6% in the Caucasian group. More importantly, 100% of all previously diagnosed participants, Black and Caucasian, reported taking blood pressure lowering medication for the duration of the study. Consequently, the results showed that Black women *without* previous hypertension diagnosis presented with a mean blood pressure of 130/83 mm Hg, placing them within the 'pre-hypertensive' classification category (Table 4; Figure 5). This was a significantly *($p<0.05$)* higher result compared to the total mean finding of 122/77 mm Hg in this group, and highlights the increased risk within this sub-sample. This finding is supported by a large between-group effect size (d=1.086). Unlike the Black women however, the Caucasian women showed no significant difference between the total group (116/70 mm Hg) and the sub-group of participants without previously diagnosed hypertension (115/69 mm Hg).

This finding highlights the importance of conducting health research within the context of existing health conditions and diseases. The 2003 nationwide DHS findings stating that both Black and Caucasian South African females had 'normal' blood pressures did not account for those who were already diagnosed and on medication. Results from the DHS study, therefore, likely represented an inaccurate picture of hypertension risk. Jackson *et al.* (2010) similarly found that Black females were classified as 'pre-hypertensive' after accounting for those who were previously diagnosed, which supports the findings of the current study.

Previous literature suggests that obesity is a powerful contributor to hypertension in Black women, thus, it is postulated that raised obesity levels within the current Black female sample may account for increased hypertension risk (Schutte *et al.,* 2006; Norman *et al.,* 2007; Tibazarwa *et al.,* 2009; WHO, 2011). The relationship between obesity and hypertension in this study will be addressed in more detail below.

5.4 CORRELATIONS BETWEEN CVD RISK CONDITIONS

All five measured CVD risk conditions (Mass, BMI, WC, WSR, and MAP) showed a significant positive correlation in both groups, except for WSR and MAP in the Caucasian group (Table 20). This supports the evidence surrounding the clustering of conditions evident in the development of the metabolic syndrome or 'syndrome X' (Reaven, 2005). The most recent diagnostic criteria, however, for metabolic syndrome include the presentation of three or more of the following five conditions: central obesity (WC ≥ 88 cm), Hypertension (BP ≥ 130/85 mm Hg), high triglycerides (>1.7 $mmol.l^{-1}$), low HDL cholesterol (< 1.29 $mmol.l^{-1}$) and fasting hyperglycaemia (fasting plasma glucose ≥5.6 $mmol.l^{-1}$). The current study only included obesity and hypertension measures, thus, the prevalence of metabolic syndrome is not possible to ascertain in the population studied.

Positive significant relationships, nevertheless, are evident between obesity surrogates (Mass, WC, and WSR), and MAP in both groups, highlighting the link between obesity and hypertension (Black: r^2=0.18, r^2=0.18, r^2=0.17, and Caucasian: r^2=0.16, r^2=0.15, r^2=0.05) (Table 9, pp x). In addition, both groups expressed strong, positive and significant relationships between waist circumference and waist-to-stature ratios (Black, r^2=0.94, Caucasian, r^2=0.92), suggesting that the two measures are intrinsically linked despite the proposed limitations of such surrogates. Body fat distribution was, however, not measured in this study. The validity of these surrogates as obesity markers was, therefore, not ascertained.

5.5 LIFESTYLE

5.5.1 PHYSICAL ACTIVITY

Black women were significantly *($p<0.05$)* more active than the Caucasian females, taking part in 2688.86 MET-minutes/week compared to 1828.27 MET-minutes/week However, the effect size was only moderate (*d*=0.46). Both groups, however, were equally classified as 'minimally active', which is defined as taking part in 600-3000 MET-minutes/week.

In comparison to national data, however, both Black and Caucasian women in this study appeared to be notably more active (DHS, 2003). In 2003, 66.0% of Black females and 66.8% of Caucasian females were *inactive*, while only 12.4% of Black

females and 10.8% of Caucasian females were *sufficiently active*. Conversely in the current study, 13% of Black females and 29% of Caucasian females were *inactive*, while 45% of Black females and 23% of Caucasian females were *sufficiently active*. The DHS (2003) further reported that physical inactivity was higher in urban individuals, and linked this to increased risk of CVD within Black South African females.

The current data do not appear to match this trend, however, it is noteworthy that urban national data encompasses individuals from both the business sector as well as the informal sector. The inclusion of office-bound individuals within the 'urban black' cohort may have impacted on physical activity scores as the majority of participants in this study were involved in more active jobs (cleaning or kitchen assistance). This is supported by a study conducted on Black males and females in the Makana region of the Eastern Cape where 42.4% of the Black females were sufficiently active, and 14.4% were inactive (Jackson *et al.,* 2010); findings nearly identical to those in the current study (45% active, 13% inactive). Within the Caucasian cohort, however, females in the current study were found to be more active than average national scores, despite the fact that they were mostly office workers.

Nevertheless, it is noteworthy that *exercise intensity;* a key factor in determining the health benefits associated with regular exercise, was not measured in this study. It is well documented that in order to benefit from the effects of physical activity, a certain threshold or intensity of regular exercise is required (Blair *et al.,* 2004; WHO, 2010; Garber *et al.,* 2011). Despite some variations, it is generally accepted that adults should accumulate 30 minutes of moderate-intensity cardiorespiratory exercise on most, but preferably all days or the week to reap the health benefits associated with regular exercise (Blair *et al.,* 2004; Garber *et al.,* 2011). The current study however used MET-minutes as the unit for expenditure; a measure encompassing the product of intensity *and* time. Thus when comparing total MET-minutes between groups, it is possible that the Black women engaged in *low* physical activity levels spanning a great amount of time, which could result in a high number of total active MET-minutes. This low intensity exercise might therefore not have met the recommended exercise intensity guidelines despite being maintained over a long period of time. Subsequently, this could place the group as *insufficiently active.* Conversely, the Caucasian women may have engaged in higher exercise intensities in short bursts, leading to lower total

MET-minutes, but nevertheless meeting the recommended guidelines categorizing them as *sufficiently* active. Therefore the findings of this study may have inaccurately reported on physical activity levels due to the absence of exercise intensity measures, and should thus be interpreted with caution.

When comparing domains of exercise, findings from this study are once again inconsistent with national findings. Although work and leisure-related physical activity were not differentiated between in this study, transport-related physical activity was found to be significantly higher in the Black females compared to Caucasian females (15% and 5%, respectively) with a moderate effect size *(d=0.70).* National data from 2003, however, showed that both Black and Caucasian females attributed approximately 30% for their weekly physical activity expenditure to transport (30.6% and 29.0% respectively). However, this discrepancy is likely due to the fact that the current study was conducted in Grahamstown; a geographically small town spanning 65.1 km^2 (Google maps). In comparison to larger cities in South Africa (Johannesburg, 334.81 km^2), it is expected that travel time will be substantially lower in smaller towns such as Grahamstown.

With respect to seated MET-minutes/week, Caucasian females spent significantly more time sitting (3384.05 MET-minutes/week and 1952.05 MET-minutes/week, respectively) and the between-group effect size was large *(d=1.12).*This was an expected finding as the majority of Caucasian women were office workers. Nevertheless, Caucasian women in this study were significantly more active than reports from 2003 (DHS, 2003), suggesting that leisure time physical activity was likely high in this sample. Additionally, this supports the postulation that Caucasian women may have exercised at a higher intensity to their Black counterparts.

5.5.2 PHYSICAL ACTIVITY AND SOCIOECONOMIC STATUS

Throughout literature, it is well documented that a positive relationship between socioeconomic status and physical activity levels exists (DHS, 2003; McVeigh *et al.,* 2004; Wilson *et al.,* 2004; Walter & Durandt, 2011; WHO, 2016). This is typically attributed to 1) the financial costs associated with many types of sports or activities such as membership fees, equipment, and gear, and 2) the lack of education surrounding the health benefits of regular physical activity (DHS, 2003). Therefore, in accordance with the large disparities in socioeconomic status between groups in this

study, it was expected that the Caucasian women would be more physically active than the Black women. This, however, was not the case, and the opposite was found: within the Black group, a weak but significant negative relationship between education level and total MET-minutes/week was found (r^2=0.12) and no relationship was evident in the Caucasian sample (Table 19, pp. 86). This confounding finding, however, could be explained by the fact that physical activity levels may have been inaccurately measured within this study. Further studies should incorporate measures of exercise intensity as this will highlight whether or not participants are meeting the recommended dosage necessary to obtain the health benefits of exercise.

5.5.3 PHYSICAL ACTIVITY AND CVD RISK CONDITIONS

No strong relationships were evident between physical activity variables and CVD risk conditions, despite significance in some (Table 21). In particular, no reduction in CVD risk conditions was evident in the Black group, who were classified as more physically active.

This supports the postulation that physical activity may have been inaccurately represented in this study, such that Black women may have been engaging in exercise at an intensity too low to from which to receive the health benefits. Alternatively, these findings may suggest that other external or lifestyle factors may be negating the positive effects of physical activity in this group.

5.4 DIET

Due to the limited number of completed food dairies recalled, dietary analyses were done on a smaller sub-sample of participants. This subsequently led to the exclusion of correlational analyses with dietary data, as relationships were not able to be assessed.

It is widely accepted that the average recommended daily energy intake for females is approximately 8700KJ (or 2000kcal) per day (FBDG-SA, 2013). According to this criteria however, both Black and Caucasian females consumed notably less than the recommended daily amount (Figure 11). In fact, Black females consumed nearly 1500KJ less (1457KJ) and Caucasian women, nearly 2500KJ less (2299KJ). Nevertheless, the between-group effect size was moderate *(d=0.45).* This was a surprising result, particularly due to the high prevalence of obesity the Black sample.

However, literature suggests that such inconsistencies may be due to inaccurate and under-reporting of energy intake, as this is known to pose challenges in dietary-related research (Mendez *et al.,* 2004; Jackson *et al.,* 2010). Such under-reporting is often attributed to two main factors: 1) people are typically not used to recording everything they consume and thus can be inaccurate, and 2) many people feel ashamed or insecure about their dietary choices and thus tend to over report the healthier foods, and underreport unhealthy foods (Mendez *et al.,* 2004). In this study, it is likely that both of these factors played a part; however inaccurate reporting due to unfamiliarity is postulated to have been more of a factor in the Black group than the Caucasian group. This is because many of the Caucasian females expressed having done some form of food recall before, which is a common component of many formalized weight management programmes (www.myfitnesspal.com; www.fatsecret.co.za). Conversely, within the Black group, the majority of women had no previous experience in food recalls. Thus it is likely that this unfamiliarity may have led to inaccurate reporting. In addition to this, the language barriers would also have likely impacted the accuracy for the food recalls, as discussed in 'Limitations to the Study' pp. 63.

In addition, previous research shows that obese individuals are more likely to under-report than overweight or normal weight individuals, although the exact reason why was not explored (Charlton *et al.,* 2001; Vorster *et al.,* 2002; Mendez *et al.,* 2004; Jackson *et al.,* 2010). One study conducted in the North West province found that obese Black women tended to under-report to the greatest extent (Vorster *et al.,* 2002). This was to such an extent that when under-reporters where were excluded from analyses, the power of the positive relationships between BMI and total energy intake increased (Vorster *et al.,* 2002). This highlights the importance of assessing dietary data within the context of the limitations of self-reported data.

Macronutrient breakdown showed that Black women consumed significantly *($p<0.05$)* more carbohydrates per day than Caucasian women (214.93 g/day and 141.82 g/day respectively) and the effect size was large *($d=1.05$).* This accounted for 64% of total intake in the Black group, and 53% in the Caucasian group. Owing to the lower socioeconomic status of the Black females, this was an expected finding as carbohydrates are typically cheaper than foods high in protein or fat (FBDG-SA, 2013). Conversely, the higher protein intake in the Caucasian group (25% and 17%

respectively) is attributed to the same reason, as protein-rich foods are typically the most expensive foods (FBDG-SA, 2013).

Second to carbohydrate intake, the biggest difference between groups was the total grams of added sugar consumed on a daily basis. Black South African women consumed an average of 46.72 g of sugar per day; nearly two times the recommended daily allowance of 25 g (FBDG-SA, 2013; American Heart Association, 2016). Caucasian women also consumed more sugar than the recommended daily allowance, although it was significantly *($p<0.05$)* less than the Black females (35.02 g/day). Nevertheless, the between-group effect size was moderate (*$d=0.578$*). Extensive research highlights the relationship between excessive sugar intake and obesity, thus it postulated that was a likely contributor to the high prevalence of obesity in Blacks females in this study (Puoane *et al.,* 2006; Wolmarans & Danster, 2008; Jackson *et al.,* 2010; FBDS-SA, 2013; South African Heart and Stroke Foundation, 2016; WHO, 2016). As with carbohydrate intake, it is also postulated that increased sugar consumption is related to lower socioeconomic status, as sugar is a cheap and high source of energy which is readily available to those with lower income (Puoane *et al.,* 2006; FBDG-SA, 2013; Wentzel-Viljoen *et al.,* 2013). Additionally, lower education levels are also likely to be related as this results in less exposure to the dangers of excess sugar consumption from a young age (DHS, 2003; FBDG-SA, 2013). Due to the limited number of food diaries recalled in the current study however, these direct correlations were not able to be obtained.

Nevertheless, these socioeconomic factors do not account for the high levels of sugar consumption in Caucasian females. It is therefore further postulated that within both groups, sugar intake may have exceeded the recommended daily allowance through the consumption of processed and packaged foods; a common constituent of the Westernized diet. It is well documented that sugar is added to many processed foods to extend the shelf-life, and this often results in people unintentionally consuming excess sugars (Taubes, 2008; Wentzel-Viljoen *et al.,* 2013). The Food-Based Dietary Guidelines of South Africa (2013) state that 'foods containing excess sugars and salts should be consumed sparingly' (pp 1). While this advocates the reduced consumption of added sugars, it lacks sufficient detail pertaining to how much dietary sugar is in fact 'too much', and how much sugar in in certain common foods. This therefore

highlights the need for education surrounding the dangers of consuming processed foods, as well as the need for detailed national dietary guidelines pertaining to this.

With respect to fat consumption, total fat intake was relatively similar between groups, despite the higher prevalence of obesity in Black females. Nevertheless, Black females consumed significantly more polyunsaturated fat than Caucasian females (17.49 g/day and 12.35 g/day) yet the effect size was only moderate *(d=0.69).* Furthermore, there was no significant difference between saturated fat intakes between groups (Table 17). These findings challenge the notion that saturated fat intake is the primary cause of obesity, as well as the notion that polyunsaturated fats are favourable (Krauss *et al.,* 2000; Vorster *et al.,* 2002; Taubes, 2008; American Heart Foundation, 2016). Due to exclusion of correlational dietary analyses however, this relationship was unfortunately not explored in this study.

5.5 SMOKING AND ALCOHOL CONSUMPTION

Significantly more Caucasian women reported being smokers than Black women, however percentage prevalence in both groups was small. National findings from 2003 indicated that 5.1% of Black women and 26.7% of Caucasian women smoked, while this study found that only 3% of Black females and 14% of Caucasian females. This is in accordance with evidence highlighting the decline in smoking as a result of effective legislation aimed at reducing its desirability (DHS, 2003). Nevertheless, smoking prevalence in Black female populations particularly is consistently low throughout literature, thus it was an expected finding in the current study. In part, this is supported by an Eastern Cape based study conducted in 2010 similarly found that only 3% of Black females reported being smokers (Jackson *et al.,* 2010). Additionally, smoking prevalence among women in the Eastern Cape was ranked third lowest in 2003, thus it was expected that the current study would find a low prevalence in this Eastern Cape sample. Therefore, it is unlikely that smoking played an integral role in cardiovascular disease risk in these sample groups. Nevertheless, it is acknowledged that the data were self-reported, thus the accuracy of the results was not able to be tested. Due to the inherent limitations associated with self-reported data, as well as the tendency for under-reporting, the findings need to be interpreted with caution.

With respect to alcohol consumption, Caucasian females consumed significantly *($p<0.05$)* more alcohol per day than Black females (10.46g and 0.92g, respectively)

with only moderate between-group effect size *(d=0.70).* However, large standard deviations highlight pronounced inter-individual variance, suggesting that mean values may not give an accurate representation of alcohol consumption behaviours in these samples.

In part, this is evidence by differences in frequency of alcohol consumption within and between groups. The majority of Caucasian females reported consuming alcohol within the last 12 months (91%), and typically consumed alcohol weekly. Conversely, 51% of Black females reported having never consumed alcohol before, and of those who had consumed in the past 12 months, average consumption was 1 to 3 times per month. This was an expected finding as previous literature indicates that abstinence is prominent in Black African females (Martinez *et al.,* 2011). Nevertheless, it is acknowledged that these findings are drawn from self-reported data thus they may not be accurate.

In addition, the current study didn't measure alcohol dependence. This is a limitation as the difference between the beneficial effects of moderate alcohol consumption versus the damaging effects of alcohol dependence were not differentiated between. Therefore the results need to be interpreted within this context.

CHAPTER VI - SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

6.1 MAIN FINDINGS AND CONCLUSIONS

The current findings confirm and emphasize the severity of obesity in Black women of the Eastern Cape. This is evidenced by the high percentage of Black females classified as obese, particularly severely obese. In addition, the high mean waist circumference and waist-to-stature ratios highlight the prevalence of central adiposity within this population group. Despite the limitations attributed to obesity surrogates such as BMI and waist circumference, in this sample, they provided a good indication of obesity prevalence. This is evidenced by the significant and strong correlations between BMI, weight, waist circumference, and waist-to-stature ratio in both groups.

Blood pressure findings showed that of those without previous hypertension diagnoses, Black females were classified as 'pre-hypertensive', while Caucasian females presented with normal blood pressures. In addition, a greater percentage of the Black females reported being previously diagnosed as hypertensive compared to the Caucasian females, thereby highlighting the high prevalence of hypertension in Black females.

Despite the fact that Caucasian females reported consuming more alcohol and smoking more than Black females, neither groups did so excessively. Thus the majority of cardiovascular disease risk in both groups was attributed to other factors perhaps including physical activity, diet, and socioeconomic status. Black females reported being more physical activity than Caucasian females, and attributed a greater proportion of their daily physical activity to transport. Conversely, Caucasian females reported spending significantly more time sitting than Black females. Nevertheless, lack of exercise intensity measures limited the interpretation of these findings and subsequent inferences thereof.

Dietary data also showed significant differences between groups, particularly with respect to carbohydrate, sugar, and fat consumption. Specifically, Black females consumed significantly more Kilojoules, carbohydrates, polyunsaturated fats, and added sugars than Caucasian females. Caucasian females, on the other hand, consumed significantly more protein and monounsaturated fats. However, due to

postulated under-reporting, as well as a reduced number of food recall diaries that were recalled, inferences with respect to dietary data are similarly limited.

Socioeconomic status showed to be a notable predictor of cardiovascular disease risk in this study, as those of low socioeconomic status presented with the greatest risk of cardiovascular disease. However, the exact mechanisms behind this relationship were not fully explored.

6.2 SUMMARY OF THE STATISTICAL HYPOTHESES

Hypothesis 1:

Significant differences were found between groups with respect to all morphological characteristics except body composition which was not measured. Therefore the null hypothesis is rejected for measures a), b), c), and d) and neither accepted not rejected for measure e).

Hypothesis 2:

A significant difference was found between groups with respect to systolic, diastolic, and mean arterial blood pressure measures. However, glucose and insulin measures were not able to be conducted in this study. Therefore the null hypothesis is rejected for measure f), and neither accepted not rejected for measures g) and h).

Hypothesis 3:

Significant differences were found between groups with respect to all lifestyle characteristics. Therefore the null hypothesis is rejected for measures i), j), k) and l).

Hypothesis 4:

Significant differences were found between groups with respect to both education levels and income. Therefore the null hypothesis is rejected for measures m) and n).

6.3 RECOMMENDATIONS

Future studies assessing cardiovascular disease risk in South African populations should take the following recommendations into consideration:

Despite the practicalities of self-reported data in epidemiological research (Richardson *et al.,* 2001), future studies should consider incorporating objective measures of

physical activity, diet, smoking, and alcohol consumption where possible. This would negate inaccuracies and under-reporting, and would strengthen the findings of the research.

Due to some evidence highlighting the limitations of obesity surrogates in the absence of body fat distribution measures (Goedecke *et al.,* 2009), future studies should consider measuring body fat distribution in conjunction with mass, BMI, waist circumference, and waist-to-stature ratio.

It is acknowledged that race-specific 'cut-off' norms were not used for this study as they do not exist for Black South African populations. Future studies should consider ascertaining the validity of these cut-off norms, and aim to generate modified ones for Black South African populations if necessary.

In addition, future studies should incorporate measures of exercise intensity. This would enable researchers to assess whether exercise or physical activity is at an intensity high enough from which to derive health benefits.

Furthermore, future studies should consider assessing full lipid profiles as well as glucose and insulin. This would elicit a complete picture of cardiometabolic risk in a population, in addition to cardiovascular risk.

Due to the established relationship between age and cardiovascular disease risk (Dubnov *et al.,* 2003; Bray & Champagne, 2005), future studies should consider assessing a population within a small age range. Alternatively, future research should compare cardiovascular disease risk between age ranges.

Future studies should also consider assessing alcohol dependency behaviours in addition to total alcohol habitually consumed. This would allow for the differentiation between beneficial levels of alcohol consumption, versus problematic and damaging levels of consumption.

Lastly, future studies should aim to explore the exact mechanisms behind why low socioeconomic status lends itself to increased cardiovascular risk. Factors such as poor education, lack of access to health care, financial strain, and socioeconomic stress could be assessed in greater detail within each at risk population group.

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APPENDICES

APPENDIX 1

Human Kinetics and Ergonomics Ethics Committee Report

RHODES UNIVERSITY
Where leaders learn

Student Name: Sandra Remsing	
Project Title: Cardiovascular disease risk in Black and White South African women: an Eastern Cape sample	
Supervisor: Prof C Christie	**Type of Research:** MSc study
Application received: 10 Feb 2016	**Code:** 2016-02-02
Report Date: 01 March 2016	
Resubmitted on: 04 March 2016	**Final Report compiled:** 10 March 2016

Dear Sandra,

Your resubmission has been successful – the reviewer have approved your modifications. You may therefore continue with your experimental testing.

Please do however take note of the following:

- You should verbally check with participants whether you may take photographs of them.
- Should you use photographs of participants in your final thesis submission, any identifying features should also be blurred / covered.

Approved ✓	Approved, on condition that suggestions have been effected	Request for rework and resubmission	Rejected

On behalf of the HKE Ethics Committee I wish you all the best with your study.

Signed

Miriam Mattison.

MC Mattison
Chair: Human Kinetics and Ergonomics Ethics Committee

Confidential
HKE Ethical Committee Review Form

APPENDIX 2

CARDIOVASCULAR DISEASE RISK IN BLACK AFRICAN FEMALES: AN EASTERN CAPE SAMPLE

Letter to participants

Dear Participant

Thank-you for expressing interest in the study.

The aim of this study is to assess the prevalence of cardiovascular disease within women in the Eastern Cape- an area of research which has received very little attention in the past. Information gained from this study will therefore be invaluable in not only creating awareness of cardiovascular disease risk within Eastern Cape women, but will also help you to understand your health status better.

Participation in this project will require you to initially have some physical measurements taken, and then to be interviewed about questions pertaining to your lifestyle. This will all be done at the Human Kinetics and Ergonomics department at Rhodes University, in a once-off testing session.

Before any of the physical measurements are taken, you will be required to fast the night before. This means not having anything to eat or drink (even water) for 10 hours prior to your testing session. This is for accuracy purposes, particularly for your body composition measures and for fasting glucose and insulin levels, which requires you to be in a 'fasted' state. This may cause a little discomfort, but every effort will be made to minimize this as much as possible, and testing sessions will be scheduled for early morning wherever possible so that the 10-hour fast is done the night before. You will then be allowed to eat and drink as you please as soon as your physical measures have been taken.

Once you have arrived at the department (at a previously agreed upon time which you choose), the measurements taken will include the following: your stature (height); your mass (how much you weigh), your waist circumference (taken using a measuring tape around your waist), and your body composition using Bioelectrical Impedance Analysis

(BIA). BIA is a method whereby a small current (which you cannot feel) is passed through your body. What this will measure is the time take for this current to go through your body as this is used as an indirect indicator of percent body fat. This is based on the understanding that the more body fat, the slower the current passes through your body. This measure will require you to take off your shoes and socks and lie down, while the researcher places electrodes on your hands and feet. This entire procedure is painless and takes a couple of minutes.

After this, you will need to have your blood pressure taken three times (for accuracy purposes). This will be done by the primary research using a sphygmomanometer. You will be required to rest quietly for five minutes before the cuff is placed on your arm and the measurement is taken. Three measurements will be taken and each measurement will be separated by three minutes.

This type of manual assessment of blood pressure usually leads to a small level of discomfort, however this shouldn't be too much too handle. In the event that it is too uncomfortable or causes any pain, you may decide not to take this measure.

The last physical measurement will be a blood withdrawal which is needed so that your fasting glucose and fasting insulin levels can be measured (a measure done for determining type II diabetes risk). This will be done by an accredited nursing sister from AMPATH laboratories in Peppergrove mall and will be done in the Human Kinetics and Ergonomics department. It is a simple once-off blood withdrawal that will be taken from your left forearm vein, and every effort will be made to ensure that this is as painless as possible.

Once all of these measurements are taken, you will be given some time to relax before we begin the interview stage. Here, you will be asked various questions regarding your lifestyle- the kind of work you do, the kinds of activities you are involved in, and the kinds of foods you eat. Please answer these questions as honestly as possible- there is not 'right' or 'wrong' answer; the questionnaires simple allow the researcher to get to know you and understand your typical day-to-day lifestyle habits better. The more accurate and honest the responses, the better the researcher can assess your cardiovascular disease risk.

Although this interview will be carried out in English, a copy of the questions will be available in isiXhosa, and an interpreter will be present to ensure that you understand

the questions fully. Feel free to ask either myself (the primary researcher), any research assistants, or the translator any questions at any point during the testing and interview stages.

Please note that all information received from you will be treated confidentially, and will be coded for use. At no point in time will your name be used, and your information will not be released to any other individual. The data will be used for academic purposes such as for publications, presentations, and thesis work, however will remain anonymous and coded. Once the information has been analysed, it will be communicated to you. This will include detailed information in the form of a written report on your cardiovascular health, as well as lifestyle habits which may be improved, and how to do so. We trust that you will find this information helpful and educational, and that it will help you to head a healthy and low-risk lifestyle going forward!

Participation in this study is purely voluntary, and you need not provide any justification should you choose not to participate. If you would like to participate however, please indicate this to anyone on the research team so that you can begin to read and sign your consent form.

Thank-you very much for your interest thus far, and feel free to ask any questions.

Sandra Remsing

Primary researcher (student)

076 898 4678

Email: sandraremsing@gmail.com

Dr. Candice Christie

Supervisor

046 603 8470

Email: c.christie@ru.ac.za

APPENDIX 3

SAMPLE DETAILS

Due to the nature of the study, a strictly homogeneous sample group is not required. Therefore, the inclusion and exclusion criteria are minimal to ensure that the sample group is as closely representative of the naturally existing wider populations as possible. Nevertheless, a number of restrictions have been chosen where necessary in order to ensure the safety of participants.

Inclusion criteria

Black African and Caucasian women: All chosen participants will be either Black African females or Caucasian females. Participants will be exposed to the same protocol and experimental conditions, thereby forming one large sample group. Once the data are recorded, the data will then be analysed in two separate groups with respect to race for comparative purposes.

Urban, working population: As the health implications of rapid urbanization are the central area of focus in this study, participants will be chosen from an urban, working population. This will be determined by the presence of up-to-date full-time or part-time employment contracts from established institutions, business, or companies. Participants will be required to work a minimum of three days a week to ensure that all participants work for the majority of the week. This will be determined in the pre-screening questionnaire, and through above mentioned documentation.

Females of reproductive age: All women chosen will be of adult reproductive age (18-49 years). This is to ensure that the hormone-imbalances associated with menopause, and the subsequent hormonal changes and health implications of post-menopause do not influence the individuals' health status (see exclusion criteria 'menopause'). This will be determined through the pre-screening questionnaire (Appendix 8).

Exclusion criteria

Peri-menopausal and post-menopausal: Menopause is characterised by a reduction in female sex-hormones which typically leads to changes in morphology and increased cardiovascular disease risk (Dubnov *et al.,* 2000). Consequently, the biological profile

of a peri- or post-menopausal female is considerably different to that of a female or reproductive age. (Dubnov *et al.,* 2000). Nevertheless, women of reproductive age still exhibit risk of developing cardiovascular disease, particularly if lifestyle factors such as physical inactivity and unhealthy eating habits are present (Goedecke *et al.,* 2008). Therefore, in order to ensure the sample group are at similar baseline levels of cardiovascular disease risk, women who are peri-menopausal or post-menopausal will not qualify for participation. This will be determined in the pre-screening questionnaire as presence of menstruation within the last 30 days.

Pregnant or lactating: Pregnancy and lactation both lead to morphological and anthropometric changes in the body; thus measurement of individuals in these states would likely lead to an inaccurate representation of cardiovascular disease risk (Rychick, 2010). Additionally, it may be unsafe to subject pregnant woman to an over-night fast in order to determine fasting glucose and insulin levels. Therefore women who are currently pregnant or lactating upon commencement of the study will be excluded from participation. This will be determined through the pre-screening questionnaire.

Physical disabilities/ injury: The measurement and assessment of habitual physical activity levels are central to this study. Therefore, participants who are disabled or injured to the point that they are physically incapable of participating in regular activities of daily living (ADL) will be excluded from participation, as this will influence normal metabolic expenditure (Motl and McAuley, 2010). This includes disabilities such as being wheelchair bound, or immobilized in any way such that ADLs are compromised. However, if a disability doesn't impact on the person's ability to participate in ADLs, they will not be excluded from participation. This will be determined by self-reporting in the pre-screening questionnaire.

English proficiency: Basic English literacy is required for participation to ensure that the participants are able to read, understand, and communicate autonomously with the researcher and research assistants as far as possible. Any participants who do not understand basic spoken English will be excluded from participation. This will be determined in the pre-screening form. To ensure that participants understand the study requirements and protocol, medical terminology that is not a part of everyday/basic English while be translated in writing into isiXhosa wherever possible.

A first language Xhosa-speaking translator will also be present at the information and testing sessions should any terminology without direct Xhosa translation requires explanation. In addition, if any Xhosa-speaking participant doesn't fully understand what they are reading or what they are being told, they may ask the translator to translate for them.

APPENDIX 4

RISKS AND BENEFITS

RISKS

Anthropometric and morphological measures

The majority of the anthropometric and morphological measurements are minimally invasive. These include: stature, mass, waist circumference, and bioelectrical impedance analysis. To minimize any potential embarrassment or feelings of uneasiness, all women will be measured by a female researcher in a private area. Before each measure is taken, the process will be thoroughly explained to the participants and any questions they have will be answered. A female translator who is proficient in English and Xhosa will be present in order to explain the procedure to any participants who are not proficient in English, while the primary researcher will explain to all participants who are proficient in English. This translator will be thoroughly briefed before testing commences to ensure that they are well prepared to adequately answer any questions that may arise. Effort will be made to ensure that participants are as comfortable as possible at all times, and their measurements will be kept confidential. In the rare event that any participant feels too compromised or embarrassed to participate, they will be reminded that they may withdraw from the study without any justification or adverse consequences, and will be fully supported by the researchers should they decide to do so.

The more invasive and discomfort-inducing measurements include the measuring of blood pressure, and blood withdrawals (which is accompanied by period of fasting prior to the blood withdrawal). The levels of discomfort will be managed as follows:

Blood Pressure

The act of taking blood pressure may cause mild to moderate discomfort. This method involves placing an inflatable cuff around the forearm, and pumping it up with air to create pressure on the brachial artery. Once the air pressure build around the arm, this usually leads to some mild discomfort, and rarely to moderate or severe discomfort. Every effort will be made to ensure that the discomfort is minimized by pumping up the cuff no higher than 200 mm Hg, and by ensuring the researcher and research assistants are well practised in taking the measure before the

commencement of study. However, risk of some discomfort cannot be avoided entirely and is a standard consequence of the procedure. Most importantly, the discomfort is temporary, and is alleviated as soon as the cuff is released (usually within 30 seconds).

In the rare even that any participant finds this too uncomfortable or painful, they may alert the researcher and they will be excluded from this measure, but may continue participating in the study.

Fasting

In order to determine fasting insulin concentrations, the patient is required to fast for 10 hours prior to having their blood taken. This is solely to ensure that the blood insulin levels are not affected be recent ingestion of food or drink, and that the measure is an accurate representation of the body's typical insulin function. Although fasting may lead to discomfort and irritability in some participants, it is not possible to achieve an accurate representation of insulin function without it. In order to minimize the discomfort, effort will be made to commence the blood withdrawal early in the morning so that the majority of the fasting is done during the previous night while sleeping.

Some participants may experience nausea or light headedness due to having not eaten or drunk in 10 hours. In order to manage this, participants will be allowed to sit or lie down whilst waiting for their blood to be taken, and effort will be made to ensure that the blood withdrawal is done as quickly and as efficiently as possible. Participants will also be given a small amount of water to sip if the nausea and/or feeling of faintness persist. If any participant is unable to manage the feelings of nausea or faintness, they are permitted to abstain from the blood withdrawal and ingest food or drink as they please. An on call doctor may also be contacted should any participant need medical attention.

Despite the discomfort associated with taking a fasting insulin measure, it is a regular and accepted test to monitor insulin function, and it is common procedure to go for blood tests following a 10 hour fast.

Blood test

Blood will be taken by a qualified nursing sister to ensure that the procedure is done effectively and safely with minimal pain and discomfort. As some participants may have a phobia of needles or may not want to have blood taken, participants will be

informed about this step in the consent form and will need to agree to this before participating. If any participants would not like to have blood taken, they may choose not to participate in the study.

In the unlikely event that there be any health problems or injuries as a result of the testing, and the on-call Doctor will be immediately contacted. There will also be a first aid kit available and well stocked, and a researcher with experience in First-Aid will be contacted.

Completion of questionnaires

The completion of the questionnaires can be considered invasive in that it requires the participants to provide private and personal information. However, all information provided will remain private and confidential, and the participants will all be reminded of this verbally (it is also stipulated in the information letter and the consent form). Participants will also be reminded why the questionnaire information is required: that it is a necessary aspect for the study so that the impact of lifestyle factors on cardiovascular disease can be assessed. Nevertheless, if any participant feels too uncomfortable to complete parts of, or entire questionnaires, they will not need to do so.

BENEFITS

After participating in this study, participants will have received:

Information on cardiovascular disease

During the introductory meeting, participants will have been informed about the dangers of cardiovascular disease and how it manifests in the body. Participants will also be informed about common risk factors for cardiovascular disease, as well as the dangers of not diagnosing/managing cardiovascular disease risk factors (hypertension, high glucose levels, and central obesity).

Free cardiovascular health screening

By virtue of participating in this study, participants will receive free and confidential cardiovascular health screening. Herein, participants will be measured for the main cardiovascular risk factors including central obesity, hypertension, and insulin resistance, but not including blood lipid profiles. It will be communicated to participants

that should they desire a full cardiovascular health screening, that they may consider further assessing their lipid profiles with their doctors, in addition to the measures they receive from the study. Participants' results will be communicated to them personally, and should any of the results be cause for concern, participant's will be notified as well as the on-call doctor, Dr. Jameson. Thereafter, appropriate protocol will be followed by the doctor regarding diagnosing the participant, and providing them with treatment option if necessary.

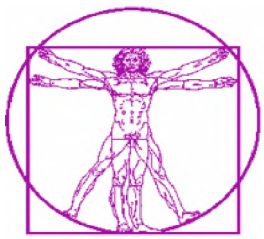
Education surrounding dangers of unhealthy eating habits and physical inactivity

As the main focus of the study is on modifiable risk behaviours, participants will be informed about the dangers of unhealthy habits (particularly consuming processed foods and foods high in sugar) as well as the dangers of being physically inactive, with the emphasis being how these habits can greatly increase a person's chance of developing cardiovascular disease. Participants will also be provided with a list of dietary and activity adjustments for them to make after they study should they wish to. This will act as a tool that the participants can use to make small improvements in their lifestyles, and will be based on each individual's circumstance.

A copy of personal measures

After participating in this study, each participant will have received a copy of their own personal cardiovascular risk measures as a reference point for any further testing.

APPENDIX 5



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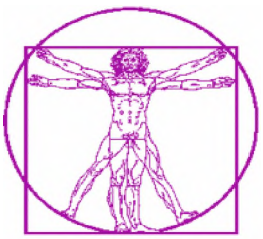


Human Kinetics and Ergonomics Department

INFORMED CONSENT AND INDEMNITY

For research involving human participants

(a) I, …… have been fully informed of the research project entitled "Cardiovascular disease risk in Black and white South African women: an Eastern Cape sample"

I have read the information sheet and understand the testing procedure that will take place. All testing procedures, associated risks and the benefits from partaking in this study have been verbally explained to me as well as in writing [*letter of information appended to this document*]. I have had ample opportunity to ask questions and to clarify any concerns or misunderstandings. I am satisfied that these have been answered satisfactorily. I understand that all data collected for publication purposes will be kept anonymous and all information gained in this regard will be treated confidentially. Furthermore, I consent to photographs, knowing that these will be altered to ensure my anonymity. I understand that I am able to withdraw from the study at any point and without any negative consequences, irrespective of external influences placed on me by the researcher.

In agreeing to participate in this research study I waive any legal recourse against the researchers from the Department of Human Kinetics and Ergonomics (HKE), Rhodes University, from claims resulting from personal injuries sustained whilst participating

in the above mentioned research. I am aware and fully understand that the Department of Human Kinetics and Ergonomics is not responsible for any injuries due to my personal negligence and non-compliance with instructions. This waiver shall be binding upon my heirs and personal representatives.

I have read and understood the above information, as well as the information provided in the letter accompanying this form. I therefore consent to voluntarily participate in this research project.

PARTICIPANT PROVIDING CONSENT:

_____ _____ _____

(Print Name) (Signed) (Date)

WITNESS:

_____ _____ _____

(Print Name) (Signed)
(Date)

PRINCIPAL RESEARCHER:

_____ _____ _____

(Print Name) (Signed) (Date)

APPENDIX 6

PRIVACY, ANONYMITY AND CONFIDENTIALITY ISSUES

To ensure anonymity of participants, each will be assigned an individual code which will be used to record their results. Their individual codes will be a combination of letters and numbers and will be assigned to them at random using a computer program. Only the primary researcher will have access to the information linking the participants' name to the code. When results are presented in the final thesis, only the individual codes will be used to differentiate between participants if necessary, but not their names.

To ensure privacy, all anthropometric and physiological measurements will be taken by the primary researcher and will be done in a closed off room that no other persons may enter during measuring except another female post-graduate assistant who will help with the measurements.

If any photos are taken, prior permission will be required from the participant in writing (consent form, Appendix 5). Participants will be assured verbally assured that they are not obligated to agree to this, and that if they do agree, their faces will be blocked out from any photos that are used in the thesis. Photos will also remain secured on the primary researcher's camera and will not be accessed by any other persons. Photos may also be used in electronic presentation or during lectures, however the identity of individuals will remain anonymous by blocking out the faces.

All measurements and results will be stored on the computer of the primary researcher and will not be sent out to other persons. At the end of the study, results will be archived on the Human Kinetics and Ergonomics results data base for security purposes; however individual participants' results will be labelled using their randomly assigned code. Stored data will only be available for re-use by the primary researcher or by supervisor Dr. Candice Christie, however participants' anonymity will still be secured if so.

The blood work results will also be archived at AMPATH laboratory but these results will remain secured and confidential; results will also be coded.

The primary researcher is aware that they are collecting sensitive personal and medical information and will retain this confidentiality by not discussing results with any persons apart from the participants whose information it is directly, or with supervisor Dr. Candice Christie if necessary.

APPENDIX 8

All medical terminology, as far as possible, is presented in English and isiXhosa. The remainder of the questionnaires are in English, and a translator will be present during the testing sessions should you require any assistance.

Participant screening questionnaire

1. Please indicate your SEX (male or female)	
2. Please circle your race	White; Black; Coloured; Indian; Other
3. Please give your AGE in years and months	
4. Are you able to read and understand basic ENGLISH?	
5. Have you experienced menstruation /*'exesheni'* within the last 30 days?	
6. If you answered 'NO' to question 5, please indicate with a tick whether you are a) pregnant / *'khulelue'* b) pre-menopausal or menopausal / *'kwixesha'* or c) unknown/other reason	a) b) c)
7. Have you ever been told that you have DIABETES / *'isifo seswekile'* by a medical health professional?	
8. Have you ever been told that you are HYPERTENSIVE / *'ukunyuka*	

kwegazi' by a medical health professional?	
9. If you have answered 'YES' to questions 7 or 8, please indicate with a tick if a) you are on any medications / *'amayeza'* for these conditions, b) what medication, and c) how long you have been on the medication for. If you answered 'NO' to questions 7 or 8, leave this question blank.	a) b) c)
10. Do you take any other chronic medication *'amayeza'* (medication you take every day)	
11. Have you had any MAJOR SURGERY / *'uqhaqho'* in the last 6 months?	
12. Are you a full-time employee of an institution, corporation, or business?	
13. Do you currently have any physical disability(s) / *'ukukhubazeka'* ?	

I [full name] _____ declare that the information provided above is truthful and accurate.

BASED ON THE INFORMATION PROVIDED, I UNDERSTAND THAT THERE IS A POSSIBILITY THAT I MAY NOT BE ALLOWED TO CONTINUE IF I DO NOT MEET THE CRITERIA, OR IF IT IS UNSAFE FOR ME TO PARTICIPATE. SHOULD I BE EXCLUDED FROM PARTICIPATION, I AM OF THE UNDERSTANDING THAT THE RESEARCH WILL COMMUNICATE THE REASONING BEHIND THIS TO ME.

_____ _____

Signed by participant Date

APPENDIX 9

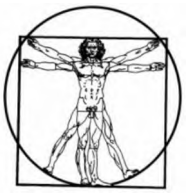
TIME	Type of food and/or drink. (If any ingredients were added to the preparation process, please add it as well i.e. oil, sugar, salt).	Quantity	Eaten at home (H) or at (W)
BREAKFAST			
MID-MORNING: Between breakfast and lunch			
LUNCH			
MID-AFTERNOON: Between lunch and dinner			
DINNER			
AFTER DINNER			

Participant code: ……

Day of the week: ……

Is this a typical day? (Yes or no): ……..

APPENDIX 10



DEPARTMENT OF HUMAN KINETICS AND ERGONOMICS

RHODES UNIVERSITY, GRAHAMSTOWN

[This research has been cleared by the Ethics Committee for research involving human subjects through the Department of Human Kinetics and Ergonomics]

ARE YOU INTERESTED IN:

- **HEALTH AND WELLBEING?**
- **RECEIVING FREE HEALTH SCREENING?**
- **HEALTHY LIFESTYLE HABITS?**
- **WAYS TO IMPROVE YOUR HEALTH?**

This study is focussed on assessing the cardiovascular health / *'Isofo sentliziyo'* of Black and White South African women residing in the Eastern Cape!

Some of the measures taken will include:

- **Weight *'ubunzima'***
- **Blood Pressure *'ukunyuka kwegazi'***
- **Blood glucose and Insulin 'isifo seswekile'**
- **Lifestyle habits such as diet, physical activity, smoking, and alcohol use**
- **AND MORE….. !**

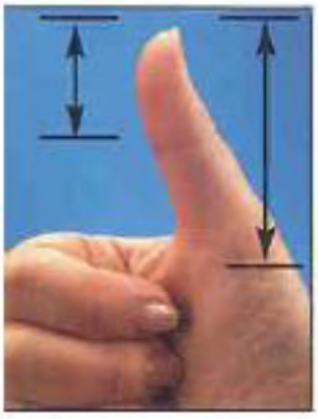
FOR MORE INFORMATION, DON'T HESITATE TO CONTACT Sandra Remsing on 0768984678 or g10r0007@campus.ru.ac.za

LOOKING FORWARD TO HEARING FROM YOU ☺

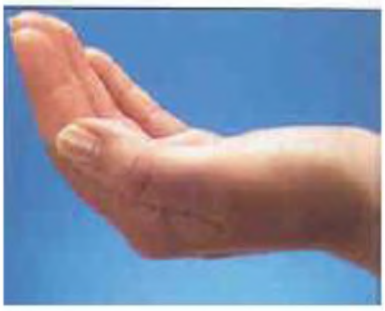
APPENDIX 11

The size of your fist refers to a medium sized fruit or vegetable
(i.e. apple, orange, potato)

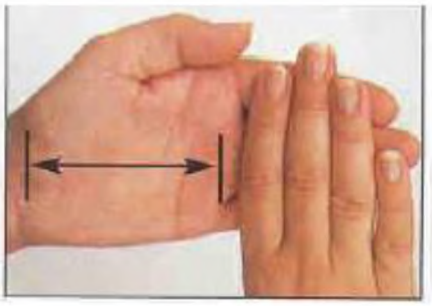
The size of your fingertip refers to one teaspoon
(i.e. sugar, butter on bread)



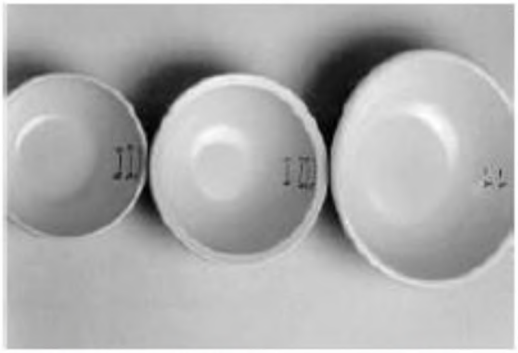
The size of your thumb refers to one tablespoon



Your cupped hand refers to approximately 45 g
(i.e. a cupped hand of chopped vegetables or rice)



The palm of your hand refers to a medium portion of cooked meat,
poultry or fish



Small Medium Large

Mug Cup

APPENDIX 12

Statistical analyses

T-tests; Grouping: Participants (Basic data start point.sta)
Group 1: W
Group 2: B

Variable	**Mean W**	Mean B	t-value	df	p	Valid N W	Valid N B	Std.Dev. W	Std.Dev. B	F-ratio Variances	p Variances
Age	38.8846	39.6705	-0.42180	164	0.673720	78	88	12.19323	11.78809	1.069918	0.757189
Height	164.5513	156.7318	8.02776	164	0.000000	78	88	6.35844	6.17826	1.059178	0.792032
weight	70.1538	84.1648	-4.93886	164	0.000002	78	88	13.43655	21.62198	2.589495	0.000031
BMI	25.8965	34.1893	-7.96726	164	0.000000	78	88	4.66984	8.07128	2.987319	0.000002
Ponderal index	15.7694	21.8417	-9.13922	164	0.000000	78	88	2.97019	5.15762	3.015294	0.000002
WC	79.8462	95.3352	-6.97278	164	0.000000	78	88	10.81074	16.76849	2.405897	0.000115
WSR	0.4794	0.6085	-8.60070	164	0.000000	78	88	0.08611	0.10487	1.482925	0.078732
BP systolic	116.2179	122.2159	-2.50564	164	0.013198	78	88	11.93559	17.90425	2.250215	0.000353
BP diastolic	70.4103	77.1250	-3.11767	164	0.002153	78	88	11.32568	15.74934	1.933731	0.003484
MAP	85.6795	92.1553	-3.01071	164	0.003019	78	88	10.89057	15.98890	2.155440	0.000701

T-tests; Grouping: Groups (Spreadsheet79)
Group 1: W
Group 2: B

Variable	**Mean W**	Mean B	t-value	df	p	Valid N W	Valid N B	Std.Dev. W	Std.Dev. B	F-ratio Variances	p Variances
KJ	6401.192	7243.596	-2.16873	93	0.032654	46	49	1796.473	1977.444	1.21162	0.518074
Kcal	1530.963	1731.262	-2.15339	93	0.033875	46	49	431.251	472.621	1.20106	0.537381
Protein (g)	69.420	58.446	2.22008	93	0.028843	46	49	29.649	17.296	2.93858	0.000326
Total fat (g)	64.262	61.729	0.56250	93	0.575127	46	49	22.652	21.240	1.13738	0.660198
Saturated fat (g)	22.233	18.440	2.15781	93	0.033519	46	49	9.031	8.099	1.24350	0.458103
Monounsaturated fat (g)	21.348	19.075	1.32779	93	0.187496	46	49	9.475	7.113	1.77465	0.052352
Polyunsaturated fat (g)	12.345	17.492	-3.39294	93	0.001017	46	49	6.745	7.947	1.38827	0.269427
Trans fat (g)	1.608	1.058	2.53486	93	0.012919	46	49	1.276	0.800	2.54307	0.001764
Carbohydrate (g)	141.815	214.933	-5.12671	93	0.000002	46	49	55.605	80.325	2.08678	0.014139
Alcohol (g)	10.425	0.922	3.39084	93	0.001024	46	49	19.208	3.899	24.27211	0.000000

T-tests; Grouping: Group (Spreadsheet1_(Recovered))
Group 1: W
Group 2: B

Variable	Mean W	Mean B	t-value	df	p	Valid N W	Valid N B	Std.Dev. W	Std.Dev. B	F-ratio Variances	p Variances
KJ	6401.192	7243.596	-2.16873	93	0.032654	46	49	1796.473	1977.444	1.21162	0.518074
kcal	1530.963	1731.262	-2.15339	93	0.033875	46	49	431.251	472.621	1.20106	0.537381
Protein (g)	69.420	58.446	2.22008	93	0.028843	46	49	29.649	17.296	2.93858	0.000326
Total fat (g)	64.262	61.729	0.56250	93	0.575127	46	49	22.652	21.240	1.13738	0.660198
S fat (g)	22.233	18.440	2.15781	93	0.033519	46	49	9.031	8.099	1.24350	0.458103
Mono fat (g)	21.348	19.075	1.32779	93	0.187496	46	49	9.475	7.113	1.77465	0.052352
Poly fat (g)	12.345	17.492	-3.39294	93	0.001017	46	49	6.745	7.947	1.38827	0.269427
Trans fat (g)	1.608	1.058	2.53486	93	0.012919	46	49	1.276	0.800	2.54307	0.001764
CHO (g)	141.815	214.933	-5.12671	93	0.000002	46	49	55.605	80.325	2.08678	0.014139
Total sugar (g)	35.016	46.721	-2.77036	91	0.006788	44	49	15.004	24.142	2.58908	0.001947
Alcohol (g)	10.425	0.922	3.39084	93	0.001024	46	49	19.208	3.899	24.27211	0.000000

T-tests; Grouping: Group (Spreadsheet83)
Group 1: W
Group 2: B

Variable	Mean W	Mean B	t-value	df	p	Valid N W	Valid N B	Std.Dev. W	Std.Dev. B	F-ratio Variances	p Variances
Total Active METminutes	1828.269	2688.864	-2.94786	164	0.003667	78	88	2238.104	1486.558	2.266713	0.000241
Transport METminutes	95.128	415.682	-4.41379	164	0.000018	78	88	271.278	588.209	4.701496	0.000000
Seated METminutes	3384.051	1952.045	7.21560	164	0.000000	78	88	1264.118	1286.734	1.036103	0.876643

Group=W
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	BMI	MAP
BMI	25.89650	4.66984	1.000000	0.355707
MAP	85.67949	10.89057	0.355707	1.000000

Group=B
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	BMI	MAP
BMI	34.18933	8.07128	1.000000	0.442757
MAP	92.15530	15.98890	0.442757	1.000000

Group=W
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC	BMI
WC	79.84615	10.81074	1.000000	0.853895
BMI	25.89650	4.66984	0.853895	1.000000

Group=B
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC	BMI
WC	95.33523	16.76849	1.000000	0.917697
BMI	34.18933	8.07128	0.917697	1.000000

Group=W
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC	MAP
WC	79.84615	10.81074	1.000000	0.386331
MAP	85.67949	10.89057	0.386331	1.000000

Group=B
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC	MAP
WC	95.33523	16.76849	1.000000	0.433420
MAP	92.15530	15.98890	0.433420	1.000000

Group=W
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WSR	MAP
WSR	0.47940	0.08611	1.000000	0.219670
MAP	85.67949	10.89057	0.219670	1.000000

Group=B
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WSR	MAP
WSR	0.60850	0.10487	1.000000	0.423231
MAP	92.15530	15.98890	0.423231	1.000000

Group=W
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	weight	WSR
weight	70.15385	13.43655	1.000000	0.627126
WSR	0.47940	0.08611	0.627126	1.000000

Group=B
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	weight	WSR
weight	84.16477	21.62198	1.000000	0.844448
WSR	0.60850	0.10487	0.844448	1.000000

Group=W
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	weight	MAP
weight	70.15385	13.43655	1.000000	0.407174
MAP	85.67949	10.89057	0.407174	1.000000

Group=B
Correlations (Basic data start point.sta)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	weight	MAP
weight	84.16477	21.62198	1.000000	0.433879
MAP	92.15530	15.98890	0.433879	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	WC (cm)
Carbohydrate (g)	141.8146	55.60481	1.000000	-0.528202
WC (cm)	80.4891	10.00472	-0.528202	1.000000

Variable	Means	Std.Dev.	Carbohydrate (g)	WC (cm)
Carbohydrate (g)	214.9329	80.32505	1.000000	-0.204805
WC (cm)	95.2551	16.67150	-0.204805	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Seated METminutes	Weight (kg)
Seated METminutes	3360.217	1305.313	1.000000	-0.067929
Weight (kg)	70.576	12.813	-0.067929	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Seated METminutes	Weight (kg)
Seated METminutes	1821.429	1109.054	1.000000	-0.130853
Weight (kg)	83.980	21.685	-0.130853	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	MAP (mmHg)
Carbohydrate (g)	141.8146	55.60481	1.000000	-0.107018
MAP (mmHg)	84.1884	10.67492	-0.107018	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	MAP (mmHg)
Carbohydrate (g)	214.9329	80.32505	1.000000	-0.296565
MAP (mmHg)	93.9116	16.00546	-0.296565	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	Weight (kg)
Carbohydrate (g)	141.8146	55.60481	1.000000	-0.395700
Weight (kg)	70.5761	12.81317	-0.395700	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	Weight (kg)
Carbohydrate (g)	214.9329	80.32505	1.000000	-0.245744
Weight (kg)	83.9796	21.68548	-0.245744	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	BMI (kg.m2)
Carbohydrate (g)	141.8146	55.60481	1.000000	-0.443535
BMI (kg.m2)	25.9001	4.72463	-0.443535	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	BMI (kg.m2)
Carbohydrate (g)	214.9329	80.32505	1.000000	-0.242707
BMI (kg.m2)	34.4916	7.98631	-0.242707	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	BP systolic (mmHg)
Carbohydrate (g)	141.8146	55.60481	1.000000	-0.006431
BP systolic (mmHg)	114.2609	11.69888	-0.006431	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	BP systolic (mmHg)
Carbohydrate (g)	214.9329	80.32505	1.000000	-0.262611
BP systolic (mmHg)	124.5102	19.04285	-0.262611	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	BP diastolic (mmHg)
Carbohydrate (g)	141.8146	55.60481	1.000000	-0.152379
BP diastolic (mmHg)	69.1522	10.99892	-0.152379	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Carbohydrate (g)	BP diastolic (mmHg)
Carbohydrate (g)	214.9329	80.32505	1.000000	-0.302049
BP diastolic (mmHg)	78.6122	15.29408	-0.302049	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Total fat (g)	Weight (kg)
Total fat (g)	64.26239	22.65185	1.000000	-0.360705
Weight (kg)	70.57609	12.81317	-0.360705	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Total fat (g)	Weight (kg)
Total fat (g)	61.72939	21.23981	1.000000	-0.184857
Weight (kg)	83.97959	21.68548	-0.184857	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Saturated fat (g)	Weight (kg)
Saturated fat (g)	22.23326	9.03085	1.000000	-0.209816
Weight (kg)	70.57609	12.81317	-0.209816	1.000000

Variable	Groups=B Correlations (Compilation of all correlation data.sta) Marked correlations are significant at p < .05000 N=49 (Casewise deletion of missing data)			
	Means	Std.Dev.	Saturated fat (g)	Weight (kg)
Saturated fat (g)	18.44020	8.09852	1.000000	-0.109818
Weight (kg)	83.97959	21.68548	-0.109818	1.000000

Variable	Groups=W Correlations (Compilation of all correlation data.sta) Marked correlations are significant at p < .05000 N=46 (Casewise deletion of missing data)			
	Means	Std.Dev.	Polyunsaturated fat (g)	Weight (kg)
Polyunsaturated fat (g)	12.34457	6.74510	1.000000	-0.275195
Weight (kg)	70.57609	12.81317	-0.275195	1.000000

Variable	Groups=B Correlations (Compilation of all correlation data.sta) Marked correlations are significant at p < .05000 N=49 (Casewise deletion of missing data)			
	Means	Std.Dev.	Polyunsaturated fat (g)	Weight (kg)
Polyunsaturated fat (g)	17.49224	7.94740	1.000000	-0.184708
Weight (kg)	83.97959	21.68548	-0.184708	1.000000

Variable	Groups=W Correlations (Compilation of all correlation data.sta) Marked correlations are significant at p < .05000 N=46 (Casewise deletion of missing data)			
	Means	Std.Dev.	Monounsaturated fat (g)	Weight (kg)
Monounsaturated fat (g)	21.34848	9.47518	1.000000	-0.258049
Weight (kg)	70.57609	12.81317	-0.258049	1.000000

Variable	Groups=B Correlations (Compilation of all correlation data.sta) Marked correlations are significant at p < .05000 N=49 (Casewise deletion of missing data)			
	Means	Std.Dev.	Monounsaturated fat (g)	Weight (kg)
Monounsaturated fat (g)	19.07510	7.11265	1.000000	-0.152835
Weight (kg)	83.97959	21.68548	-0.152835	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Weight (kg)	Active METminutes
Weight (kg)	70.576	12.813	1.000000	-0.089665
Active METminutes	1774.783	2219.375	-0.089665	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Weight (kg)	Active METminutes
Weight (kg)	83.980	21.685	1.000000	0.012723
Active METminutes	3005.714	1507.586	0.012723	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	KJ	Weight (kg)
KJ	6401.192	1796.473	1.000000	-0.438902
Weight (kg)	70.576	12.813	-0.438902	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	KJ	Weight (kg)
KJ	7243.596	1977.444	1.000000	-0.258549
Weight (kg)	83.980	21.685	-0.258549	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	KJ	BMI (kg.m2)
KJ	6401.192	1796.473	1.000000	-0.453435
BMI (kg.m2)	25.900	4.725	-0.453435	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	KJ	BMI (kg.m2)
KJ	7243.596	1977.444	1.000000	-0.288125
BMI (kg.m2)	34.492	7.986	-0.288125	1.000000

Groups=W
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=46 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	KJ	WC (cm)
KJ	6401.192	1796.473	1.000000	-0.431134
WC (cm)	80.489	10.005	-0.431134	1.000000

Groups=B
Correlations (Compilation of all correlation data.sta)
Marked correlations are significant at p < .05000
N=49 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	KJ	WC (cm)
KJ	7243.596	1977.444	1.000000	-0.230645
WC (cm)	95.255	16.671	-0.230645	1.000000

T-tests; Grouping: Group (Spreadsheet231)
Group 1: W
Group 2: B

Variable	Mean W	Mean B	t-value	df	p	Valid N W	Valid N B	Std.Dev. W	Std.Dev. B	F-ratio Variances	p Variances
Gross monthly income (R)	17242.31	4710.614	10.49943	164	0.000000	78	88	10834.93	2671.844	16.44486	0.000000
Years of completed education	17.05	11.659	14.15858	164	0.000000	78	88	2.80	2.095	1.78103	0.009258

T-tests; Grouping: Group (Spreadsheet241)
Group 1: W
Group 2: B

Variable	Mean W	Mean B	t-value	df	p	Valid N W	Valid N B	Std.Dev. W	Std.Dev. B	F-ratio Variances	p Variances
SBP (mmHg)	115.2297	129.6620	-6.64133	143	0.000000	74	71	11.50765	14.54151	1.596783	0.049154
DBP (mmHg)	69.4865	82.9014	-6.45302	143	0.000000	74	71	11.23868	13.71772	1.489817	0.093408
MAP (mmHg)	84.7342	98.4883	-6.83445	143	0.000000	74	71	10.67123	13.45478	1.589734	0.051319

T-tests; Grouping: Group (Spreadsheet243)
Group 1: B Without Diagnosis
Group 2: B All

Variable	Mean B Without Diagnosis	Mean B All	t-value	df	p	Valid N B Without Diagnosis	Valid N B All	Std.Dev. B Without Diagnosis	Std.Dev. B All	F-ratio Variances	p Variances
SBP (mmHg)	129.6620	122.2159	2.830619	157	0.005254	71	88	14.54151	17.90425	1.515979	0.072112
DBP (mmHg)	82.9014	77.1250	2.433830	157	0.016062	71	88	13.71772	15.74934	1.318138	0.231501
MAP (mmHg)	98.4883	92.1553	2.662154	157	0.008572	71	88	13.45478	15.98890	1.412159	0.135210

T-tests; Grouping: Group (Spreadsheet245)
Group 1: W Without known hypertension
Group 2: W All

Variable	Mean W Without known hypertension	Mean W All	t-value	df	p	Valid N W Without known hypertension	Valid N W All	Std.Dev. W Without known hypertension	Std.Dev. W All	F-ratio Variances	p Variances
SBP (mmHg)	115.2297	116.0127	-0.411398	151	0.681364	74	79	11.50765	11.99839	1.087108	0.719821
DBP (mmHg)	69.4865	70.1519	-0.361867	151	0.717957	74	79	11.23868	11.48476	1.044270	0.853328
MAP (mmHg)	84.7342	85.4388	-0.401113	151	0.688904	74	79	10.67123	11.02994	1.068360	0.776795

Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Weight (kg)	Seated MET-minutes
Weight (kg)	70.154	13.437	1.000000	0.034425
Seated MET-minutes	3384.051	1264.118	0.034425	1.000000

Group=B
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Weight (kg)	Seated MET-minutes
Weight (kg)	84.165	21.622	1.000000	-0.117367
Seated MET-minutes	1952.045	1286.734	-0.117367	1.000000

Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	BMI (kg/m2)	Seated MET-minutes
BMI (kg/m2)	25.897	4.670	1.000000	-0.054347
Seated MET-minutes	3384.051	1264.118	-0.054347	1.000000

Group=B
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	BMI (kg/m2)	Seated MET-minutes
BMI (kg/m2)	34.189	8.071	1.000000	-0.152167
Seated MET-minutes	1952.045	1286.734	-0.152167	1.000000

Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Weight (kg)	Total active MET-minutes
Weight (kg)	70.154	13.437	1.000000	-0.173719
Total active MET-minutes	1828.269	2238.104	-0.173719	1.000000

Group=B
Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	DBP (mmHg)	Total active MET-minutes
DBP (mmHg)	70.410	11.326	1.000000	-0.053030
Total active MET-minutes	1828.269	2238.104	-0.053030	1.000000

Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Total active MET-minutes	MAP (mmHg)
Total active MET-minutes	1828.269	2238.104	1.000000	-0.107748
MAP (mmHg)	85.679	10.891	-0.107748	1.000000

Group=B
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Total active MET-minutes	MAP (mmHg)

Group=B
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000

Group=B
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC (cm)	Total active MET-minutes
WC (cm)	95.335	16.768	1.000000	0.017526
Total active MET-minutes	2688.864	1486.558	0.017526	1.000000

N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	DBP (mmHg)	Total active MET-minutes
DBP (mmHg)	77.125	15.749	1.000000	0.307708
Total active MET-minutes	2688.864	1486.558	0.307708	1.000000

Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC (cm)	Total active MET-minutes
WC (cm)	79.846	10.811	1.000000	-0.242304
Total active MET-minutes	1828.269	2238.104	-0.242304	1.000000

Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WSR	Total active MET-minutes
WSR	0.486	0.066	1.000000	-0.193350
Total active MET-minutes	1828.269	2238.104	-0.193350	1.000000

Group=B
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WSR	Total active MET-minutes
WSR	0.608	0.105	1.000000	0.036671
Total active MET-minutes	2688.864	1486.558	0.036671	1.000000

Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC (cm)	Seated MET-minutes
WC (cm)	79.846	10.811	1.000000	0.053181
Seated MET-minutes	3384.051	1264.118	0.053181	1.000000

Variable	Group=B Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=88 (Casewise deletion of missing data) **Means**	Std.Dev.	WC (cm)	Seated MET-minutes
WC (cm)	95.335	16.768	1.000000	-0.159832
Seated MET-minutes	1952.045	1286.734	-0.159832	1.000000

Variable	Group=W Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=78 (Casewise deletion of missing data) **Means**	Std.Dev.	MAP (mmHg)	Seated MET-minutes
MAP (mmHg)	85.679	10.891	1.000000	-0.204477
Seated MET-minutes	3384.051	1264.118	-0.204477	1.000000

Variable	Group=B Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=88 (Casewise deletion of missing data) **Means**	Std.Dev.	MAP (mmHg)	Seated MET-minutes
MAP (mmHg)	92.155	15.989	1.000000	-0.380804
Seated MET-minutes	1952.045	1286.734	-0.380804	1.000000

Variable	Group=W Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=78 (Casewise deletion of missing data) **Means**	Std.Dev.	SBP (mmHg)	Seated MET-minutes
SBP (mmHg)	116.218	11.936	1.000000	-0.191993
Seated MET-minutes	3384.051	1264.118	-0.191993	1.000000

Variable	Group=B Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=88 (Casewise deletion of missing data) **Means**	Std.Dev.	SBP (mmHg)	Seated MET-minutes
SBP (mmHg)	122.216	17.904	1.000000	-0.359231
Seated MET-minutes	1952.045	1286.734	-0.359231	1.000000

Variable	Group=W Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=78 (Casewise deletion of missing data) **Means**	Std.Dev.	DBP (mmHg)	Seated MET-minutes
DBP (mmHg)	70.410	11.326	1.000000	-0.193766
Seated MET-minutes	3384.051	1264.118	-0.193766	1.000000

Variable	Group=B Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=88 (Casewise deletion of missing data) **Means**	Std.Dev.	DBP (mmHg)	Seated MET-minutes
DBP (mmHg)	77.125	15.749	1.000000	-0.375703
Seated MET-minutes	1952.045	1286.734	-0.375703	1.000000

Variable	Group=W Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=78 (Casewise deletion of missing data) **Means**	Std.Dev.	BMI (kg/m2)	Total active MET-minutes
BMI (kg/m2)	25.897	4.670	1.000000	-0.117243
Total active MET-minutes	1828.269	2238.104	-0.117243	1.000000

Variable	Group=B Correlations (Spreadsheet8) Marked correlations are significant at p < .05000 N=88 (Casewise deletion of missing data) **Means**	Std.Dev.	BMI (kg/m2)	Total active MET-minutes
BMI (kg/m2)	34.189	8.071	1.000000	-0.000467
Total active MET-minutes	2688.864	1486.558	-0.000467	1.000000

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet101)
By variable Group
Marked tests are significant at p <.05000

variable	Rank Sum group W	Rank Sum group B	U	Z	p-value	Z adjusted	p-value	Valid N group W	Valid N group B
Smoke daily	6278.000	7583.000	3197.000	-0.758729	0.448015	-1.76102	0.078236	78	88

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet101)
By variable Group

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet101)
By variable Group
Marked tests are significant at p <.05000

variable	Rank Sum group W	Rank Sum group B	U	Z	p-value	Z adjusted	p-value	Valid N group W	Valid N group B
Used to smoke	5848.000	8013.000	2767.000	-2.15000	0.031556	-4.09412	0.000042	78	88

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet101)
By variable Group
Marked tests are significant at p <.05000

variable	Rank Sum group W	Rank Sum group B	U	Z	p-value	Z adjusted	p-value	Valid N group W	Valid N group B
Do not smoke	6880.000	6981.000	3065.000	1.185817	0.235695	2.463607	0.013755	78	88

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet110)
By variable Group
Marked tests are significant at p <.05000

variable	Rank Sum W	Rank Sum B	U	Z	p-value	Z adjusted	p-value	Valid N W	Valid N B
Sufficiently Active	7320.000	6541.000	2625.000	2.609445	0.009069	3.147529	0.001647	78	88

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet110)
By variable Group
Marked tests are significant at p <.05000

variable	Rank Sum W	Rank Sum B	U	Z	p-value	Z adjusted	p-value	Valid N W	Valid N B
Insufficiently Active	5930.000	7931.000	2849.000	-1.88469	0.059473	-2.69620	0.007014	78	88
Minimally Active	6269.000	7572.000	3208.000	-0.725158	0.468350	-0.841120	0.400261	78	88

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet110)
By variable Activity levels
Marked tests are significant at p <.05000

variable	Rank Sum SA	Rank Sum MA	U	Z	p-value	Z adjusted	p-value	Valid N SA	Valid N MA
Caucasian	6786.000	5460.000	2379.000	2.348184	0.018866	2.862402	0.004205	78	78

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet110)
By variable Activity Levels
Marked tests are significant at p <.05000

variable	Rank Sum MA	Rank Sum IA	U	Z	p-value	Z adjusted	p-value	Valid N MA	Valid N IA
Caucasian	5538.000	6708.000	2457.000	-2.07172	0.038293	-2.46646	0.013646	78	78

Group=W
Correlations (Spreadsheet142)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Weight (kg)	BMI (kg/m2)	WC	WSR	SBP	DBP	MAP	Total Active MET-minutes
Income (R/month)	-0.132026	-0.162174	-0.010022	-0.023844	0.123902	0.095000	0.111127	-0.001491

Group=W
Correlations (Spreadsheet142)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Weight (kg)	BMI (kg/m2)	WC	WSR	SBP	DBP	MAP	Total Active MET-minutes
Years educated	-0.148533	-0.166473	-0.125426	-0.129970	-0.086744	-0.070402	-0.080499	0.063799

Group=B
Correlations (Spreadsheet142)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Weight (kg)	BMI (kg/m2)	WC	WSR	SBP	DBP	MAP	Total Active MET-minutes
Years educated	-0.129945	-0.181738	-0.206296	-0.238681	-0.280576	-0.200416	-0.236338	-0.353807

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet110)
By variable Activity Levels
Marked tests are significant at p <.05000

variable	Rank Sum MA	Rank Sum IA	U	Z	p-value	Z adjusted	p-value	Valid N MA	Valid N IA
Black South African	6688.000	8888.000	2772.000	-3.25324	0.001141	-4.24539	0.000022	88	88

Mann-Whitney U Test (w/ continuity correction) (Spreadsheet110)
By variable Activity Levels
Marked tests are significant at p <.05000

variable	Rank Sum SA	Rank Sum MA	U	Z	p-value	Z adjusted	p-value	Valid N SA	Valid N MA
Black South African	7612.000	7964.000	3696.000	-0.519276	0.603568	-0.605253	0.545012	88	88

Group=B
Correlations (Spreadsheet142)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Weight (kg)	BMI (kg/m2)	WC	WSR	SBP	DBP	MAP	Total Active MET-minutes
Income (R/month)	-0.098229	-0.083705	-0.031224	-0.016002	-0.074349	0.031381	-0.007144	0.033872

Group=B
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	BMI (kg/m2)	WSR
BMI (kg/m2)	34.18933	8.071284	1.000000	0.920726
WSR	0.60850	0.104866	0.920726	1.000000

Group=W
Correlations (Spreadsheet8)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	BMI (kg/m2)	WSR
BMI (kg/m2)	25.89650	4.669838	1.000000	0.868217
WSR	0.48569	0.066275	0.868217	1.000000

Group=B
Correlations (Spreadsheet142)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC	WSR
WC	95.33523	16.76849	1.000000	0.973133
WSR	0.60850	0.10487	0.973133	1.000000

Group=W
Correlations (Spreadsheet142)
Marked correlations are significant at p < .05000
N=78 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	WC	WSR
WC	79.84615	10.81074	1.000000	0.958762
WSR	0.48569	0.06628	0.958762	1.000000

Group=B
Correlations (Spreadsheet142)
Marked correlations are significant at p < .05000
N=88 (Casewise deletion of missing data)

Variable	Means	Std.Dev.	Weight (kg)	BMI (kg/m2)
Weight (kg)	84.16477	21.62198	1.000000	0.948494
BMI (kg/m2)	34.18933	8.07128	0.948494	1.000000

Variable	Group=W Correlations (Spreadsheet142) Marked correlations are significant at p < .05000 N=78 (Casewise deletion of missing data) **Means**	Std.Dev.	Weight (kg)	BMI (kg/m2)
Weight (kg)	70.15385	13.43655	1.000000	0.906017
BMI (kg/m2)	25.89650	4.66984	0.906017	1.000000

Variable	Group=B Correlations (Spreadsheet142) Marked correlations are significant at p < .05000 N=88 (Casewise deletion of missing data) **Means**	Std.Dev.	Weight (kg)	WC
Weight (kg)	84.16477	21.62198	1.000000	0.914601
WC	95.33523	16.76849	0.914601	1.000000

Variable	Group=W Correlations (Spreadsheet142) Marked correlations are significant at p < .05000 N=78 (Casewise deletion of missing data) **Means**	Std.Dev.	Weight (kg)	WC
Weight (kg)	70.15385	13.43655	1.000000	0.850169
WC	79.84615	10.81074	0.850169	1.000000

Variable	Group=B Correlations (Spreadsheet142) Marked correlations are significant at p < .05000 N=88 (Casewise deletion of missing data) **Means**	Std.Dev.	WSR	Seated MET-minutes
WSR	0.608	0.105	1.000000	-0.177507
Seated MET-minutes	1952.045	1286.734	-0.177507	1.000000

Variable	Group=W Correlations (Spreadsheet142) Marked correlations are significant at p < .05000 N=78 (Casewise deletion of missing data) **Means**	Std.Dev.	WSR	Seated MET-minutes
WSR	0.486	0.066	1.000000	-0.007489
Seated MET-minutes	3384.051	1264.118	-0.007489	1.000000

WHO/NMH/CCS/03.03
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STEPS Instrument
for NCD Risk Factors
(Core and Expanded Version 1.4)

The WHO STEPwise approach to Surveillance of noncommunicable diseases (STEPS)

Noncommunicable Diseases and Mental Health
World Health Organization
20 Avenue Appia, 1211 Geneva 27, Switzerland
For further information: ncd_surveillance@who.int

Copies of this document are available from:

Noncommunicable Diseases and Mental Health

World Health Organization
20 Avenue Appia
1211 Geneva 27
Switzerland
Fax: +41 22 791 4769
Email: ncd_surveillance@who.int
URL:
The content of this document is available on the Internet at: http://www.who.int/ncd_surveillance

STEPS Instrument (V1.4)

- This is the generic template which countries use to develop their own Instrument. It contains the CORE (unshaded and in double lined boxes) and EXPANDED items (shaded and in single lined boxes) and response options for Step 1, Step 2 and Step 3.
- The introductory statements, questions and response options should be translated and adapted where necessary to suit local conditions. *Italic typeface indicates where local examples should be inserted.*
- All CORE items should be included in the country-specific STEPS Instrument. Wording and response options for CORE questions should not be changed.
- Some countries may wish to expand the CORE questions. Recommendations for EXPANDED questions for the key risk factors are included in the shaded areas. These items may be modified but it is preferable to use them where possible.
- Additional questions can be added as OPTIONAL items to meet local needs. For example questions asked in previous surveys could be added to link to previous data.
- The use of the coding column (as is used in this Instrument) facilitates easy, fast and accurate manual data entry. Using this approach does not replace the need for double data entry for maximum quality control (see data coding manual).
- Relevant skip patterns are shown on the right hand side of the coding column. They should be carefully reviewed. Modifications to the skip patterns will be needed according to the final items included.

EXAMPLE- for a current smoker who eats 8 servings of fruit on a typical day

		Response		Coding column	Skip
S 1a	Do you currently smoke any **tobacco products**, such as cigarettes, cigars or pipes?	Yes No Don't know	1 2 7	1	*If No, go to Next Section*
D 1b	How many **servings** of fruit do you eat on one of those days? USE SHOWCARD	Number of servings Don't know	 77	0 8	

- "Do not know", "Don't remember", "Not applicable", "Refuse" are all response options but should be used only as a last resort. In such cases, the first two categories and the last two categories are coded as "7", "77" or "777" and as "8", "88", or "888", respectively depending on the number of numerals in the other response options. Missing responses should be entered as "9", "99" or "999" at time of data entry.
- Interviewer training is essential to develop thorough knowledge of the instrument format, introductory statements, questions, skip patterns, response options, use of show cards and prompts (where needed). The STEPS Field Manual is a guide and resource for training sessions.
- Undertaking pilot work with the draft country-specific STEPS instrument is essential.
- Each country will need to prepare a list of the question numbers (e.g. D1a) and response code cross-referenced with the standard numbers and codes used in this generic template. This cross-referencing will facilitate communication and comparison.

This document is available electronically on the NCD Surveillance website:
http://www.who.int/ncd_surveillance
Other documents cross-referenced in above are available by contacting *ncd_surveillance@who.int*

Identification Information:

This is a draft cover page. Each country will adapt this page to suit their local needs. The exact details to be collected in each country–specific STEPS instrument will vary depending on the survey design and implementation procedures. However, regardless of how the interview is administered (e.g., household, clinic or other) a process by which the cover page containing personal identifying information is stored should be carefully designed and must meet recommended ethical standards. Clear instructions on handling and storage of the cover sheets must be provided to the interviewers.

I 1	Country/district code	☐☐
I 2	Centre (Village name):	☐☐☐☐☐☐☐☐
I 3	Centre (Village code): (SEE NOTE BELOW)	☐☐☐
I 4	Interviewer code	☐☐☐
I 5	Date of completion of the questionnaire	☐☐/☐☐/☐☐☐☐ Day Month Year

Respondent Id Number ☐☐☐☐☐☐

	Consent			
I 6	Consent has been read out to respondent	Yes 1 No 2	☐	**If NO, read consent**
I 7	Consent has been obtained (verbal or written)	Yes 1 No 2	☐	**If NO, END**
I 8	Interview Language [*Insert Language*]	English 1 [Add others] 2	☐	
I 9	Time of interview (24 hour clock)			☐☐:☐☐
I 10	Family Name			
I 11	First Name			

Additional Information that may be helpful

I 12	Contact phone number where possible		
I 13	Specify whose phone	Work 1 Home 2 Neighbour 3 Other (specify) 4	☐

Note: Identification information I6 to I13 should be stored separately from the questionnaire because it contains confidential information. Please note: village code (or household code) is required as part of main instrument for data analyses.
Date of interview is required to calculate age.

Respondent Identification Number ☐☐ ☐☐ ☐☐

Step 1 Core Demographic Information

			Coding Column
C1	Sex (*Record Male / Female as observed*)	Male 1 Female 2	☐
C2	What is your date of birth? *If Don't Know, See Note* below and Go to C3*	Day ☐☐ Month ☐☐ Year ☐☐☐☐	
C3	How old are you?	Years	☐☐
C4	In total, how many years have you spent at school or in full-time study (excluding pre-school)?	Years	☐☐

EXPANDED: Demographic Information			
C5	What is your [*insert relevant ethnic group / racial group / cultural subgroup / others*] background?	[*Defined according to local demographic needs*]	☐☐
C6	What is the highest level of education you have completed? [*INSERT COUNTRY-SPECIFIC CATEGORIES*]	No formal schooling 0 1 Less than primary school 0 2 Primary school completed 0 3 Secondary school completed 0 4 High school completed 0 5 College/University completed 0 6 Post graduate degree 0 7	☐☐
C7	Which of the following best describes your main work status over the last 12 months? [*INSERT COUNTRY-SPECIFIC CATEGORIES*] USE SHOWCARD	Government employee 0 1 Non-government employee 0 2 Self-employed 0 3 Non-paid 0 4 Student 0 5 Homemaker 0 6 Retired 0 7 Unemployed (able to work) 0 8 Unemployed (unable to work) 0 9	☐☐
C8	How many people older than 18 years, including yourself, live in your household?	Number of people	☐☐
C9	Taking **the past year**, can you tell me what the average earnings of the household have been?	Per week ☐☐☐☐☐☐ OR per month ☐☐☐☐☐☐ OR per year ☐☐☐☐☐☐☐ *Go to Next Section* Refused 8	☐ *If Refused Go to C10*
C10	If you don't know the amount, can you give an **estimate** of the annual household income if I read some options to you? Is it *[READ OPTIONS]* *[INSERT QUINTILE VALUES]*	≤ Quintile (Q) 1 1 More than Q 1, ≤ Q 2 2 More than Q 2, ≤ Q 3 3 More than Q 3, ≤ Q 4 4 More than Q 4 5 Refused 8	☐

*Note: Coding Rule: Code "Don't Know" 7 (or 77 or 777 as appropriate).

Step 1 Core Behavioural Measures

CORE Tobacco Use (Section S)

Now I am going to ask you some questions about various health behaviours. This includes things like smoking, drinking alcohol, eating fruits and vegetables and physical activity. Let's start with smoking.

		Response		Coding Column	
S 1a	Do you currently smoke any **tobacco products**, such as cigarettes, cigars or pipes?	Yes No	1 2	☐	*If No, go to Next Section**
S 1b	**If Yes,** Do you currently smoke tobacco products **daily**?	Yes No	1 2	☐	*If No, go to Next Section**
S 2a	How old were you when you **first started** smoking daily?	Age (years) Don't remember	7 7	☐☐	*If Known, go to S 3*
S 2b	Do you remember how long ago it was? *(CODE 77 FOR DON'T REMEMBER)*	In Years OR in Months OR in Weeks		Years ☐☐ Months ☐☐ Weeks ☐☐	
S 3	On average, **how many** of the following do you smoke each day? *(RECORD FOR EACH TYPE)* *(CODE 88 FOR NOT APPLICABLE)* ☐☐☐☐☐☐☐☐	Manufactured cigarettes Hand-rolled cigarettes Pipes full of tobacco Cigars, cheroots, cigarillos ← Other (please specify):		☐☐ ☐☐ ☐☐ ☐☐ ☐☐	

EXPANDED: Tobacco Use

S 4	In the past, did you **ever** smoke **daily**?	Yes No	1 2	☐	*If No, go to S 6a*
S 5a	**If Yes,** How old were you when you **stopped** smoking **daily**?	Age (years) Don't remember	7 7	☐☐	*If Known, go to S 6a* *If 7 7, go to S 5b*
S 5b	How **long ago** did you stop smoking daily?	Years ago OR Months ago OR Weeks ago		Years ☐☐ Months ☐☐ Weeks ☐☐	
S 6a	Do you **currently use** any **smokeless tobacco** such as [*snuff, chewing tobacco, betel*] ?	Yes No	1 2	☐	*If No, go to S 8*
S 6b	**If Yes,** Do you **currently use smokeless tobacco** products **daily?**	Yes No	1 2	☐	*If No, go to S 8*

* Amend skip instructions if EXPANDED or OPTIONAL items are added to the Tobacco section

* Amend skip instructions if EXPANDED or OPTIONAL items are added to the Tobacco section

S 7	On average, how many **times a day** do you use …. *(RECORD FOR EACH TYPE)* ☐☐☐☐☐☐☐☐	Snuff, by mouth Snuff, by nose Chewing tobacco Betel, quid ← Other (specify)	☐☐ ☐☐ ☐☐ ☐☐ ☐☐
S 8	In the past, did you **ever use** smokeless tobacco such as [*snuff, chewing tobacco, or betel*] **daily**?	Yes 1 No 2	☐

CORE Alcohol Consumption (Section A)				
The next questions ask about the consumption of alcohol.				
		Response		**Coding Column**
A 1a	Have you **ever consumed** a drink that contains alcohol such as beer, wine, spirit, fermented cider *or [add other local examples]* ? *USE SHOWCARD or SHOW EXAMPLES*	Yes 1 No 2		☐ *If No, Go to Next Section**
A 1b	Have you consumed alcohol within the **past 12 months**?	Yes 1 No 2		☐ *If No, Go to Next Section**
A 2	In the past 12 months, **how frequently** have you had at least one drink? *(READ RESPONSES)* *USE SHOWCARD*	5 or more days a week 1 1-4 days per week 2 1-3 days a month 3 Less than once a month 4		☐
A 3	When you drink alcohol, **on average**, how many drinks do you have during one day?	Number Don't know 7 7		☐☐
A 4	During each of the **past 7 days**, how many standard drinks of any alcoholic drink did you have each day? *(RECORD FOR EACH DAY* *USE SHOWCARD)*	Monday Tuesday Wednesday Thursday Friday Saturday Sunday		☐☐ ☐☐ ☐☐ ☐☐ ☐☐ ☐☐ ☐☐

EXPANDED : Alcohol			
A 5	In the past 12 months, what was the **largest number** of drinks you had on a single occasion, counting all types of standard drinks together?	Largest number	☐☐
A 6a	**For men only:** In the past 12 months, on how many days did you have **five or more** standard drinks in a single day?	Number of days	☐☐☐
A 6b	**For women only**: In the past 12 months, on how many days did you have **four or more** standard drinks in a single day?	Number of days	☐☐☐

* Amend skip instructions if EXPANDED or OPTIONAL items are added to the Alcohol section

CORE Diet (Section D)

The next questions ask about the fruits and vegetables that you usually eat. I have a nutrition card here that shows you some examples of local fruits and vegetables. Each picture represents the size of a serving. As you answer these questions please think of a typical week in the last year.

D 1a	In a typical week, on how many days do you **eat fruit**? *USE SHOWCARD*	Number of days	☐☐	*If Zero days, go to D 2a*
D 1b	How many **servings** of fruit do you eat on **one** of those days? *USE SHOWCARD*	Number of servings	☐☐	
D 2a	In a typical week, on how many days do you **eat vegetables**? *USE SHOWCARD*	Number of days	☐☐	If Zero days, go to Section P
D 2b	How many **servings** of vegetables do you eat on one of those days? *USE SHOWCARD*	Number of servings	☐☐	

EXPANDED: Diet

D 3	What type of **oil or fat is most often** used for meal preparation in your household? *USE SHOWCARD* *SELECT ONLY ONE* ☐☐☐☐☐☐☐☐	Vegetable oil 0 1 Lard or suet 0 2 Butter or ghee 0 3 Margarine 0 4 Other 0 5 None in particular 0 6 None used 0 7 Don't know 7 7	☐☐

CORE Physical Activity (Section P)

Next I am going to ask you about the time you spend doing different types of physical activity. Please answer these questions even if you do not consider yourself to be an active person.
Think first about the time you spend doing work. Think of work as the things that you have to do such as paid or unpaid work, household chores, harvesting food, fishing or hunting for food, seeking employment. *[Insert other examples if needed]*

P 1	Does your work involve mostly sitting or standing, with walking for no more than 10 minutes at a time?	Yes 1 No 2	☐	*If Yes, go to P6*
P 2	Does your work involve vigorous activity, like [*heavy lifting, digging or construction work*] for at least 10 minutes at a time? *INSERT EXAMPLES & USE SHOWCARD*	Yes 1 No 2	☐	*If No, go to P4*
P 3a	In a typical week, on how many days do you do vigorous activities as part of your work?	Days a week	☐☐	
P 3b	On a typical day on which you do vigorous activity, how much time do you spend doing such work?	In hours and minutes OR in Minutes only	hrs ☐☐ : mins ☐☐ or minutes ☐☐☐	
P 4	Does your work involve moderate-intensity activity, like brisk walking *[or carrying light loads]* for at least 10 minutes at a time? *INSERT EXAMPLES & USE SHOWCARD*	Yes 1 No 2	☐	*If No, go to P6*
P 5a	In a typical week, on how many days do you do moderate-intensity activities as part of your work?	Days a week	☐☐	
P 5b	On a typical day on which you did moderate-intensity activities, how much time do you spend doing such work?	In hours and minutes OR in Minutes only	hrs ☐☐ : mins ☐☐ or minutes ☐☐☐	
P 6	How long is your typical work day?	Number of hours	hrs ☐☐	
Other than activities that you've already mentioned, I would like to ask you about the way you travel to and from places. For example to work, for shopping, to market, to place of worship. [*insert other examples if needed*]				
P 7	Do you walk or use a bicycle (*pedal cycle*) for at least 10 minutes continuously to get to and from places?	Yes 1 No 2	☐	*If No, go to P9*
P 8a	In a typical week, on how many days do you walk or bicycle for at least 10 minutes to get to and from places?	Days a week	☐☐	
P 8b	How much time would you spend walking or bicycling for travel on a typical day?	In hours and minutes OR in Minutes only	hrs ☐☐ : mins ☐☐ or minutes ☐☐☐	
The next questions ask about activities you do in your leisure time. Think about activities you do for recreation, fitness or sports *[insert relevant terms]*. Do not include the physical activities you do at work or for travel mentioned already.				
P 9	Does your [*recreation, sport or leisure time*] involve mostly sitting, reclining, or standing, with no physical activity lasting more than 10 minutes at a time?	Yes 1 No 2	☐	*If Yes, go to P 14*
P 10	In your [*leisure time*], do you do any vigorous activities like [*running or strenuous sports, weight lifting*] for at least 10 minutes at a time? *INSERT EXAMPLES & USE SHOWCARD*	Yes 1 No 2	☐	*If No, go to P 12*
P 11a	If Yes, In a typical week, on how many days do you do vigorous activities as part of your [*leisure time*]?	Days a week	☐☐	
P 11b	How much time do you spend doing this on a typical day?	In hours and minutes OR in Minutes only	hrs ☐☐ : mins ☐☐ or minutes ☐☐☐	

P 12	In your [*leisure time*], do you do any moderate-intensity activities like brisk walking,[*cycling or swimming*] for at least 10 minutes at a time? *INSERT EXAMPLES & USE SHOWCARD*	Yes 1 No 2	☐ *If No, go to P 14*
P 13a	If Yes In a typical week, on how many days do you do moderate-intensity activities as part of [*leisure time*]?	Days a week	☐☐
P 13b	How much time do you spend doing this on a typical day?	In hours and minutes OR in Minutes only	hrs ☐☐ : mins ☐☐ or minutes ☐☐☐
The following question is about sitting or reclining. Think back over the past 7 days, to time spent at work, at home, in *[leisure],* including time spent sitting at a desk, visiting friends, reading, or watching television, but do not include time spent sleeping.			
P 14	Over the past 7 days, how much time did you spend sitting or reclining on a typical day?	In hours and minutes OR in Minutes only	hrs ☐☐ : mins ☐☐ or minutes ☐☐☐

EXPANDED : History of High Blood Pressure				
H 1	When was your blood pressure last measured by a health professional?	Within past 12 months 1-5 years ago Not within past 5 yrs	1 2 3	☐
H 2	During the past 12 months have you been told by a doctor or other health worker that you have elevated blood pressure or hypertension?	Yes No	1 2	☐ *If No, skip to Next Section*
H 3	Are you currently receiving any of the following treatments for high blood pressure prescribed by a doctor or other health worker?			
H 3a	Drugs (medication) that you have taken in the last 2 weeks	Yes No	1 2	☐
H 3b	Special prescribed diet	Yes No	1 2	☐
H 3c	Advice or treatment to lose weight	Yes No	1 2	☐
H 3d	Advice or treatment to stop smoking	Yes No	1 2	☐
H 3e	Advice to start or do more exercise	Yes No	1 2	☐
H 4	During the past 12 months have you seen a traditional healer for elevated blood pressure or hypertension	Yes No	1 2	☐
H 5	Are you currently taking any herbal or traditional remedy for your high blood pressure?	Yes No	1 2	☐

EXPANDED : History of Diabetes				
H 6	Have you had your blood sugar measured in the last 12 months?	Yes No	1 2	☐
H 7	During the past 12 months, have you ever been told by a doctor or other health worker that you have diabetes?	Yes No	1 2	☐ *If No, skip to Next Section*
H 8	Are you currently receiving any of the following treatments for diabetes prescribed by a doctor or other health worker?			
H 8a	Insulin	Yes No	1 2	☐
H 8b	Oral drug (medication that you have taken in the last 2 weeks	Yes No	1 2	☐
H 8c	Special prescribed diet	Yes No	1 2	☐
H 8d	Advice or treatment to lose weight	Yes No	1 2	☐
H 8e	Advice or treatment to stop smoking	Yes No	1 2	☐
H 8f	Advice to start or do more exercise	Yes No	1 2	☐
H 9	During the past 12 months have you seen a traditional healer for diabetes?	Yes No	1 2	☐
H 10	Are you currently taking any herbal or traditional remedy for your diabetes?	Yes No	1 2	☐

Respondent Identification Number ☐☐ ☐☐ ☐☐

Step 2 Physical Measurements

Height and weight			Coding Column
M 1	Technician ID Code		☐☐☐
M 2a & 2b	Device IDs for height and weight	(2a) height ☐☐	(2b) weight ☐☐
M 3	Height	(in Centimetres)	☐☐☐.☐
M 4	Weight *If too large for scale, code 666.6*	(in Kilograms)	☐☐☐.☐
M 5	***(For women)*** Are you pregnant?	Yes 1 No 2	☐ *If Yes, Skip Waist*
Waist			
M 6	Technician ID		☐☐☐
M 7	Device ID for waist		☐☐
M 8	Waist circumference	(in Centimetres)	☐☐☐.☐

Blood pressure				Coding Column
M 9	Technician ID			☐☐☐
M 10	Device ID for blood pressure			☐☐
M 11	Cuff size used	Small 1 Normal 2 Large 3		☐
M 12a	Reading 1 **Systolic BP**	Systolic	mmHg	☐☐☐
M 12b	**Diastolic BP**	Diastolic	mmHg	☐☐☐
M 13a	Reading 2 **Systolic BP**	Systolic	mmHg	☐☐☐
M 13b	**Diastolic BP**	Diastolic	mmHg	☐☐☐
M 14a	Reading 3 **Systolic BP**	Systolic	mmHg	☐☐☐
M 14b	**Diastolic BP**	Diastolic	mmHg	☐☐☐
M 15	During the past two weeks, have you been treated for high blood pressure with drugs (medication) prescribed by a doctor or other health worker ?	Yes 1 No 2		☐

SELECTED EXPANDED ITEMS			
M 16	Hip circumference	(in Centimetres)	☐☐☐.☐
Heart Rate (Record if automatic blood pressure device is used)			
M 17a	Reading 1	Beats per minute:	☐☐☐
M 17b	Reading 2	Beats per minute:	☐☐☐
M 17c	Reading 3	Beats per minute:	☐☐☐

Step 3 Biochemical Measurements

CORE	Blood glucose		Coding Column
B 1	During the last 12 hours have you had anything to eat or drink, other than water?	Yes 1 No 2	☐
B 2	Technician ID Code		☐☐☐
B 3	Device ID code		☐☐
B 4	Time of day blood specimen taken (24 hour clock)		hrs ☐☐ : mins ☐☐
B 5	Blood glucose	Low 1 High 2 Unable to assess 3	mmol/l ☐☐.☐ ☐
CORE	Blood Lipids		
B 6	Technician ID Code		☐☐☐
B 7	Device ID code		☐☐
B 8	Total cholesterol	Low 1 High 2 Unable to assess 3	mmol/l ☐☐.☐☐ ☐

SELECTED EXPANDED ITEMS			
B 9	Technician ID Code		☐☐☐
B 10	Device ID code		☐☐
B 11	Triglycerides	Low 1 High 2 Unable to assess 3	mmol/l ☐☐.☐☐ ☐
B 12	Technician ID Code		☐☐☐
B 13	Device ID code		☐☐
B 14	HDL Cholesterol	Low 1 High 2 Unable to assess 3	mmol/l ☐.☐☐ ☐