CARDIOVASCULAR DISEASE RISK IN BLACK AFRICAN FEMALES AND THE EFFICACY OF A WALKING PROGRAMME ON BLOOD PRESSURE IN A SUB-SAMPLE

BY

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THESIS

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ABSTRACT

The purpose of the study was to investigate the cardiovascular disease (CVD) risk profile of black African females in the Makana region, Eastern Cape, South Africa. Baseline measures from 40 participants, who met the criteria, were compared against the 2003 South African Demographic and Health Survey (SADHS) and the 2013 South African National Health and Nutritional Examination Survey (SANHANES-1). The risk factors measured were anthropometric (stature, body mass and body mass index (BMI)), morphological (waist circumference (WC), fat mass and lean mass), cardiovascular (heart rate and blood pressure (BP)), physical activity (step count and energy expenditure), biochemical (glycated haemoglobin and full blood lipid profile) and behavioural (alcohol and tobacco use). Results showed significantly higher ($p \leq 0.05$) values for overweight/obesity (BMI 37.60 $kg.m^{-2}$; WC 1130.58 mm; fat mass 45.23%) and high BP (130/88 mmHg) compared to the previous national surveys, highlighting these CVD risk factors as problematic. The subsequent sub-study aimed to assess the efficacy of a pedometer-based walking intervention on high BP. The walking programme (n=25) was based on individual step goals to be completed at a moderate-intensity on five $days.week^{-1}$ for 12 weeks. The same measurements were taken at monthly intervals, Week 0, Week 4, Week 8 and Week 12, with the addition of dietary intake and fitness level, and the exclusion of the behavioural variables. There were no significant differences ($p \leq 0.05$) in systolic and diastolic BP with the exercise intervention, although there was a strong, negative relationship with time for diastolic BP ($r^2=0.9857$). This trend suggests that the lack of significance may be a result of poor compliance and/or the small sample size. Individual results, however, showed no compliance-result relationship for the two risk factors of interest: overweight/obesity and high BP. Future recommendations include supervised or group-based exercise interventions to improve compliance, and the addition of resistance training to the aerobic programme.

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CHAPTER 1

INTRODUCTION

1.1. BACKGROUND TO THE STUDY

The disease profile of the world is changing very rapidly, especially in low- and middle-income countries (WHO, 2005). There has been an accelerated growth in non-communicable diseases (NCDs) which places them as the leading health burden worldwide (Yach *et al*., 2004; Beaglehole *et al*., 2011; Bloom *et al*., 2011; Laslett *et al*., 2012; Smith *et al*., 2012). This group of diseases, often regarded as 'diseases of lifestyle', are affecting all countries across income groups, age, race and sex demographics which demonstrates its extensive effects (Beaglehole *et al*., 2011). In South Africa, a developing country, the epidemiological transition has created a unique situation whereby both communicable diseases and NCDs are placing strain on the country's health profile which has recently expanded to a 'quadruple burden of disease' (Goosen *et al*., 2003). This NCD epidemic has created a need for a better understanding locally of both the NCD prevalence and the associated risk factors. This information can then be translated into effective health policies, programmes and services, and can further be applied to cost-effective approaches (Lemon, 2004; SANHANES-1, 2013). The effect of this would be significant, especially in the poorer provinces of South Africa such as the Northern Province and the Eastern Cape Province – two areas with the lowest standards of living (Stats SA, 2006). The Eastern Cape Province is specifically burdened by this shift in disease profile, evident by their reported top ten leading causes of death in 2010, of which half were attributed to NCDs (Stats SA, 2013). These listed NCDs were shown to be more prevalent in females than in males, with the exception of chronic lower respiratory diseases (Stats SA, 2013). Unfortunately, there are no race-specific data in this regard, so it is difficult to draw conclusions at this point as to possible different health risks.

Cardiovascular disease (CVD) is the biggest contributor to NCDs, and remains one of the main causes of premature deaths on a global scale (Gaziano, 2005; Bloom *et al*., 2011). This is particularly true in developing regions which account for an estimated 80% of all CVD mortality and can be largely attributed to poor lifestyles and associated increase in risk factors (Assmann *et al*., 1999; Bonow *et al*., 2002; Levenson *et al*., 2002; Yusuf and Ounpuu, 2003; Gaziano, 2005; Perk *et al*., 2012). In South Africa in 2008 it was reported that CVD was the second leading cause of death across all ages, with an 11% mortality rate, after HIV/AIDS (Alwan *et al*., 2011; Matsha *et al*., 2012). Little information exists in these low- and middle-income countries, however, especially in vulnerable populations for whom modifiable risk factors have previously been rare, as is associated with urbanisation, and health-care resources already over-burdened (Sliwa *et al*., 2008; Rayner, 2010). In general, this describes the majority black African race in South Africa (Bourne *et al*., 2002).

There are numerous modifiable risk factors for CVD. These include high blood pressure (BP), overweight/obesity, low cardiovascular fitness, poor diet, tobacco use, alcohol abuse, diabetes, high cholesterol and psychological distress. Among these nine listed risk factors, high BP/hypertension is responsible for more deaths worldwide than any other (Ezzati *et al*., 2002; Gaziano *et al*., 2005). The previously published South African national surveys (1998, 2003 and 2013) accurately report on these risk factors although specific data on municipalities, such as the Makana Municipality, do not exist. The most recent survey, SANHANES-1 (2013), designates low cardiovascular fitness, obesity and high systolic blood pressure (SBP) as the three most prevalent risk factors within the general population. Importantly, the World Health Organisation (WHO) stated that over three-quarters of all CVD mortality could be prevented with adequate behavioural changes (Perk *et al*., 2012), including increased physical activity and decreased energy consumption (Beaglehole and Yach, 2003; Alberts *et al*., 2005; Gilbert-Ouimet *et al*., 2012). These behavioural changes are needed in areas such as the Eastern Cape, South Africa, where the gross domestic product is less than half the national average (Lemon, 2004; Stats SA, 2006) and so it can be assumed that access to health services is minimal. The Makana Municipality in the Eastern Cape, which is mainly populated by black Africans (78%), has a very high unemployment and poverty rate (Stats SA, 2013)

and therefore this region would benefit greatly from cost-effective prevention and treatment approaches.

Hypertension, which is the primary risk factor for CVD, affects approximately one billion people worldwide, a number that is expected to reach 1.56 billion by 2025 (Kearney *et al*., 2005; Gasperin *et al*., 2009; Kumar, 2013). Furthermore, the burden of hypertensive individuals in developing countries outweighs that of developed countries by more than twofold (Kearney *et al*., 2005). The 1998 South African Demographic and Health Survey (SADHS), used as opposed to the 2003 version due to inaccurate BP measurements, reported that an estimated six million people were living with hypertension in the country, 22.9% males and 24.6% females (SADHS, 1998; Armstrong and Bull, 2006; Seedat and Rayner, 2012). Over a decade later, and despite advances in treatment and prevention, the prevalence of hypertension still remains high at 10.2% across the South African adult population (SANHANES-1, 2013). In general, the black race is known to have a unique susceptibility to hypertension compared to other ethnic groups (Lopes *et al*., 2003; Mainous *et al*., 2004; Hertz *et al*., 2005; Brown, 2006; Forouhi and Sattar, 2006; Howard *et al*., 2006; Sharp *et al*., 2008; Thorpe *et al*., 2008; Tullio, 2008; Carson *et al*., 2011; Redmond *et al*., 2011; Boykin *et al*., 2012), although little comparable data exist for black Africans (Tibazarwa *et al*., 2009). The few studies which have investigated this difference in Africa have yielded similar findings (Dennison *et al*., 2007; Sliwa *et al*., 2008; Tibazarwa *et al*., 2009; Stewart *et al*., 2011), although no such comparative research has been undertaken in the Eastern Cape Province.

From an economic perspective, hypertension, and more generally CVD, is a major healthcare cost in South Africa. Seedat and Rayner (2012) found the hypertension burden to constitute 7.5% of the direct total healthcare cost. The impact of untreated, or poorly treated, hypertension contributes to the burden of disease including strokes, heart attack, kidney disease or failure and congestive heart failure (SADHS, 2003). The 1998 SADHS identified substantial ethnic differences in awareness, treatment and control of hypertension with black Africans having the lowest rates in all three categories. The SADHS (2003) report paints a slightly different picture, with

black Africans having the second worst treatment status in both male and female categories. This finding may be due to the poorer socio-economic status of black African people and their limited access to health care (Rayner, 2010). This burden of disease can be avoided if early detection and management occurs, thus highlighting the need to address this epidemic in the general population, and specifically the under-researched and, as a generalisation, impoverished black African group.

The treatment options available to combat hypertension can be broadly divided into two categories: pharmacological and non-pharmacological options (Brown *et al*., 2003; Joint National Committee, 2004; Cheung *et al*., 2005; Svetkey *et al*., 2005; Yiannakopoulou *et al.*, 2005; Lewanczuk and Tobe, 2007). Pharmacological treatment primarily involves antihypertensive medications, which have been associated with poor compliance, adverse side-effects and high costs (Kjellgren *et al.*, 1995; Cheung *et al*., 2005; Yiannakopoulou *et al*., 2005; Lewanczuk and Tobe, 2007). The South African Hypertension Guidelines recommend non-pharmacological treatment in the form of lifestyle modifications as initial therapy in individuals with mild-to-moderate hypertension (Svetkey *et al*., 2005). These lifestyle modifications primarily come in the form of education, stress management, dietary approaches and increased physical activity.

Exercise has been recommended as an important lifestyle modification for the prevention and management of hypertension (Higashi *et al*., 1999; Armstrong and Bull, 2006; Bravata *et al*., 2007; Sohn *et al.*, 2007; Lee *et al*., 2010). Importantly, it has been noted that fewer women than men are active (Peterson *et al*., 2005), which highlights the sub-population who could benefit most from this type of intervention. Black females in South Africa have the highest overweight (24.9%) and obesity (39.9%) rates of all population groups (SANHANES-1, 2013). This finding can be attributed to several factors, of which poor dietary habits and decreased physical activity are two of the biggest (Kaplan and Opie, 2006; Stupar *et al*., 2012). Since regular aerobic exercise and BP are inversely associated, there is justification for the promotion of physical activity in black African females with this risk factor (Higashi *et al*., 1999). Furthermore, aerobic exercise in the form of walking is one of the simplest

exercises for hypertensive patients of all age groups, thus making its promotion feasible in poorer populations (Higashi *et al*., 1999; Sohn *et al*., 2007; Lee *et al*., 2010).

Although the majority of research has found walking to be beneficial in reducing BP, the optimal 'dose' of exercise for improved systolic and diastolic BP remains ill-defined, with findings being inconsistent (Higashi *et al*., 1999; Murtagh *et al*., 2005; Tully *et al*., 2005; Sohn *et al*., 2007; Tully *et al*., 2007; Murphy *et al*., 2007; Lee *et al*., 2010). Previous walking intervention findings have been unsupervised and typically last 8-12 weeks, with longer intervention periods resulting in more beneficial results, although one must also consider the feasibility and attrition rates of prolonged programmes (Kang *et al*., 2009; Lee *et al*., 2010). Another layer to this issue is the differing responses of sub-populations to exercise, and since there has been very little research done on the effect of walking programmes on BP in African people, more research is warranted.

1.2. STATEMENT OF THE PROBLEM AND RESEARCH AIMS

Cardiovascular disease is rapidly increasing in South Africa and so improved knowledge of the populations' disease risk profiles are required. Due to high levels of poverty and recent urbanisation in black African females in the Makana region, the purpose of the first part of this study was to add new data to this literature. Furthermore, there has been little research done on the impact of physical activity on BP within an African context. Therefore, the second aim of this study was to assess the efficacy of a 12 week walking programme in a group of mild-to-moderately hypertensive, black African women from the same area.

1.3. RESEARCH HYPOTHESES

It was expected that the cardiovascular disease risk of black African females in the Makana region of the Eastern Cape would be higher than the existing South African

population-matched data. It was further expected that a 12 week progressive walking programme would positively affect BP, and other cardiovascular risk factors, in a specific sub-sample.

1.4. STATISTICAL HYPOTHESES

1) There will be no difference at baseline in cardiovascular disease risk compared to previous South African surveys/existing norms.

a) H_0: $\mu_{CVR1} = \mu_{CVR2}$
H_a: $\mu_{CVR1} \neq \mu_{CVR2}$

2) With the 12 week walking programme systolic blood pressure and diastolic blood pressure will not change with:

Group effect

a) H_0: $\mu_{SBPC} = \mu_{SBPE}$
H_a: $\mu_{SBPC} \neq \mu_{SBPE}$

b) H_0: $\mu_{DBPC} = \mu_{DBPE}$
H_a: $\mu_{DBPC} \neq \mu_{DBPE}$

Time effect

a) H_0: $\mu_{SBP0} = \mu_{SBP4} = \mu_{SBP8} = \mu_{SBP12}$
H_a: $\mu_{SBP0} \neq \mu_{SBP4} \neq \mu_{SBP8} \neq \mu_{SBP12}$

b) H_0: $\mu_{DBP0} = \mu_{DBP4} = \mu_{DBP8} = \mu_{DBP12}$
H_a: $\mu_{DBP0} \neq \mu_{DBP4} \neq \mu_{DBP8} \neq \mu_{DBP12}$

Interaction effect

a) H_0: No interaction effect
H_a: Interaction effect

3) With the 12 week walking programme waist circumference will not change with:

Group effect

a) H_0: $\mu_{WCC} = \mu_{WCE}$

H_a: $\mu_{WCC} \neq \mu_{WCE}$

Time effect

a) H_0: $\mu_{WC0} = \mu_{WC4} = \mu_{WC8} = \mu_{WC12}$

H_a: $\mu_{WC0} \neq \mu_{WC4} \neq \mu_{WC8} \neq \mu_{WC12}$

Interaction effect

a) H_0: No interaction effect

H_a: Interaction effect

4) With the 12 week walking programme body composition will not change with:

Group effect

a) H_0: $\mu_{LMC} = \mu_{LME}$

H_a: $\mu_{LMC} \neq \mu_{LME}$

b) H_0: $\mu_{FMC} = \mu_{FME}$

H_a: $\mu_{FMC} \neq \mu_{FME}$

Time effect

a) H_0: $\mu_{LM0} = \mu_{LM4} = \mu_{LM8} = \mu_{LM12}$

H_a: $\mu_{LM0} \neq \mu_{LM4} \neq \mu_{LM8} \neq \mu_{LM12}$

b) H_0: $\mu_{FM0} = \mu_{FM4} = \mu_{FM8} = \mu_{FM12}$

H_a: $\mu_{FM0} \neq \mu_{FM4} \neq \mu_{FM8} \neq \mu_{FM12}$

Interaction effect

a, b) H_0: No interaction effect

H_a: Interaction effect

5) With the 12 week walking programme resting heart rate will not change with:

Group effect

a) H_0: $\mu_{HRC} = \mu_{HRE}$

H_a: $\mu_{HRC} \neq \mu_{HRE}$

Time effect

a) H_0: $\mu_{HR0} = \mu_{HR4} = \mu_{HR8} = \mu_{HR12}$

H_a: $\mu_{HR0} \neq \mu_{HR4} \neq \mu_{HR8} \neq \mu_{HR12}$

Interaction effect

a) H_0: No interaction effect

H_a: Interaction effect

6) With the 12 week walking programme fitness will not change with:

Group effect

a) H_0: $\mu_{FC} = \mu_{FE}$

H_a: $\mu_F \neq \mu_{FE}$

Time effect

a) H_0: $\mu_{F0} = \mu_{F4} = \mu_{F8} = \mu_{F12}$

H_a: $\mu_{F0} \neq \mu_{F4} \neq \mu_{F8} \neq \mu_{F12}$

Interaction effect

a) H_0: No interaction effect

H_a: Interaction effect

7) With the 12 week walking programme the lipid profile will not change with:

Group effect

a) H_0: $\mu_{TCC} = \mu_{TCE}$

H_a: $\mu_{TC} \neq \mu_{TCE}$

b) H_0: $\mu_{TGC} = \mu_{TGE}$

H_a: $\mu_{TGC} \neq \mu_{TGE}$

c) H_0: $\mu_{LDL\text{-}CC} = \mu_{LDL\text{-}CE}$

H_a: $\mu_{LDL\text{-}CC} \neq \mu_{LDL\text{-}CE}$

d) H_0: $\mu_{HDL\text{-}CC} = \mu_{HDL\text{-}CE}$

H_a: $\mu_{HDL\text{-}CC} \neq \mu H_{DL\text{-}CE}$

Time effect

a) H_0: $\mu_{TC0} = \mu_{TC4} = \mu_{TC8} = \mu_{TC12}$

H_a: $\mu_{TC0} \neq \mu_{TC4} \neq \mu_{TC8} \neq \mu_{TC12}$

b) H_0: $\mu_{TG0} = \mu_{TG4} = \mu_{TG8} = \mu_{TG12}$

H_a: $\mu_{TG0} \neq \mu_{TG4} \neq \mu_{TG8} \neq \mu_{TG12}$

c) H_0: $\mu_{LDL\text{-}C0} = \mu_{LDL\text{-}C4} = \mu_{LDL\text{-}C8} = \mu_{LDL\text{-}C12}$

H_a: $\mu_{LDL\text{-}C0} \neq \mu_{LDL\text{-}C4} \neq \mu_{LDL\text{-}C8} \neq \mu_{LDL\text{-}C12}$

d) H_0: $\mu_{HDL\text{-}C0} = \mu_{HDL\text{-}C4} = \mu_{HDL\text{-}C8} = \mu_{HDL\text{-}C12}$

H_a: $\mu_{HDL\text{-}C0} \neq \mu_{HDL\text{-}C4} \neq \mu_{HDL\text{-}C8} \neq \mu_{HDL\text{-}C12}$

Interaction effect

a, b, c, d) H_0: No interaction effect

H_a: Interaction effect

8) With the 12 week walking programme glycated haemoglobin will not change with:

Group effect

a) H_0: $\mu_{GHC} = \mu_{GHE}$

$H_{a:}$ $\mu_{GH} \neq \mu_{GHE}$

Time effect

a) H_0: $\mu_{GH0} = \mu_{GH12}$

$H_{a:}$ $\mu_{GH0} \neq \mu_{GH12}$

Interaction effect

a) H_0: No interaction effect

H_a: Interaction effect

Where:

CVR	= cardiovascular risk	SBP	= systolic blood pressure
C	= control group	E	= exercise group
DBP	= diastolic blood pressure	0	= 0 weeks (pre-walking programme)
4	= 4 weeks (during walking programme)	8	= 8 weeks (during walking programme)
12	= 12 weeks (post-walking programme)	WC	= waist circumference
LM	= lean mass	FM	= fat mass
HR	= heart rate	F	= fitness
TC	= total cholesterol	TG	= triglycerides
LDL-C	= low-density lipoprotein cholesterol	HDL-C	= high-density lipoprotein cholesterol
GC	= glycated haemoglobin		

1.5. STRUCTURE OF THE THESIS

This thesis is presented in six chapters. The first chapter outlines the background to the study and identifies the research problem with associated hypotheses. Chapter 2 is a critical review of related literature, with specific emphasis on cardiovascular disease and its primary risk factor, blood pressure, within the South African context. The prevalence of these disease burdens is presented within different South African population groups, and possible treatment and prevention options are discussed. This is because the main area of interest of this research is to better understand the cardiovascular disease risk profile of Eastern Cape black African women and further to test the efficacy of an exercise intervention against high blood pressure. Chapter 3 is a detailed methodology which communicates the design, recruitment and testing processes of this study and is comprehensive enough to allow for replication. Chapter 4 reports on the results of this research, both graphically and textually, with results being supported with statistical analyses. This leads into the fifth chapter which discusses the findings with reference to previous literature, as well as personal opinions. In conclusion, Chapter 6 summarises the key findings and links these to the initial statistical hypotheses. Finally, recommendations for future research are given to assist in improving the knowledge base in this specific ‘exercise is medicine’ field of study. A Bibliography of all cited material is then provided, as well as list of appendices which were relevant throughout the research process.

CHAPTER 2

REVIEW OF LITERATURE

2.1 GLOBAL HEALTH PROFILE

The disease profile of the world is changing and this shift is occurring at a much faster rate in low- and middle-income countries (WHO, 2005). In 2005, 61% of all global deaths stemmed from non-communicable diseases (NCDs) (Figure 1) and in 2008 this burden had increased to 63% (WHO, 2011; Wagner and Brath, 2012). This places NCDs, led by cardiovascular disease (CVD), as the largest burden on human health worldwide, having overtaken communicable diseases (CDs) (Yach *et al*., 2004; Beaglehole *et al*., 2011; Bloom *et al*., 2011; Laslett *et al*., 2012; Smith *et al*., 2012).

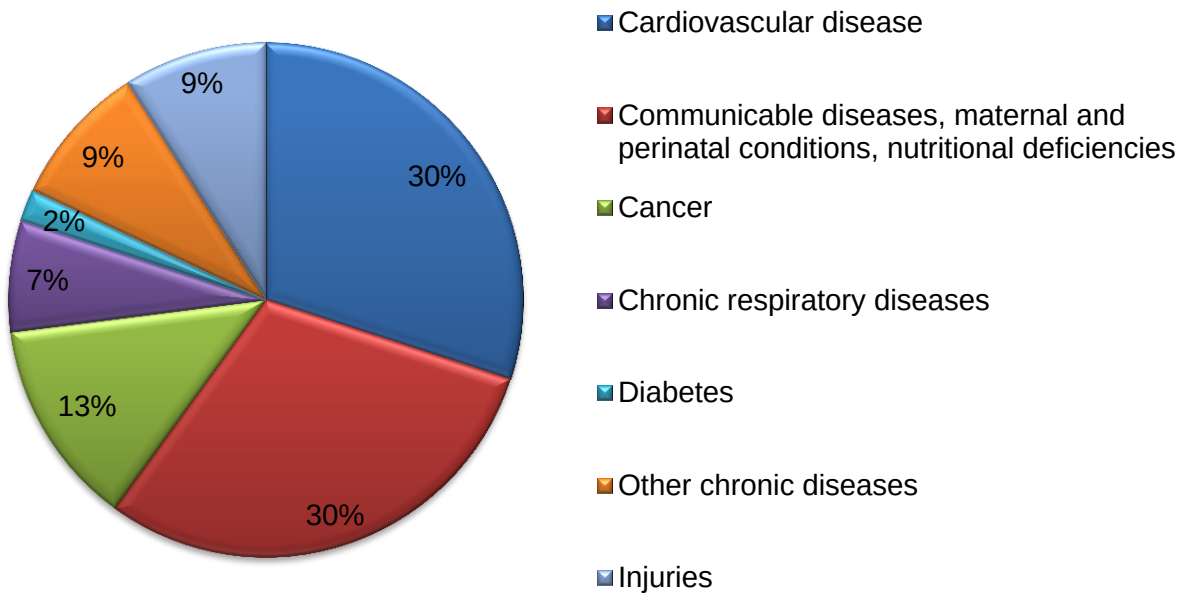

Figure 1: Projected main causes of death globally, all ages, 2005 (Adapted from WHO, 2005)

The spread of NCDs is a global crisis across all countries and income groups and men, women and children are all at risk of these diseases (Beaglehole *et al*., 2011). Although age is a risk factor for NCDs, there is a common misconception that these deaths are restricted to older individuals while, in fact, NCDs are having an effect throughout the age distribution (WHO, 2005). Already, one-quarter of all NCD-related deaths are among people below the age of 60 (WHO, 2011). The '3:4:50' rule has recently been updated to the '4:4:80' rule by the World Health Organisation (WHO) and acts to illustrate the severity of NCDs. This guideline outlines the domino effect of four main behavioural risk factors (physical inactivity, tobacco use, harmful use of alcohol and unhealthy diet) which can lead to four of the most common NCDs (CVD, cancer, chronic respiratory diseases and diabetes), which in turn contribute 80% to global mortality rates (WHO, 2011).

Adding urgency to this NCD debate is the predicted increase in the number of people affected. If the present trend is maintained, it is predicted that by 2020, NCDs will account for 80% of the global burden of disease, causing seven out of every ten deaths in developing nations (Boutayeb, 2006). This is attributable to an increasing world population as well as the exploding prevalence of key risk factors, particularly those associated with urbanisation and globalisation (Cappuccio, 1997; Yach *et al*., 2004; Alberts *et al*., 2005; Bloom *et al*., 2011).

2.2. SOUTH AFRICA'S HEALTH PROFILE

South Africa is a middle-income country which consists of nine provinces spread over an area of 1.22 million km^2 (Myer *et al*., 2004; Wallis *et al*., 2008; Stats SA, 2013), as seen in Figure 2. The 2013 mid-year population estimates for South Africa show a population of 52.98 million divided up as follows: 79.8% black African, 9.0% coloured, 8.7% white and 2.5% Indian or Asian (Stats SA, 2013). Females make up approximately 51% of this population. The country is also very diverse, ranging from highly industrialised cities with an urban advanced-economy lifestyle to remote rural regions with more traditional lifestyles (Vorster, 2002; Sliwa *et al*., 2008).

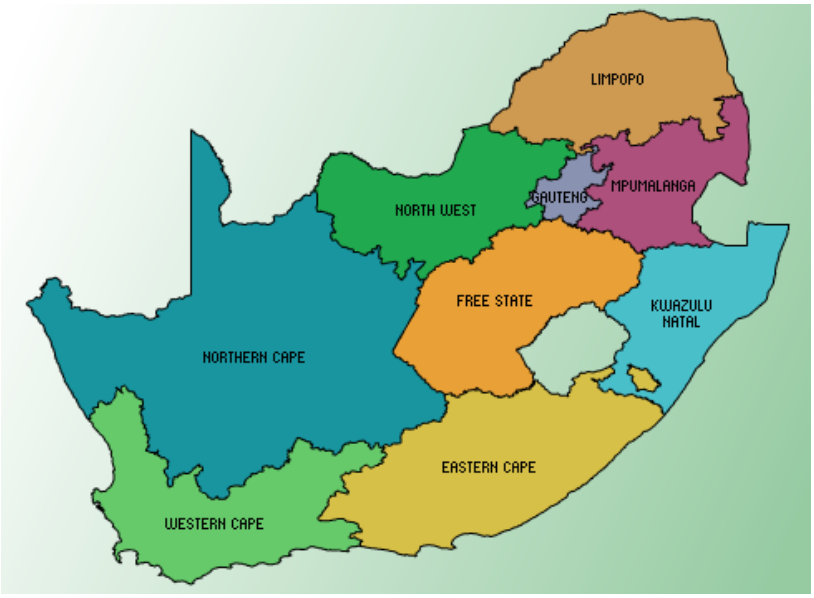

Figure 2: Geographic composition of South Africa (Stats SA, 2006)

The black African population is the poorest of all South African groups and while the majority reside in rural areas (56.7%), the urban proportion (43.3%) is steadily increasing, with many living in informal housing on the borders of cities (Bourne *et al*., 2002; Stats SA, 2013). High levels of poverty (56.8%) and unemployment (24.7%) contribute to an increasing burden of disease, and this disease profile is dramatically modified by the HIV/AIDS epidemic as well as the persistence of external causes of death (Goosen *et al*., 2003; Wallis *et al*., 2008; Stats SA, 2013). The country faces huge inequality based on the 'living condition survey' with a Gini coefficient of 0.7 (Stats SA, 2013). With regard to access to services, the '80:20 rule' applies, 80% of the population rely on the public sector while the wealthy and insured 20% have access to the private sector (Goosen *et al*., 2003; Wallis *et al*., 2008). The public health systems have minimal resources and must meet the needs of the majority of the population who are mostly poor and uneducated (Bradshaw *et al*., 2005; Wallis *et al*., 2008). The ten leading underlying natural causes of death for South Africa in 2010 can be seen in Table 1, with five of the top ten causes falling into the NCD category (Stats SA, 2013). Unpacking this information a bit more exposes sex differences. Deaths due to CVD are more common in females (8.8%) than males (8.7%), and this becomes more pronounced when focussing solely on hypertensive diseases: females (3.5%) compared to males (2.0%) (Stats SA, 2013).

Table 1: The ten leading underlying natural causes of death, all ages, in South Africa 2010 (Stats SA, 2013)

CAUSE	DEATHS (%)		
	ALL	MALE	FEMALE
Tuberculosis	11.6	12.6	10.4
Influenza and pneumonia	7.2	6.9	7.5
Intestinal infectious diseases	5.0	4.6	5.4
Other forms of heart disease	4.7	4.2	5.3
Cerebrovascular diseases	4.5	3.6	5.5
Diabetes mellitus	3.9	3.0	5.0
HIV	3.4	3.2	3.6
Hypertensive diseases	2.7	2.0	3.5
Chronic lower respiratory diseases	2.4	2.8	/
Other viral diseases	2.3	/	2.6

* Non-communicable diseases are highlighted

2.2.1. The Eastern Cape Province

The Eastern Cape is the second largest province in the country with a population of 6.62 million, making up 12.5% of South Africa's total population (Stats SA, 2013). However, with 7.2% of the country's gross domestic product (GDP), the Eastern Cape has less than half the national average which is lower than all other provinces apart from the Northern Province (Lemon, 2004; Stats SA, 2006). This standard of living indicator is supported by self-reports of health status which place the Eastern Cape population as the third worst, with 8.2% reportedly having 'bad or very bad' health. No information exists on sex and race-specific data within this Eastern Cape population. However, since black Africans constitute the majority (87%), it can be assumed that this information is representative of this racial group (Stats SA, 2006). The province is divided into 38 local and 5 district municipalities. The main economic centres are Port Elizabeth and East London, while Grahamstown represents the largest urban settlement within the Makana local municipality (Stats SA, 2006). The disease death profile of the Eastern Cape differs slightly from the national profile as

can be seen in Table 2, with the NCD category highlighted. Of importance are hypertensive diseases which are shown to again be more common in females compared to males, where this cause of death ranks in the top ten. The statistics provided by Stats SA (2013) do not differentiate between ethnic groups, however, which is a limitation of the data.

Table 2: The ten leading underlying natural causes of death, all ages, in the Eastern Cape 2010 (Stats SA, 2013)

CAUSE	DEATHS (%)		
	ALL	MALE	FEMALE
Tuberculosis	12.7	14.0	11.3
Other forms of heart disease	4.6	4.0	5.1
Cerebrovascular diseases	4.3	3.6	5.1
Influenza and pneumonia	4.3	4.2	4.4
Intestinal infectious diseases	3.8	3.3	4.2
Chronic lower respiratory diseases	3.7	4.2	3.3
Diabetes mellitus	3.6	2.5	4.6
Other viral diseases	3.3	2.7	3.8
HIV	3.1	2.7	3.5
Hypertensive diseases	2.6	/	3.4

* Non-communicable diseases are highlighted

2.2.2. The Makana Municipality and Grahamstown

The Makana local municipality is approximately 4221 km^2, and at the 2011 South African census, comprised a population of 80390 people (Stats SA, 2013). The most recent Stats SA (2013) data do not provide any cause of death information on district municipalities within South Africa, making it difficult to draw any conclusions about these areas. However, basic demographic information shows that the population is made up as follows: black African (78.0%), coloured (12.1%), white (8.7%) and Indian or Asian (0.7%). Of the economically active, the unemployment (32.5%) and poverty rates are high with 57.3% of the Makana households having a monthly

income of <R2300 and 12.7% of this group having no income whatsoever (Stats SA, 2013). The most common language spoken is isiXhosa (66.5%), followed by Afrikaans (13.8%) and English (9.7%).

2.2.3. The Quadruple Burden of Disease

South Africa has recently moved from a 'triple burden of disease' to having a 'quadruple burden of disease' which is formed from HIV/AIDS, CDs, NCDs and injury (Bradshaw *et al*., 2005; Kahn, 2006; Delobelle *et al*., 2010; Rispel and Barron, 2010; Westaway, 2010; Mathee, 2011; Bertram *et al*., 2012) (Figure 3). It is common for developing countries to experience a double burden, resulting from the simultaneous occurrence of CDs and NCDs, but, in the case of South Africa, there are the added burdens of HIV/AIDS and injury (Bourne *et al*., 2002; Econex, 2009). This places a high demand on the health services, which struggle to cope with already limited resources and structural developments (Delobelle *et al*., 2010). As a result of these disease burdens, the average life expectancy of the country is low, at 57.7 years for males and 61.4 years for females (Wallis *et al*., 2008; Stats SA, 2013).

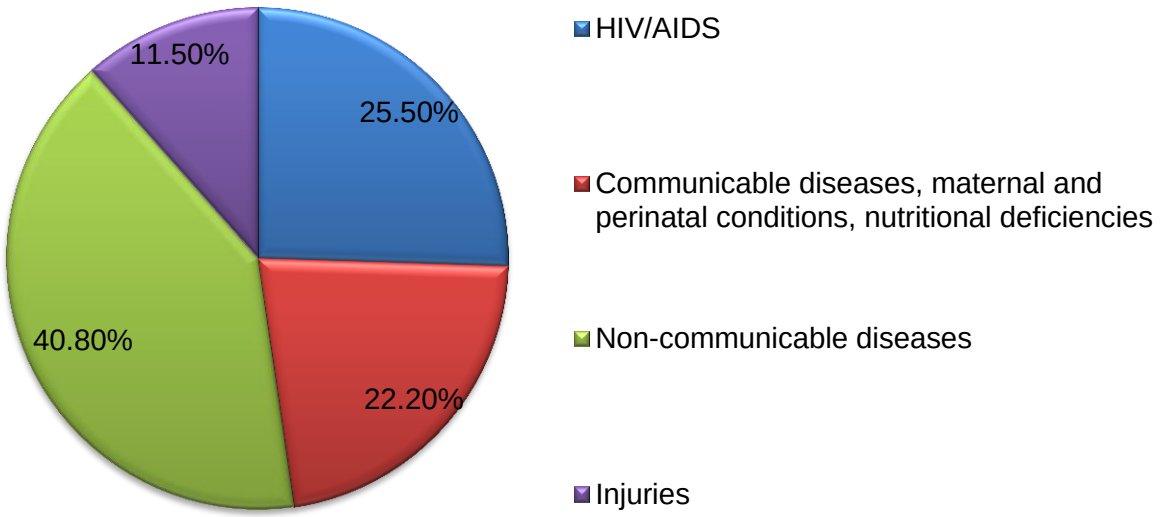

Figure 3: Estimated deaths by broad group for South Africa, 2000 (Adapted from Econex, 2009)

2.3. THE DEMOGRAPHIC TRANSITION

The classic ‘demographic transition’ explains the shift from a pattern of high fertility and mortality to one of low fertility and mortality (which is typical of modern industrialised countries) (Drewnowski and Popkin, 1997; Mulder, 1998; Lee, 2003; Popkin, 2003). This demographic transition period can be divided into two distinct phases. During the first phase, the population growth rate rises as the death rate declines while the birth rate remains high. In the second phase, the growth rate declines (but remains positive) due to a decline in the birth rate. This is a lengthy transition and can typically take more than a century to complete and ends with a much larger population size (Bongaarts, 2009). The populations of the industrialised world have basically completed their transitions, and, even including immigration, the average population growth rate is only a fraction of 1% per year. In contrast, in much of the developing world, population growth continues at a high rate despite large reductions in fertility over the past several decades (Bongaarts and Bulatao, 1999). This growth, which can be found in Africa, can be attributable to three demographic factors: fertility remains above the replacement level, mortality continues to decline and the proportion of the population under the age of 30 years is relatively high (Bongaarts and Bulatao, 1999). Africa, with 0.56 billion people in 2005, is likely to experience by far the most rapid relative expansion, more than double, to reach two billion people by 2050 (Bongaarts, 2009).

2.4. THE EPIDEMIOLOGICAL TRANSITION

As societies develop, infant mortality generally decreases, the causes of death shift and average life expectancy increases; this evolution is referred to as the ‘epidemiological transition’ (Reddy and Yusuf, 1998; Levenson *et al*., 2002; Gaziano, 2005). This transition is primarily due to the industrial and technological revolutions and their associated economic and social changes, resulting in its taking place at different rates in different countries/regions (Reddy and Yusuf, 1998; Levenson *et al*., 2002; Yusuf and Ounpuu, 2003; Gaziano, 2005). The epidemiological transition can be divided into four main ages: pestilence and famine, receding pandemics, degenerative and man-made diseases and delayed degenerative diseases (Omran,

1971; Olshansky and Ault, 1986; Pearson, 1999; Levenson *et al*., 2002; Gaziano *et al*., 2005).

Many of the Western countries experienced this epidemiological transition in the first half of the 20th century, leading to increases in CVD during the first half of the century, with CVD rates peaking between the 1950s and 1970s (Yusuf and Ounpuu, 2003). Thereafter, most of the countries have experienced significant declines in CVD. By contrast, in most developing countries, this epidemiological transition is more evident in the latter half of the 20th century with continuing increases expected in most chronic diseases, including CVD, over the next few decades (Yusuf and Ounpuu, 2003). Most developing regions appear to be following a similar pattern to that of developed regions but the transition has occurred at a more accelerated rate (Gaziano, 2007). Between 1990 and 2020, coronary heart disease alone is expected to increase by 120% for women and 137% for men. However, there remain distinct differences in how severely the burden is affecting various population groups (Gaziano, 2007; Wagner and Brath, 2012).

In sub-Saharan Africa, CD rates still exceed those of chronic diseases, placing this region in the first phase (Levenson *et al*., 2002). However, with CVD rates on the rise, hypertension has emerged as a major public health concern; as a result, stroke is the dominant form of CVD (Gaziano, 2007). Even within developing countries there are likely to be wealthy, educated sub-populations at later phases in the epidemiologic transition. These populations will require different services from the community as a whole (Pearson, 1999). In South Africa there is the unique situation whereby two disease profiles (CDs and NCDs) seem set to co-exist for a long time (Goosen *et al*., 2003).

2.5. THE NUTRITION TRANSTION

It is thought that economic development, and the associated rise in income, pushes populations through what has been termed the ‘nutrition transition’ from

undernutrition to overnutrition (Drewnowski and Popkin, 1997; Pearson, 1999; Stuckler *et al*., 2012). Generally, with the nutrition transition, there is a shift in food preferences from traditional diets characterised by low salt, saturated fat and glycaemic indexes to less healthy and complex 'Western diets' that lead to obesity and NCDs. This dietary shift is often accompanied by reduced physical activity (Drewnowski and Popkin, 1997; Popkin, 1999; Vorster *et al*., 1999; Popkin, 2001; Popkin, 2003; Kimani-Murage *et al*., 2010; Popkin *et al*., 2012; Stupar *et al*., 2012). These lifestyle changes have been supported by rural/urban comparisons of African populations (Popkin, 1994; Bourne *et al*., 2002; Steyn *et al*., 2006). Although the nutrition transition typically begins with urban populations and those in higher socio-economic strata, it is not limited to these populations (Kimani-Murage *et al*., 2010).

The nutrition transition can be divided into three main stages. In stage one, famine begins to recede as income rises and physical activity is predominantly labour-intensive work. In stage two there are adverse changes in diet and activity patterns leading to the emergence of new disease problems (particularly diet-related NCDs) and increased disability. In stage three, behavioural change begins to reverse the negative tendencies and can result in 'successful ageing'. This transition is driven by rapid economic growth, urbanisation, globalisation and technological and social changes (Popkin, 2003; Kimani-Murage *et al*., 2010). The nutrition transition in South Africa is complex because of large differences that exist between different ethnic, cultural and socio-economic groups (Puoane *et al*., 2002; Stupar *et al*., 2012). South African national prevalence data show that shifts in dietary intake are occurring with increasing momentum, particularly in black African people, who constitute more than three-quarters of the population (Puoane *et al*., 2002; Mayosi *et al*., 2009). The black African population has two distinct types of eating patterns, differentiated by rural and urban living. The rural population still follows a very traditional diet, which is high in carbohydrates (CHO) (>65% total energy) and moderately high in fibre. On the other hand, the urban black African population adopts the Western diet which has low CHO (<65% total energy) and fibre intakes while the fat intake is higher (>25% total energy) (Steyn *et al*., 2006). This suggests that there is a nutritional transition among urban black African people from a traditional rural diet to an urban one that is approaching the completely Westernised diet of the white population (Vorster *et al*.,

1999; Puoane *et al*., 2002; Steyn *et al*., 2006). This is exacerbated by the rapid urbanisation which the black African population is experiencing (Vorster *et al*., 1999).

2.6. NON-COMMUNICABLE DISEASES

Four of the most widespread NCDs – CVD, cancers, chronic respiratory diseases and diabetes – are linked by common preventable risk factors including physical inactivity, tobacco use, alcohol consumption and unhealthy diet (WHO, 2005; Mayosi *et al*., 2009; Habib and Saha, 2010; Wagner and Brath, 2012). Despite advances in treatment, CVD represents nearly half of NCD deaths and approximately 30% of all deaths (Bloom *et al*., 2011). While popular belief is that NCDs afflict high-income populations, the evidence tells a very different story. What were once considered 'diseases of affluence' and associated with economic development, have now encroached on developing countries (Boutayeb, 2006; Bloom *et al*., 2011; Wagner and Brath, 2012). Nearly 80% of NCD deaths occur in low- and middle-income countries, with this category of diseases being the most frequent cause of death in most countries, except in Africa. By 2020 it is predicted that NCDs will be causing seven out of every ten deaths in developing countries (Habib and Saha, 2010). Even in African nations, NCDs are rising rapidly and are projected to exceed communicable, maternal, perinatal and nutritional diseases as the most common causes of death by 2030 (WHO, 2011; Wagner and Brath, 2012).

The primary reason behind this rapid increase in NCD prevalence in developing regions is lifestyle changes associated with urbanisation (Delobelle *et al*., 2010). In South Africa specifically the rise is driven by an increase in relevant risk factors in urban and rural areas, and by an ageing population despite the substantial overall reduction in life expectancy as a result of HIV/AIDS (Mayosi *et al*., 2009). The prevention and treatment of NCDs is marginalised in South Africa because of the overwhelming prevalence of CDs such as HIV/AIDS and tuberculosis (TB) (Mayosi *et al*., 2009). In 2004, WHO estimates of the burden of disease in South Africa suggested that NCDs caused 28% of the total burden of disease measured by disability-adjusted life years (DALYs) (Mayosi *et al*., 2009). Based on the DALYs per

100000 population, WHO estimates place South Africa into the highest burden quintile, with the burden being 2-3 times higher than in developed countries (Mayosi *et al*., 2009). These data illustrate the rapidly increasing risk of NCD in South Africa, which is driving a rise in the demand for chronic disease care (Mayosi *et al*., 2009). It is therefore important that the modifiable risk factors contributing to the development of NCDs in South Africa receive increased attention through the promotion of healthy lifestyles (Bourne *et al*., 2002; SANHANES-1, 2013).

2.7. CARDIOVASCULAR DISEASE

Globally, CVD represents the leading cause of death (Assmann *et al*., 1999; Pearson, 1999; Bonow *et al*., 2002; Levenson *et al*., 2002; Beaglehole and Yach, 2003; Gaziano *et al*., 2005; Bloom *et al*., 2011; Celermajer *et al*., 2012; Mendis, 2013). In 2008, CVD caused an estimated 17.3 million deaths per year, a number that is expected to grow to more than 23.6 million by 2030 (Laslett *et al*., 2012; Smith *et al*., 2012). Although CVD is a multifactorial disease, 80% of this global 'epidemic' can be explained by three main behavioural risk factors: physical inactivity, tobacco use and unhealthy diet (Gaziano *et al*., 2010). Yusuf and Ounpuu (2003) add two population risk factors to this epidemic: the ageing factor and the unique susceptibility of certain sub-populations.

The populations affected most by CVD are those in low- and middle-income countries where 80% of these deaths occur, and where the human and financial resources to address them are most limited (Gaziano, 2005; Alwan *et al*., 2010; Laslett *et al*., 2012; Wagner and Brath, 2012; Mendis, 2013). In developing countries, the increase in CVD burden is largely the result of an increase in the prevalence of risk factors and low budgets for health (including screening, prevention and treatment) (Beaglehole and Yach, 2003; Alberts *et al*., 2005; Celermajer *et al*., 2012; Gilbert-Ouimet *et al*., 2012; Wagner and Brath, 2012). In South Africa specifically, CVD is the second leading cause of death after HIV/AIDS (Figure 4) (Matsha *et al*., 2012).

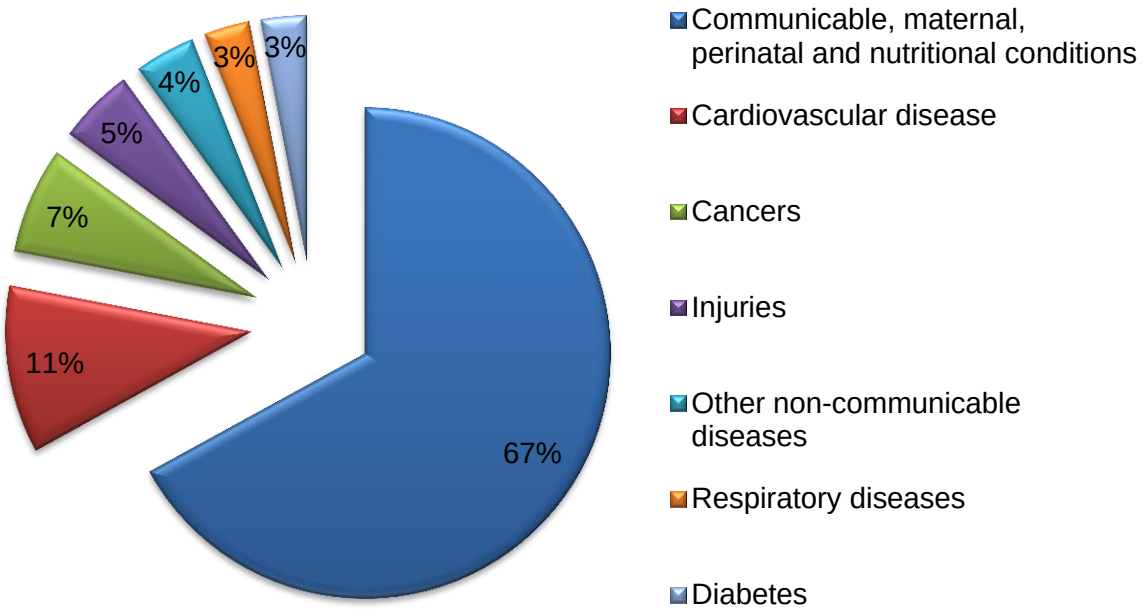

Figure 4: Proportional mortality (% of total deaths, all ages) in South Africa in 2008 (Alwan *et al*., 2011)

Given the nearly $400 billion in direct and indirect annual costs related to CVD in the United Sates in 2006, the economic effects of this problem are equally important for the sustainability of many developing countries (Gaziano, 2007). Age-adjusted death rates from stroke and ischemic heart disease are increasing in some developing regions, and a relatively younger population is affected by CVD leading to an increased number of deaths in the working-age population (Gaziano, 2007; Wagner and Brath, 2012). This epidemic therefore has the potential to place a large social and economic burden on developing countries. Since resources for managing CVD are limited, it is important that interventions be guided by cost-effective results, especially for low- and middle-income countries (Gaziano, 2007). A systematic review found that, generally, there is a dose-dependent reduction in CVD risk with higher walking duration, distance, energy expenditure and pace (Boone-Heinonen *et al*., 2009). This general association is supported by other studies (Zheng *et al*., 2009; Sattelmair *et al*., 2011). The causal risk factors of CVD include several different modifiable and non-modifiable factors (Table 3).

Table 3: Risk factors for cardiovascular disease (Adaped from Habib and Saha, 2010; Van Zyl *et al*., 2012)

MODIFIABLE	NON-MODIFABLE
High blood pressure	Age
Overweight/Obesity	Sex
Physical inactivity	Race
Unhealthy diet	Family history
Tobacco use	
Alcohol abuse	
Type II diabetes	
High blood cholesterol	
Stress	

Within a South African context, Table 4 illustrates the modifiable risk factors for CVD and those sub-population groups representing the highest risk for each variable along with their associated prevalence (%), based on the SANHANES-1 (2013) data. The highlighted entries (poor cardiovascular fitness, obesity and raised systolic blood pressure (SBP)) represent the three most prevalent risk factors across sex, race and age strata.

2.8. HIGH BLOOD PRESSURE

High BP is the primary risk factor for CVD (Forrester, 2004; Gaziano *et al*., 2005; Cheung *et al*., 2005; Kearney *et al*., 2005; Chockalingam *et al*., 2006; Kaplan and Opie, 2006; Kemp *et al*., 2011). The relationship between BP and the risk of a cardiovascular event is continuous, consistent and independent of other risk factors (Joint National Committee, 2004; Bentley and Dezorzi, 2012). Data from observational studies involving more than one million individuals, aged 40-89 years, have indicated that death from both ischemic heart disease and stroke increase progressively and linerarly from levels as low as 115 mmHg SBP and 75 mmHg diastolic blood pressure (DBP) upward. For every 20 mmHg systolic or 10 mmHg diasolic increase in BP, there is a doubling of mortality from both ischemic heart disease and stroke (Joint National Committee, 2004; Kokkinos and Myers, 2010).

Table 4: Cardiovascular disease risk factors and their most 'at risk' population groups within South African adults (Adapted from SANHANES-1, 2013)

RISK FACTOR	DEFINITION	HIGHEST RISK WITH ASSOCIATED PREVALENCE (%)			
		SEX	RACE	AGE (years)	AVERAGE
Pre-hypertension (SBP)	120-139 mmHg	Male (43.9)	All white (41.6)	All 35-44 (45.2)	43.6
Pre-hypertension (DBP)	80-89 mmHg	Female (20.5)	All Coloured (26.8)	All 45-54 (31.0)	26.1
Hypertensive	≥140/90 mmHg	Both (10.2)	All white (12.2)	All 55-64 (21.5)	14.6
Overweight	BMI 25-29.9 $kg.m^{-2}$	Female (24.8)	Asian/Indian male (32.3)	Male ≥65 (40.4)	32.5
Obese	BMI >30 $kg.m^{-2}$	Female (39.2)	African female (39.9)	Female 45-54 (56.3)	45.1
Poor cardiovascular fitness	Unfit	Female (45.4)	Coloured female (62.0)	Female 18-24 (50.2)	52.5
Poor diet^	N-index >27.6-45	Male (24.9)	African male (27.4)	Male ≥65 (35.6)	29.3
Tobacco use	Have ever smoked	Male (32.8)	Coloured male (50.8)	Male 45-54 (44.3)	42.6
Alcohol abuse^	CAGE questionnaire ≥2	Male (21.4)	Coloured male (31.2)	Male 25-34 (26.7)	26.4
High HbA_{1c}	Diabetic >6.5%	Female (11.0)	All Asian/Indian (30.7)	All 55-64 (24.4)	22.0
High serum cholesterol	Abnormal >5 $mmol.L^{-1}$	Female (28.1)	Asian/Indian female (45.3)	Female ≥55 (50.3)	41.2
Psychological distress	Kessler-10	Female (31.4)	African female (34.2)	Female ≥65 (45.3)	37.0

* SBP = systolic blood pressure; DBP = diastolic blood pressure; BMI = body mass index; N-index = nutrition index; HbA_{1c} = glycated haemoglobin

* Highlighted rows represent the three most prevalent risk factors across sex, race and age strata

^ Data obtained from the 2003 SADHS

In addition, longitudinal data obtained from the Framingham Heart Study have indicated that BP values between 130-139/85-89 mmHg are associated with a more than twofold increase in relative risk from CVD as compared with BP levels below 120/80 mmHg (Joint National Committee, 2004). Due to new risk data of hypertension and the increase in cardiovascular complications associated with levels of BP previously considered to be normal, a new classification has been introduced (Table 5). This aims to identify those individuals in which early intervention by adoption of healthy lifestyles could reduce BP, decrease the rates of progression of BP to hypertensive levels with age or prevent hypertension entirely (Joint National Committee, 2004).

Table 5: Revised blood pressure classification for adults (Adapted from Joint National Committee, 2004)

SBP (mmHg)	DBP (mmHg)	BP CLASSIFICATION
<120	<80	Normal
120-139	80-89	Pre-hypertension
140-159	90-99	Stage 1 hypertension
≥160	≥100	Stage 2 hypertension

* SBP = systolic blood pressure; DBP = diastolic blood pressure; BP = blood pressure

Hypertension is a public health concern due to its magnitude, risks, difficulty in management, high medical and social costs and severe cardiovascular and renal complications (Gasperin *et al*., 2009). Hypertension affects approximately one billion people worldwide, a number expected to reach 1.56 billion by 2025 (Kearney *et al*., 2005; Gasperin *et al*., 2009; Kumar, 2013). Kearney *et al*. (2005) indicated that the estimated number of hypertensive individuals in developing countries outweighed those in developed countries by almost twofold (639 million in developing countries compared to 333 million in developed countries). The Centres for Disease Control and Prevention indicated that one in three adults in America has hypertension and the same finding is seen in South Africa, where 55% of adults ≥30 years have hypertension (Kemp *et al*., 2011). The burden of hypertension has increased over the past decade due to population growth, ageing and increase in behavioural risk

factors including lack of physical activity, unhealthy diets, harmful use of alcohol, excess weight and exposure to persistent stress (Mendis, 2013; SANHANES-1, 2013). It can therefore be concluded that this modifiable, preventable and controllable risk factor has reached epidemic proportions (Chockalingam *et al*., 2006).

In sub-Saharan Africa, hypertension remains the most threatening risk factor, with national prevalence ranging from 15 to 30% in adults (Deaton *et al*., 2011). By using the international criteria for hypertension (BP ≥140/90 mmHg or persons aged >18 years receiving antihypertensive medication), the 1998 South African Demographic and Health Survey (SADHS) estimated that there were six million people with hypertension in the country (Kearney *et al*., 2005; De Bacquer and De Backer, 2006; Addo *et al*., 2007; Elliott, 2007; Seedat and Rayner, 2012). Over a decade later, and despite advances in treatment and prevention, the prevalence of hypertension still remains high within the South African adult population (Table 6). The Eastern Cape sample made up a good proportion of the national sample in all three of these surveys and so the data are applicable; the 1998 SADHS had 1919 participants (13.8%), the 2003 SADHS had 929 participants (11.4%) and the 2013 SANHANES-1 contributed 991 participants (14.1%) from the Eastern Cape area. However, this sample size is very small compared to the areas overall population of 6.62 million.

Table 6: Prevalence (%) of hypertension in South African adults

STUDY	YEAR	PREVALENCE (%) OF HYPERTENSION (≥140/90 mmHg)	
		MALE	FEMALE
SADHS	1998	22.9	24.6
*SADHS	2003	12.5	17.9
SANHANES-1	2013	10.2	10.2

* Inaccurate blood pressure data due to the measurements not being taken correctly by the field-workers

The Prospective Urban Rural Epidemiology study in the North West Province, South Africa, studied almost two thousand black African adults (≥30 years) for five years. They found that during this time 24% of the participants with optimal BP developed hypertension which is largely explained by modifiable risk factors (Schutte *et al*., 2012). This confirms the urgent need to develop strategies to prevent, detect, treat and control hypertension effectively in the African region. Public health strategies should focus on lifestyle to prevent a massive burden on the national health system (Kaplan and Opie, 2006; Addo *et al*., 2007; Schutte *et al*., 2012). However, it is challenging to make hypertension a national priority in developing countries when other diseases such as HIV/AIDS must take priority for restricted health budgets (Vorster, 2002; Kaplan and Opie, 2006).

NCDs such as hypertension should be considered of great economic importance because of their high frequency in young populations, frequent under-diagnosis, under-treatment and severity of complications. Hence, the promotion of preventive measures from an early age, such as reduced alcohol intake and healthy diets, may be of great importance in South Africa (Schutte *et al*., 2012). In low- and middle-income countries, many people do not seek treatment for early stage hypertension because it is unaffordable. Households then spend a substantial share of their income on hospitalisation and care of hypertension complications and may be driven into poverty (Mendis, 2013). Hypertension is a costly contributor to CVD in South Africa, as evident in 1991 when it accounted for between R4 and 5 billion in direct and indirect expenditure and was previously shown to make up 7.5% of the direct total healthcare cost (Seedat and Rayner, 2012). Hence the social and economic demands of effectively treating hypertension are of considerable importance, especially within the South African context (Gaziano *et al*., 2005). Global approaches need to focus on healthy lifestyle changes and increased awareness through education which can be widely initiated as preventative measures, whereas approaches for individuals should be associated with antihypertensive drug therapy (Kaplan and Opie, 2006; Addo *et al*., 2007).

2.8.1. Non-Modifiable Risk Factors for High Blood Pressure

2.8.1.1. AGE

Increasing age is a major risk factor for developing hypertension as well as a strong confounder of its independent influence on cardiovascular and renal events (Elliott, 2007). It has been found that the prevalence of hypertension increases steadily from the youngest to the highest age groups. This is similar for males and females and in rural and urban studies (Addo *et al*., 2007). The Framingham Heart Study reported that approximately 90% of men and women who are not hypertensive by the age of 55-65 years will develop hypertension between the ages of 80-85 years (Joint National Committee, 2004; Kumar, 2013). The rise in SBP continues throughout life, in contrast to DBP, which rises until approximately age 50, tends to level off over the next decade, and may remain the same or fall later in life (Joint National Committee, 2004; Kaplan and Opie, 2006; Kumar, 2013).

South Africa has one of the most rapidly aging populations in Africa, with 7.3% of the population aged ≥60 years in 2001 (Mayosi *et al*., 2009; Westaway, 2010). Despite the impact of HIV/AIDS, this proportion is expected to increase to 9.5% in 2015, with a large majority of the country's population being black African race and aged ≥50 years (Westaway, 2010). Connected with this aging profile is the increase in prevalence of hypertension, overweight and obesity (Westaway, 2010). Of added concern is the early age of CVD deaths in the developing countries compared with the developed countries. In 1990, the proportion of CVD deaths occurring below the age of 70 years was 26.5% in developed countries compared with 46.7% in developing countries (Reddy and Yusuf, 1998). It is predicted that even without changes in the risk-factor profile or the mortality rates from CVD, the demographic changes will result in a doubling in the number of cardiovascular deaths in South Africa by 2040 (Mayosi *et al*., 2009).

2.8.1.2. SEX

Traditionally, CVD was considered a group of diseases predominantly affecting men, and for a long time women were not included in cardiovascular research programs (Roeters *et al*., 2002; Mounier-Vehier *et al*., 2012). The World Health Statistics (2012) have estimated the prevalence of hypertension globally to be 29.2% in males and 24.8% in females (Kumar, 2013). A sexual dimorphism clearly exists with BP, across all ethnic groups, such that women have lower SBP levels than men during early adulthood (August and Oparil, 1999; Dubey *et al*., 2002; Joint National Committee, 2004; Collier *et al*., 2011). DBP tends to be just slightly lower in women than men regardless of age. This gender gap begins to narrow by the fourth decade of life, even before the onset of menopause, and the prevalence of hypertension in women is equal to, or exceeds, that in men during the sixth decade of life (Burt *et al*., 1995; August and Oparil, 1999; Joint National Committee, 2004; Ljungman *et al*., 2009). Although the mechanisms responsible for these sex differences in BP control are not clear, there is significant evidence that sex hormones, such as testosterone and estradiol, play an important role (Harshfield *et al*., 1994; August and Oparil, 1999; Reckelhoff, 2001). On the other hand, the mechanisms responsible for hypertension in these populations are complicated by co-morbid conditions of obesity and type II diabetes, both of which lead to increases in BP (Burt *et al*., 1995).

A different pattern can be observed within the South African population, however. When looking at the age-standardised prevalence of hypertension from the 1998 SADHS, one sees a slight difference between the sexes, with females having a greater prevalence (24.6%) than males (22.9%) (Addo *et al*., 2007). Fast-forward 15 years and the SANHANES-1 (2013) data show no difference in hypertension prevalence (10.2% for both sexes). Homing in on the Eastern Cape data, hypertension prevalence was slightly higher than the national average (10.4% compared to 10.2% respectively), with no differentiation made between the sexes. All of these data are of added concern for females since they are more likely to have been detected, be on treatment and have their hypertension controlled than males (Joint National Committee, 2004; Addo *et al*., 2007; Steyn *et al*., 2008), yet their hypertension risk is still very high.

From the Heart of Soweto study of the urban black African participants who underwent BP measurements (1691 participants), 19% showed evidence of both systolic and diastolic forms of hypertension with no difference between the sexes (Tibazarwa *et al*., 2009). Overall, 33% of these participants had either an elevated systolic or diastolic BP: 33% and 34% of males and females, respectively (Tibazarwa *et al*., 2009). Another study on the rural black African population in Limpopo found that, for their population compared to the SADHS (1998), in each age category the women (but not men) had higher systolic and diastolic BP levels (Alberts *et al*., 2005). These higher systolic and diastolic BP levels in comparison are suggestive of poor BP control. Hypertension is particularly important in women because it is a modifiable risk factor that is extremely prevalent in older women (August and Oparil, 1999). The highest prevalence rates of hypertension are observed in elderly black women, with hypertension occurring in 75% of women ≥75 years (Joint National Committee, 2004). CVD presents differently in men and women and there is evidence that treatment efficacy is different between sexes (Mounier-Vehier *et al*., 2012), thus further research is warranted in the female population.

2.8.1.3. RACE

There is substantial evidence, particularly from the United Kingdom and United States, that the black race has a higher prevalence and more severe hypertension than other ethnic groups (Lopes *et al*., 2003; Mainous *et al*., 2004; Hertz *et al*., 2005; Brown, 2006; Forouhi and Sattar, 2006; Howard *et al*., 2006; Sharp *et al*., 2008; Thorpe *et al*., 2008; Tullio, 2008; Carson *et al*., 2011; Redmond *et al*., 2011; Boykin *et al*., 2012). This is thought to be linked with the higher incidence of low birthweight (Lopes, 2002; Haas, 2006). Low birthweight, defined as <2500 g, is most common in low- and middle-income regions, with a 15% prevalence in sub-Saharan Africa and a reported 8% prevalence in South Africa (SADHS, 2003; Sabet *et al*., 2009). The Eastern Cape had a lower prevalence (5.3%); however, this province had the highest amount of missing data as 36.7% of birth weights were not provided. But even though this racial difference in hypertension prevalence is known to exist, very little comparable information exists for black Africans (Tibazarwa *et al*., 2009). These previous studies further conclude that blacks tend to develop hypertension at an

earlier age, and the target organ damage differs from that in 'whites' (Caucasians) (Brown, 2006). Tibazarwa *et al*. (2009) agrees with this, stating that "hypertension in black Africans occurs at a younger age, being more severe and leading to earlier damage to vital organs".

Vorster (2002) compared different population groups within South Africa and discovered that although all groups had relatively high hypertension prevalence (≥160/95 mmHg), black African women (13%), white men (15.2%) and coloured women (17.1%) had the highest. Further, hypertension, at 59% prevalence, has been reported as the most common CVD risk factor among black South Africans (Dennison *et al*., 2007). The Heart of Soweto study found similar results, with a reported 56% hypertension prevalence in black Africans (Sliwa *et al*., 2008). A follow-up prospective study stated that "in the absence of effective primary and secondary prevention strategies, these unique data highlight the potentially devastating impact of advanced forms of hypertensive heart disease in urban black African communities with more women than men affected" (Stewart *et al*., 2011).

2.8.1.4. FAMILY HISTORY

Family history is a simple yet powerful clinical tool. Family members may resemble each other in risk of disease due to shared biological, cultural and behavioural factors (Valdez *et al*., 2010). Diseases of major public health importance such as heart disease, diabetes and stroke, have consistently shown that the risk of developing one of these conditions is significantly increased by having one or more close relatives with the disease (Meigs *et al*., 2000; Lloyd-Jones *et al*., 2004; Flossmann and Rothwell 2005; Valdez *et al*., 2010). Routine collection of family history in adults who may be at risk of a common chronic disease could therefore motivate patients to adopt healthier lifestyles and undergo recommended screening (Valdez *et al*., 2010).

A family history of hypertension is considered one of the stronger risk factors for developing high BP (Tozawa *et al*., 2000). Specifically, the number of first-degree family members with hypertension was quantitatively correlated with the risk of hypertension: an association which occurs independently of other risk factors (Tozawa *et al*., 2000). Considering the disease process, it is likely that a positive family history of hypertension may lead to an increased risk through genetic heritability of factors associated with hypertension. These factors include elevated serum cholesterol and diabetes; the inheritance of susceptibility to the effects of such factors; family sharing of environmental and lifestyle factors such as higher sodium and fat diets, lower physical activity, smoking habit, and drinking habits; and/or the interaction between genetic and environmental factors (Tozawa *et al*., 2000). Tozawa *et al*. (2000) found that an adult with a positive family history has a 2.7-6.0 times higher likelihood of being hypertensive than an adult with no such family history. Katz *et al*. (2012) agree that parental history of hypertension is associated with higher levels of systolic and diastolic BP among offspring, as well as a greater risk of hypertension. The SANHANES-1 (2013) recorded the prevalence of self-reported family history of four listed NCDs, with the most reported condition being high BP (30.9%), followed by high blood sugar (20.7%), stroke (8.9%) and then heart diseases (heart attack, angina, chest pain; 7.6%) (SANHANES-1, 2013).

2.8.2. Modifiable Risk Factors for High Blood Pressure

Of the modifiable risk factors for hypertension previously mentioned, most have been associated primarily with urbanisation, which is being experienced predominantly by the black African population (Vorster, 2002). These adverse behavioural risk factors include excessive dietary sodium, weight gain, physical inactivity, excessive alcohol consumption, tobacco use and excess psychological stress (van Rooyen *et al*., 2000; Kaplan and Opie, 2006; Schutte *et al*., 2012). This is supported by a systematic review by Addo *et al*. (2007) who found urban populations to consistently have a higher prevalence of hypertension compared with their rural counterparts in almost all studies that covered both types of areas, with black Africans experiencing the most rapid migration (Vorster, 2002). A study of prevalence and control of hypertension in South Africa suggested that the observed differences in the

prevalence of hypertension are a result of socio-demographic parameter differences (Steyn *et al*., 2008). Rural black Africans had a significantly lower risk of hypertension (odds ratio (OR) 1.00) than urban black African (OR 1.42), coloured (OR 1.54) and white (OR 1.84) participants, suggesting a clear urban-rural difference, but no inherent differences among the population groups regarding the risk of developing hypertension (Steyn *et al*., 2008). Analyses further showed that this urban-rural difference among black Africans is more pronounced among women, with an OR of 1.65 compared with 1.16 for men (Steyn *et al*., 2008). Recently, however, results from a cross-sectional study on black South Africans suggests a drift of CVD risk factors from groups with higher socio-economic status to groups with a lower socio-economic status from 1996 to 2005. This indicates that interventions to prevent CVD should also be targeted at Africans living in rural areas, those with low education levels and the unemployed (Pisa *et al*., 2012).

2.8.2.1. EXCESSIVE DIETARY SODIUM

It has been shown that a high salt intake causes a rise in BP, thereby increasing the risk of hypertension and CVD (He and MacGregor, 2010; Batuman, 2012; Bertram *et al*., 2012). Furthermore, excessive dietary sodium intake has been found to contribute to resistance to antihypertensive treatment (Pimenta *et al*., 2009). The South African Hypertension Guidelines recommend a maximum salt intake of 6 $g.day^{-1}$ which falls in the upper boundary of the 4-6 $g.day^{-1}$ recommended by the WHO (Bertram *et al*., 2012). Of concern is the high average daily salt intake of the different South African population groups: black African (7.8 g), mixed race (8.5 g) and white (9.5 g) (Bertram *et al*., 2012). These high values arise primarily through non-discretionary intake, the highest proportion from bread contributing 25-40% of sodium intake, with smaller contributions from margarine, soup mixes and gravies.

Restricting the dietary salt intake is commonly associated with a reduction in SBP, DBP or both (Joint National Committee, 2004; Meland and Aamland, 2009; Bibbins-Domingo *et al*., 2010; Klaus *et al*., 2010; Bertram *et al*., 2012; He *et al*., 2012). Specifically in low- and middle-income countries, salt reduction is one of the most

cost-effective strategies to combat the high BP epidemic, thereby improving CVD risk and population health (Mohan and Campbell, 2009; He *et al*., 2012). Batuman (2012) states that even a modest reduction in salt intake can result in major improvements in public health and lead to cost reduction in healthcare expenditures. This is supported by He *et al*. (2013), who performed a Cochrane systematic review and meta-analysis to determine the effects of longer term modest salt reduction on BP, hormones and lipids. BP results showed that a modest reduction in salt intake (average of 4.4 $g.day^{-1}$) for four or more weeks caused significant and, from a population viewpoint, important falls in SBP (4.18 mmHg) and DBP (2.06 mmHg). This decrease occurred in both hypertensive and normotensive individuals, irrespective of sex and ethnic group (He *et al*., 2013). Further support comes from Bertram *et al*. (2012) who investigated the potential effect of a salt reduction policy on South African health using average salt intake statistics from 2005. By using regression equations it was determined that for each 100 mmol reduction in sodium intake, a SBP reduction of 5-10 mmHg is expected, with variations according to age. With an average salt reduction of 0.85 $g.person^{-1}.day^{-1}$ they concluded that there would be 7400 fewer CVD deaths (2000 from hypertensive heart disease alone) and 4300 less non-fatal strokes per year compared with 2008, thus relieving pressure on our overburdened health system. Data also indicated that the direct costs of treating a single stroke case amount to R76 thousand and so such a policy could potentially result in a total annual saving of R300 million (Bertram *et al*., 2012).

2.8.2.2. WEIGHT GAIN

Of all the modifiable risk factors, weight gain is increasing most rapidly in both developed and developing societies, other than for societies with persistent inadequacy of food (Kaplan and Opie, 2006). Obesity can be described as an imbalance between energy intake and expenditure, so that excess energy is stored in fat cells which enlarge or increase in number (Mikhail *et al*., 1999; Goedecke *et al*., 2005). Obesity is affected by a host of contributing factors and can be defined as a body mass index (BMI) of >30 $kg.m^{-2}$, according to the WHO criteria (Table 7) (Goedecke *et al*., 2005; Apovian and Gokce, 2012).

There is a significant association and positive correlation between increasing BMI and the presence of systolic and/or diastolic hypertension (Puoane *et al*., 2002; Tibazarwa *et al*., 2009; Benmohammed *et al*., 2011; Apovian and Gokce, 2012; van Zyl *et al*., 2012; Barnes, 2013).

Table 7: WHO classification of obesity (Adapted from James *et al*., 2001)

BMI ($kg.m^{-2}$)	CLASSIFICATION	RISK OF CO-MORBIDITIES
<18.5	Underweight	Low
18.5-24.9	Normal range	Average
25.0-29.9	Overweight	Increased
30.0-34.9	Obese class I	Moderate
35.0-39.9	Obese class II	Severe
≥40.0	Obese class III	Very Severe

Obesity is an independent risk factor for the development of hypertension, and according to a population-based survey on American adults, is associated with a relative adjusted risk of 3.5 for hypertension (Mertens and Van Gaal, 2000; Mokdad *et al*., 2003). Furthermore, the risk estimates from the Framingham Heart Study suggest that approximately 75% and 65% of the cases of hypertension in men and women, respectively, are directly attributable to overweight and obesity (Davy and Hall, 2004). The WHO estimates that by 2015, the number of overweight people worldwide will increase to 2.3 billion, whilst more than 700 million will be obese (van Zyl *et al*., 2012). Consequently, this growing prevalence of obesity is increasingly recognised as one of the most important risk factors for the development of hypertension (Narkiewicz, 2006).

Within South Africa, BMI rates are rapidly increasing, especially in black African women who have the highest prevalence of obesity (39.9%) across race and sex strata (Puoane *et al*., 2002; Goedecke *et al*., 2005; Vorster *et al*., 2005; Kaplan and Opie, 2006; Stupar *et al*., 2012; SANHANES-1, 2013). Worryingly, this population in the Eastern Cape have an even greater prevalence of obesity (41.8%) than the

reported national average (SANHANES-1, 2013). Poor eating habits combined with menstruation, pregnancy, HIV/AIDS and decreased physical activity, as well as the cultural desirability value of obesity all accentuate the risk of poor nutrition in this population group (Kaplan and Opie, 2006; Stupar *et al*., 2012). The Heart of Soweto Study found the most prevalent risk factor for heart disease to be obesity, with 70% of the participants being overweight and 43% being obese (Tibazarwa *et al*., 2009). Furthermore, the prevalence of obesity was significantly higher in women than men (55% versus 23%) (Tibazarwa *et al*., 2009). This study was limited to participants in the Gauteng Province and so more research is required in other South African regions including the Eastern Cape.

Abdominal obesity has more adverse health consequences than peripheral obesity, as this is associated with hypertension, diabetes, CVD and stroke (Mertens and Van Gaal, 2000; Alberts *et al*., 2005). Abdominal obesity is principally measured as waist circumference (WC), waist-to-hip ratio (WHR) and more recently, waist-to-height ratio (WHtR) (Shields *et al*., 2012). Within South Africa, abdominal obesity (defined by a WHR of ≥1.0 for men and ≥0.85 for women) was found in 42.2% of women and was most prevalent in urban black African women and women of mixed ancestry (Puoane *et al*., 2002). A study done on the rural black African population of Limpopo found that 50% of the women participants were either overweight or obese or had a WC above the usual cut-off point (Alberts *et al*., 2005). Although WC limits and classification vary according to various population groups, the guidelines put forward by the WHO are based on numerous global studies and therefore provide useful criteria for the identification of individuals at risk (Table 8).

Table 8: WHO waist circumference guidelines for females (Adapted from James *et al*., 2001)

WAIST CIRCUMFERENCE (mm)	RISK CLASSIFICATION
≥800	Above action level 1
≥880	Above action level 2

Weight loss has been recommended as an effective non-pharmacological therapy for hypertensive individuals (Mertens and Van Gaal, 2000; Kuller, 2009; Tyson *et al*., 2013). A positive linear relationship between weight loss and BP has been shown to exist which is most probably attributable to loss of both body water and salt and perhaps reduced sympathetic activity and insulin resistance (Kuller, 2009). Neter *et al*. (2003) conducted a meta-analysis of 25 randomised controlled trials (RCTs) to estimate the effect of weight reduction on BP. The authors found that weight loss (by means of energy restriction, increased physical activity or both) reduced SBP by 4.44 mmHg and DBP by 3.57 mmHg. When expressed per kilogram of weight loss, SBP reduced by 1.05 mmHg and DBP by 0.92 mmHg. Further, it was found that significantly larger BP reductions were observed in populations with an average weight loss of >5 kg compared with those with less weight loss. Other previous meta-analyses of clinical trials have confirmed this association, however, most of the studies have had relatively small sample sizes and have been short in duration (<1 year) (Kuller, 2009).

2.8.2.3. PHYSICAL INACTIVITY

Physical inactivity, or sedentarism, is the fourth leading cause of death worldwide (Kohl *et al*., 2012). It is considered a major risk factor for a number of adverse health outcomes, including hypertension, with fewer women being active than men (Higashi *et al*., 1999; Tudor-Locke and Bassett, 2004; Peterson *et al*., 2005; Armstrong and Bull, 2006; Lee *et al*., 2012; SANHANES-1, 2013). An analysis of burden of disease and life expectancy from physical inactivity estimated that, in 2008, this risk factor caused 9% (±3.9) of premature mortality, or more than 5.3 million of the 57 million deaths, worldwide (Lee *et al*., 2012). Yet, despite this, data suggest that 31%, nearly a third, of the worldwide adult population are not meeting the minimum recommendations for physical activity (Hallal *et al*., 2012; Kohl *et al*., 2012). These guidelines for healthy adults, put forward by the American College of Sports Medicine (ACSM), recommend the accumulation of at least 30-minutes of moderate-intensity cardiorespiratory exercise on five or more days of the week (Haskell *et al*., 2007; Murphy *et al*., 2007; Garber *et al*., 2011; Whitt-Glover *et al*., 2012). Furthermore, physical inactivity is insufficiently recognised in both developed and

developing countries. Figure 5 illustrates the prevalence of physical inactivity on a global scale, with particular attention to the very high levels (>50%) in South African females.

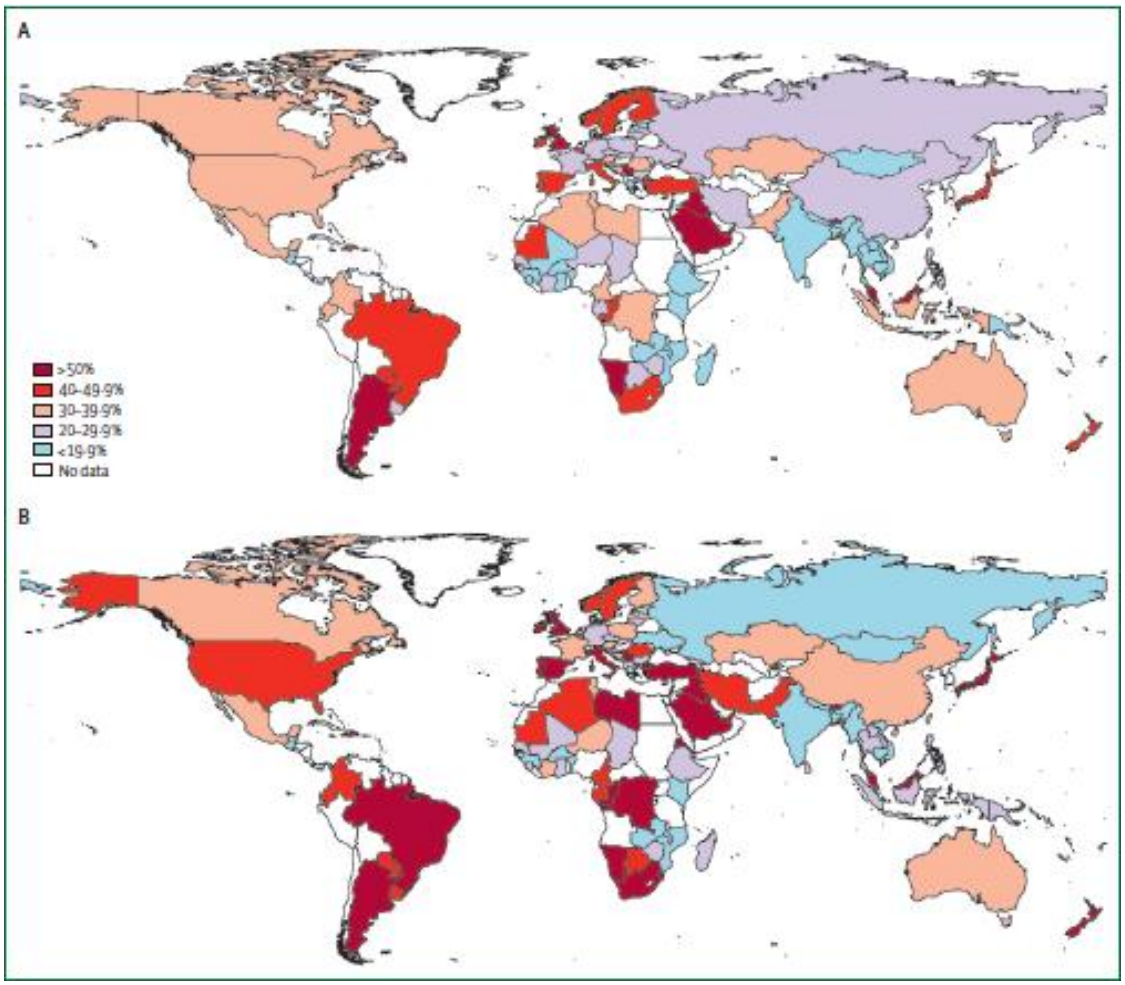

Figure 5: Physical inactivity in adults (≥15 years) worldwide in (A) men and (B) women (Hallal et al., 2012)

Blair (2009) goes so far as to say that physical inactivity is the biggest public health problem of the 21st century. He supports this notion with evidence from a follow-up of a very large population of men and women in the Aerobics Centre Longitudinal Study (ACLS). The ACLS provides data on the population attributable fraction (PAF), adjusted for age, which is the estimated number of deaths in a population that would have been avoided if a specific risk factor were absent. Low cardiorespiratory fitness

was shown to account for approximately 16% of all deaths in both men and women in this population, and this is substantially more, with the exception of hypertension in men, than the other risk factors (Blair, 2009). Research data on the magnitude and impact of physical inactivity and cardiovascular fitness in sub-Saharan Africa, including South Africa, remain sparse (SANHANES-1, 2013). Lee *et al*. (2012) aimed to quantify, using PAFs and life-table analysis, the effect of physical inactivity on major NCDs by estimating how much disease could be avoided if inactive people were to become active and to estimate gain in life expectancy at the population level. In South Africa specifically, the PAF for all-cause mortality was calculated at 14% (±2.9), with a gain in life expectancy of 1.26 years (±0.27).

Looking at sex-specific data, studies indicate that physically inactive women have a 60-75% higher risk of CVD than their active counterparts (Peterson *et al*., 2005). A survey conducted by the WHO (2003) found that less than one-third of adult South Africans met the ACSM recommendation for health-enhancing physical activity, and 46% of all South African adults were reported to be inactive (<600 metabolic equivalent value minutes per week (METmins.week^{-1})). These low levels of physical activity were observed to be more prevalent in urban than rural settings (van Zyl *et al*., 2012). More recently the SANHANES-1 (2013) reported that one out of every two South African females (45.2%) was unfit and it was also concluded that being unfit was more prevalent in urban than in rural settings.

2.8.2.4. ALCOHOL CONSUMPTION

Regular alcohol consumption can have positive psychosocial and health effects (Cushman, 2001; Schneider *et al*., 2007). Several studies have shown that the regular consumption of moderate amounts of alcohol is associated with reduced BP and decreased overall mortality (Flesch *et al*., 2001; Fuchs *et al*., 2001; Di Castelnuovo, 2002; Yusuf *et al*., 2004). However, excessive alcohol intake results in many serious adverse psychosocial and health consequences. One of the main harmful effects of excess alcohol intake is its association with an increased risk of hypertension (Cushman, 2001; Fuchs *et al*., 2001; Klatsky, 2002; Schneider *et al*.,

2007). Alcohol has been estimated to account for 5-20% of hypertension in populations, and this positive relationship between amount of alcohol consumed and BP is one of the strongest associations of potentially modifiable risk factors for hypertension (MacMahon, 1987; Cushman, 2001).

Clinical guidelines on the primary prevention of hypertension recommend limiting alcohol intake ≤2 drinks.day^{-1} in men and ≤1 drink.day^{-1} in women (Campbell *et al*., 1999; Xin *et al*., 2001; Whelton, 2002; Joint National Committee, 2004; Huntgeburth *et al*., 2005; Appel *et al*., 2006). A 'standard drink' is defined as an alcoholic beverage containing 10g of ethanol. However there is some variance around these values as Sesso *et al*. (2008) found that the threshold above which alcohol became deleterious for hypertension risk emerged at ≥4 drinks.day^{-1} in women versus a moderate level of ≥1 drink.day^{-1} in men. Other epidemiological studies and intervention trials have found that the consumption of ≥3 drinks.day^{-1} is associated with an elevation of BP, independent from other factors such as age, sex, race, body weight and salt intake (Klatsky *et al*., 1986; Fuchs *et al*., 2001; Stamler, 2002). The impact of alcohol consumption on the prevalence of hypertension is comparable with obesity and greater than the daily intake of dietary sodium. Although much research has addressed the relationship between alcohol consumption and BP, there are limited data from studies conducted in black populations (Klatsky *et al*., 1986). Fuchs *et al*. (2001) addressed this by conducting a study to determine whether there is a threshold level for the association between alcohol consumption and hypertension in different ethnic groups. Results showed that the consumption of low-to-moderate amounts of alcohol appears to be associated with a higher risk of hypertension in black men (Fuchs *et al*., 2001).

Judging from the South African data over the past 30 years, there seems to be an increase in the proportion of people aged ≥15 years who drink alcohol (Parry, 2005). The data further show that there is an increase in life-time drinking among young black African males and females (Parry, 2005). In 2000, surveys indicated that approximately 50% of men and 20% of women drank alcohol in South Africa, although this is likely to be an underestimate (Schneider *et al*., 2007). The pattern of

drinking in South Africa is worrying given that a large portion of the population does not consume alcohol and so the amount consumed per adult drinker is close to 20 litres of absolute alcohol per year - amongst the highest in the world (Schneider *et al*., 2007).

2.8.2.5. TOBACCO USE

One of the major lifestyle changes associated with urbanisation is smoking, the prevalence of which is increasing in low- and middle-income countries, particularly in urban areas (Pearson, 1999; Boutayeb, 2006; Myer *et al*., 2004). This trend is found in South Africa and contributes to the increasing burden of chronic diseases, especially among the black African majority (Myer *et al*., 2004). Tobacco use is the leading cause of premature mortality globally. This 'bad habit' causes approximately 71% of lung cancer, 42% of chronic respiratory disease and nearly 10% of CVD. Presently it is estimated that tobacco use kills almost six million people a year, and, if the current trend continues, by 2030 tobacco will kill more than eight million people each year (WHO, 2011; SANHANES-1, 2013).

Tobacco use represents an important modifiable risk factor of CVD, with excess mortality from CVD and stroke being two-to-threefold higher among smokers than non-smokers (Bonow *et al*., 2002). Tobacco use is also known to increase the risk of complications among those already with hypertension (Mendis, 2013). According to the THUSA study, conducted in the North West Province of South Africa, there was no association between smoking and BP in men, but, in women, smoking was significantly associated with an increase in both systolic and diastolic BP. The lack of association in men may be due to other factors being more important determinants of BP in this population (van Rooyen *et al*., 2000). More recently, 4.8% of black South African females reported a history of ever having smoked tobacco and 4.6% reported on having ever used other tobacco products (SANHANES-1, 2013).

2.8.2.6. PSYCHOLOGICAL STRESS

The impact of stress on the development of hypertension is believed to involve a sympathetic nervous system response in which release of catecholamines leads to increased heart rate, cardiac output and BP (Spruill, 2010). The Medical Subject Headings defines stress as “a pathological process resulting from body response to external forces and abnormal states that tend to affect its homeostasis”. When emotional stressors prevail, this condition is known as psychological stress. Modern life events such as work-related and family problems, social withdrawal, financial worries and violence can predispose to or cause stress (Gasperin *et al*., 2009).

Several studies have identified and confirmed the link between chronic exposure to psychological stress and an increase in BP which may lead to the development of hypertension (Holroydf and Gorkin, 1983; Yan *et al*., 2003; Spruill, 2010; Carroll *et al*., 2012), with one study investigating this link in the black ethnic group (Wright *et al*., 2011). A systematic review followed by a meta-analysis by Gasperin *et al*. (2009) showed that individuals who had stronger responses to stressor tasks were 21% more likely to develop BP increase than those with less strong responses. Results therefore suggest the relevance of the control of psychological stress in non-therapeutic management of high BP. With the increase in urbanisation in South Africa, many black individuals have been exposed to a process of rapid urbanisation which may lead to social and cultural disruption causing increased levels of stress (van Rooyen *et al*., 2000). According to the most recent national survey, psychological distress (measured using the Kessler-10) is significantly greater in females than males with this risk factor being the most prevalent (34.2%) in black African females (SANHANES-1, 2013).

2.8.2.7. OTHER

2.8.2.7.1. Diabetes

CVD is the most prevalent and detrimental complication of diabetes mellitus as it is associated with a 2-4 times higher risk of CVD as well as an increased risk of mortality by up to three times (Mooradian, 2003; Li and Aronow, 2011; Matsha *et al*., 2012; Eskesen *et al*., 2013). Despite advances in the management of CVD, a large proportion of diabetic people continue to have uncontrolled hyperglycaemia, hypertension and dyslipidemia (Mooradian, 2003). Hypertension and diabetes are two of the leading risk factors for atherosclerosis and its complications, including heart attacks and strokes. There is substantial overlap between diabetes and hypertension, reflecting shared etiology and disease mechanisms (Cheung and Li, 2012). High BP is reported in over two-thirds of patients with type II diabetes (Ferrannini and Cushman, 2012).

The haemoglobin A_{1c} (HbA_{1c}) test evaluates the weighted mean plasma glucose concentration over the last two to three months and is relatively insensitive to short-term lifestyle changes (Eskesen e*t al*., 2013). The WHO recommends that HbA_{1c} be used as a diagnostic test for diabetes mellitus and the American Diabetes Association guidelines of 2013 state that HbA_{1c} levels ≥6.5% is one of four diagnostic criteria for diabetes mellitus (Eskesen *et al*., 2013) (Table 9).

Table 9: Diabetes classification (American Diabetes Association, 2013)

DIAGNOSIS	A_{1c} (%)	A_{1c} ($mmol.mol^{-1}$)
Non-diabetic	4-6	20-42
Increased risk of diabetes (pre-diabetes)	5.7-6.4	39-46
Diabetic	≥6.5	≥47

2.8.2.7.2. Hypercholesterolemia

Hypercholesterolemia is defined as excessively high plasma cholesterol levels and is a strong risk factor for many negative cardiovascular events (Stapleton *et al*., 2010). Increased BP and elevated total cholesterol levels are two major modifiable risk factors for CVD (Yang *et al*., 2011; Khonputsa *et al*., 2012). The full blood lipid profile is used as part of a cardiac risk assessment to help determine an individual's risk of heart disease (Walker *et al*., 2013). This lipid profile test includes total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TGs) (Table 10) (Maradit-Kremers *et al*., 2013; Walker *et al*., 2013; Zaman and Kumari, 2013). HDL-C is viewed as beneficial in higher amounts due to its association with a reduced risk of adverse cardiovascular events (Stapleton *et al*., 2010). Conversely, elevated LDL-C and TGs are considered detrimental as their increased concentration is associated with adverse cardiovascular outcomes. A high total cholesterol level is considered to be a major independent risk factor for the development of peripheral vascular disease and coronary artery disease (Stapleton *et al*., 2010).

Table 10: Female cholesterol classification according to the National Cholesterol Education Programme (Adapted from Stapleton *et al*., 2010)

TEST	RISK ($mmol.L^{-1}$)			
	Low	Borderline high	High	Very high
Total Cholesterol	<5.18	5.18-6.18	≥6.22	
HDL-C	≥1.55	1.3-1.5	<1.3	
LDL-C	≤3.34	3.37-4.12	4.15-4.90	≥4.90
TG	<1.70	1.7-2.2	2.3-5.6	>5.6

*HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; TG = triglycerides

2.8.2.7.3. Menstruation

Hormonal changes associated with the menstrual cycle are known to affect different organ systems. Fluctuation in the ovarian hormones during different phases of the menstrual cycle – menstrual, follicular and luteal – may affect cardiovascular function

in women (Dubey *et al*., 2002b; Pai *et al*., 2004), and therefore BP variations (Arifuddin *et al*., 2012). It is increasingly apparent that many of the cardiovascular adaptations to normal pregnancy are initiated in the luteal phase of every menstrual cycle and are amplified should conception occur (Pipkin and Roberts, 2000). A marked decrease in mean arterial pressure and total peripheral resistance in the mid-luteal phase has been observed (Pipkin and Roberts, 2000; Dubey *et al*., 2002; Robb *et al*., 2009). However there have been contradictory findings, as Pai *et al*. (2004) examined 75 healthy Indian females and reported that arterial BP does not significantly fluctuate during different phases of menstrual cycle.

2.8.2.7.4. Oral Contraceptive Use

The incidence of oral contraceptive-induced adverse effects is thought to be much lower now with the new generation of preparations, which contain lower doses of estradiol (Park and Kim, 2013). However, oral contraceptive use is still known to contribute to adverse health effects, including increased BP. This exogenous hormone administration contributes to the burden of hypertension in women (Joint National Committee, 2004; August, 2013; Park and Kim, 2013). The observed increase in BP in some women quickly returns to pre-treatment levels with cessation of oral contraceptives (three to four weeks), indicating that this effect is readily reversible (Clezy *et al*., 1972; Weir *et al*., 1974; Joint National Committee, 2004; Boldo and William, 2011).

2.8.2.7.5. Pregnancy

Hypertension complicates approximately 8-10% of pregnancies and is associated with an increase in maternal and neonatal morbidity and mortality worldwide (Symonds, 1995; von Dadelszen *et al*., 2000; Roberts *et al*., 2003; Joint National Committee, 2004). These hypertension-related disorders include chronic hypertension, pre-eclampsia, chronic hypertension with superimposed pre-eclampsia, gestational hypertension and transient hypertension (Pipkin and Roberts, 2000; Roberts *et al*., 2003; Joint National Committee, 2004). Hypertension in

pregnancy is also associated with increased rates of hypertension and coronary heart disease later in life (Ness *et al*., 2003).

2.8.2.7.6. Breastfeeding

Lactation results in a number of physiological adaptations which exert direct effects on maternal health, some of which may confer both short- and long-term advantages for breastfeeding mothers (Heinig and Dewey, 1997; Sikorski *et al*., 2003; Hoddinott *et al*., 2008). Stuebe *et al*. (2011) conducted an observational cohort study of just over 55.5 thousand parous women and found that not breastfeeding was associated with an increased risk of maternal hypertension, compared with the recommended ≥6 months of exclusive or ≥12 months of total lactation per child. Reasons behind this finding are that lactation activates central neuroendrocrine pathways, including oxytocin and prolactin, and these hormones have been associated with BP regulation and hypertension risk (Stuebe *et al*., 2011). In the short-term, the physiological changes of breastfeeding include the advantage of an immediate decrease in BP and cortisol levels in response to each individual breastfeeding session (Jonas *et al*., 2008).

2.8.2.7.7. Menopause

The menopause is defined as the absence of menstrual periods for 12 consecutive months, and occurs at the average age of 51 years (±10). This age range directly corresponds with the documented increase in coronary heart disease mortality in women (Agrinier *et al*., 2010), and thus menopause is now being considered as a special cardiovascular risk factor for women (He *et al*., 2012). The loss of endogenous oestrogen production after menopause is accompanied by an increase in BP, contributing to the high prevalence of hypertension in older women (Barton and Meyer, 2009; Agrinier *et al*., 2010; Woodard *et al*., 2011; He *et al*., 2012; Lisabeth and Bushnell, 2012). The first decade after menopause is also accompanied by an increase in BP. Specifically, there are pronounced increases in SBP and pulse pressure in postmenopausal women, whereas DBP remains at a

similar level compared with age-matched men. Importantly, SBP is considered a more sensitive predictor of future cardiovascular events than DBP (Barton and Meyer, 2009).

2.8.2.7.8. Drugs and other agents

Many prescription drugs, and some over-the-counter agents and herbal supplements, may affect BP and complicate its control in treated hypertensive individuals (Joint National Committee, 2004).

2.8.3. Interventions for High Blood Pressure

The quality of hypertension care received by South Africans is reflected in the proportion of persons with hypertension who are aware of having the condition, the proportion taking antihypertensive medication and the proportion with BP levels below the accepted target level (Steyn, 2005). Based on this criteria, and according to the 1998 SADHS data, black Africans had the worst treatment status for both sexes (Steyn, 2005). The SADHS (2003) data paint a slightly different picture, with black Africans having the second worst treatment status in both the male category (coloureds had the worst) and the female category (Caucasians had the worst). From a broad perspective, these generally low levels of detection, treatment and control of hypertension reported across all race and sex strata emphasize the difficulty in managing chronic health conditions that are usually asymptomatic and yet need life-long management to ensure control (Addo *et al*., 2007). Increased attention therefore needs to be applied to hypertension management, which encompasses pharmacological and/or non-pharmacological therapeutic approaches (Mertens and Van Gaal, 2000; Gasperin *et al*., 2009).

2.8.3.1. PHARMACOLOGICAL INTERVENTIONS

2.8.3.1.1. Antihypertensive Medication

Due to hypertension often being asymptomatic, patients are often required to take multiple medications at increased dosages, and long-term to prevent its complications (Brown *et al*., 2003; Joint National Committee, 2004; Cheung *et al*., 2005; Yiannakopoulou *et al*., 2005; Lewanczuk and Tobe, 2007). Hence, clinicians have been aiming at better BP control, using more classes of drugs to achieve it. A large number of drugs (including calcium channel blockers, β-blockers, angiotensin-converting enzyme inhibitors, thiazide diuretics, α-blockers and angiotensin receptor blockers) are available for lowering elevated BP (Cheung *et al*., 2005; Gu *et al*., 2006). Although these drugs have been shown to reduce hypertension-related morbidity and mortality, they are also associated with adverse side-effects (Gasperin *et al*., 2009). Side-effects arising from multiple drugs at high dosage may affect the quality of life of the patient, and reduce compliance, hence side-effects are becoming an important issue (Cheung *et al*., 2005). Cheung *et al*. (2005) carried out a survey of patients in a hypertension clinic as part of the Queen Mary Utilisation of Antihypertensive Drugs Study programme. They found that among patients on antihypertensive drug therapies, 34% reported adverse effects: dizziness (9%), ankle swelling (7%), headache (5%), fatigue (4%), chest discomfort (3%) and cough (3%). The likelihood of experiencing side-effects was unrelated to sex, age, weight, BMI, years of treatment, numbers of drugs used, heart rate on treatment or compliance (Cheung *et al*., 2005).

Poor compliance with antihypertensive drug regimens is a well-documented reason for inadequate control of hypertension (Kjellgren *et al*., 1995; Benson and Britten, 2002; Yiannakopoulou *et al*., 2005). Depending on differences in definition and measurement, a broad estimate of how many patients do not take their prescribed medication is approximately 50% (Kjellgren *et al*., 1995; Benson and Britten, 2002; Thrall *et al*., 2004). Regimens with fewer pills or once-daily dosing are more likely to be associated with enhanced adherence than more complicated regimens. As a result, one strategy that may be effective in promoting adherence is reducing the

number of pills by using fixed-dose combination therapies (Lewanczuk and Tobe, 2007). Another barrier to achieving effective BP control is the cost of medications. From an individual perspective, patients may be unable to afford multiple antihypertensive medications. From a system perspective, drug costs are a major contributor to overall health care costs (Lewanczuk and Tobe, 2007; Gasperin *et al*., 2009). There is also the psychological aspect to the use of fixed-dose combination medications in chronic diseases such as hypertension. There is an association between the number of pills patients take and their self-perceived health, which has implications for both mental well-being and physical health (Lewanczuk and Tobe, 2007).

2.8.3.2. NON-PHARMACOLOGICAL INTERVENTIONS

Despite the abundance of pharmaceutical options for the treatment of hypertension, the identification of non-pharmacological methods to prevent, or significantly delay, the onset of hypertension represents an important approach to the prevention of CVD (Gasperin *et al*., 2009; Woolf and Bisognano, 2011). Lifestyle changes are recommended, in this context specifically by the South African Hypertension Guidelines, for pre-hypertensive individuals, and as initial therapy in Stage 1 hypertension (Joint National Committee, 2004; Svetkey *et al*., 2005; Rayner, 2010). These lifestyle changes to lower BP include cessation of smoking, limited alcohol consumption, education/awareness, stress management, weight reduction, an improved diet (particularly the Dietary Approaches to Stop Hypertension – DASH diet), salt restriction and physical activity (Svetkey *et al*., 2005; Gasperin *et al*., 2009; Woolf and Bisognano, 2011). For individuals taking antihypertensive medication, lifestyle modification is recommended as adjunctive therapy (Svetkey *et al*., 2005). Lifestyle modifications may be cost-free or may even save money (for example cessation of smoking and reduced alcohol consumption). Furthermore, the beneficial effects of lifestyle modifications may include reduction in the amount and cost of prescribed medications and the cost of insurance (Joint National Committee, 2004). For the purposes of this review I will focus on exercise interventions.

2.8.3.2.1. Physical Activity

Increased physical activity has specifically been recommended as an important lifestyle modification for the prevention and management of hypertension (Bravata *et al*., 2007; Sohn *et al*., 2007; Lee *et al*., 2010). Evidence supporting the effect of exercise on BP reduction is well documented, however, despite the strength of this association, the exact characteristics for a physical activity programme to generate optimal effect on BP control remains a matter of substantial debate (Higashi *et al*., 1999; Bassuk and Manson, 2005; Murphy *et al*., 2007; Sharman and Stowasser, 2009; Lee *et al*., 2010). The Australian Association for Exercise and Sports Science state that the minimum amount of exercise that is recommended in patients with hypertension should comprise a mix of moderate-to-vigorous aerobic activity (up to five days.week^{-1}) in combination with resistance training (on two or more non-consecutive days.week^{-1}) (Sharman and Stowasser, 2009).

2.8.3.2.1.1. Resistance Exercise

Resistance exercise (also known as weight or strength training) is characterised by performance in which muscles of a specific body segment are contracted against an opposing force (Cardoso *et al*., 2010). Dynamic resistance exercise has been recommended as a component of exercise programmes for health maintenance in patients with CVD. Similarly, the American Heart Association and the ACSM have endorsed moderate-intensity resistance training as a complement to aerobic exercise in the prevention, treatment and control of hypertension (Braith and Stewart, 2006; Kokkinos and Myers, 2010). Cardoso *et al*. (2010) performed a systematic review of the acute and chronic effects of aerobic and resistance exercise on ambulatory BP in normotensive and hypertensive participants. The authors concluded that aerobic training should be recommended to decrease ambulatory BP in hypertensive individuals, while resistance exercise could be prescribed as a complementary strategy.

The Australian Association for Exercise and Sports Science recommend that dynamic resistance exercises be performed in a rhythmical fashion, through the full range of motion, at a moderate-to-slow and controlled speed, with emphasis on eccentric contractions and maintenance of a normal breathing pattern (Sharman and Stowasser, 2009). However, the cardiovascular responses during this kind of exercise are poorly understood, especially in individuals with hypertension (Sharman and Stowasser, 2009; Collier *et al*., 2011). A meta-analysis conducted by Fagard (2006) looked at nine RCTs (12 study groups and 341 participants) and the resultant BP effect from resistance training. There was no differentiation between normotensive and hypertensive participants (which may compromise the applicability of results) although the average BP across all studies was reported as 131/81 mmHg. Results revealed a weighted net reduction of DBP (3.6 mmHg) associated with exercise and a non-significant reduction of SBP (3.2 mmHg) (Fagard, 2006). A second, more recent meta-analysis looking at the same issue investigated 28 RCTs (33 study groups and 1012 participants). Overall, resistance training induced a significant BP reduction in normotensive or pre-hypertensive study groups (average decrease of 3.9/3.9 mmHg) but the reduction was not significant in the hypertensive study groups (average decrease of 4.1/1.5 mmHg) (Cornelissen *et al*., 2011). Although these reductions seem modest, a SBP reduction of 3 mmHg in average populations has been estimated to reduce cardiac morbidity by 5-9%, stroke by 8-14% and all-cause mortality by 4% (Braith and Stewart, 2006).

2.8.3.2.1.2. Aerobic Exercise

Aerobic exercise (also known as endurance exercise) is characterised by the execution of cyclic exercises, carried out with large muscle groups contracting at mild-to-moderate intensities for long periods (Cardoso *et al*., 2010). Regular aerobic exercise by patients with essential hypertension is associated with beneficial changes in BP (Higashi *et al*., 1999). A meta-analysis of controlled trials (≥ four weeks) in healthy adults investigated the effect of exercise on cardiovascular risk factors. With regard to dynamic aerobic training effects on BP, this meta-analysis found a significant average reduction (7.1/5.2 mmHg) (Pattyn *et al*., 2013). Aerobic exercise can include activities such as walking, jogging, swimming, cycling and stair

climbing (Agarwal, 2012; Dimeo *et al*., 2012). Importantly, in terms of sustainability in sedentary and/or overweight populations, equivalent energy expenditures by moderate (walking) and vigorous (running) exercise have been shown to produce similar risk reductions for hypertension (Williams and Thompson, 2013).

Walking has been referred to as “the nearest activity to perfect exercise” as it is a low-cost and low-risk physical activity that most people can undertake (Murtagh *et al*., 2005; Murphy *et al*., 2007; Lee *et al*., 2010; Rowe *et al*., 2013). Further, walking is one of the safest and simplest exercises for hypertensive patients of all age groups and so should be considered in physical activity promotional campaigns (Murphy *et al*., 2007; Sohn *et al*., 2007). Walking can be performed at a variety of speeds, in a group or alone and without the need for special equipment or clothing (Murphy *et al*., 2007; Kassavou *et al*., 2013). Walking at an easy or moderate pace is considered a low-intensity activity, while brisk and very brisk walking are considered moderate-intensity exercise and sometimes greater than moderate-intensity (Lee *et al*., 2010). Ten minutes of brisk/moderate-intensity walking can be equated to approximately 1000 steps (Baker and Mutrie, 2005). Many interventions that aim to increase total volume of walking use simple intensity instructions such as “brisk”, and this gives confidence that previously inactive adults will respond to this instruction by walking at a pace that is above moderate-intensity (Murphy *et al*., 2007; Rowe *et al*., 2013).

Despite the appeal of walking as a method of reducing cardiovascular risk, interventions which have considered the effects on BP of a programme of brisk walking have yielded unclear results (Murphy *et al*., 2007; Lee *et al*., 2010) so further research is required. A meta-analysis of 9 RCTs by Murphy *et al*. (2007) showed that a regular brisk walking programme in healthy, sedentary individuals had no effect on SBP but significantly reduced DBP by -1.54 mmHg (±0.79). The average baseline DBP was 77.7 mmHg (±4.5), which underlines the potential of a walking programme to improve DBP levels even in normotensive individuals. Similar support for this alternative therapy comes from Lee *et al*. (2010) who conducted a meta-analysis of 27 RCTs, of which 9 reported an effect of walking on BP. These beneficial effects

tended to occur in studies which had a large sample size, higher baseline BP levels, employed moderate- to high-intensity walking and had longer intervention periods than those trials not showing an effect. Among the trials that showed an effect, systolic and diastolic BP reduced by 5.2-11 mmHg and 3.8-7.7 mmHg, respectively. Most walking interventions in this review followed a commonly applied physical activity recommendation which involved exercising on most, preferably all, days of the week, at a moderate-intensity, for 20-60 min.session^{-1} (Lee *et al*., 2010). Table 11 illustrates numerous walking intervention studies which investigated the exercise effect on BP.

Noteworthy from Table 11 are the several studies with effective walking programmes that have been completed unsupervised and in the participants' own locality and time (Iwane *et al*., 2000; Moreau *et al*., 2001; Tully *et al*., 2005; Sohn *et al*., 2007; Tully *et al*., 2007; Miyazaki *et al*., 2013). The unsupervised nature of these programmes has been possible through the use of pedometers which are simple and inexpensive devices that can successfully be used to monitor, assess and motivate physical activity (Tudor-Locke and Bassett, 2004; Baker and Mutrie, 2005; Crouter *et al*., 2005; Heesch *et al*., 2005; Bravata *et al*., 2007; Jackson and Howton, 2008; Nakae *et al*., 2008; Lubans *et al*., 2009; McKay *et al*., 2009; Tudor-Locke and Lutes, 2009; Zoellner *et al*., 2009; Lee *et al*., 2010; Zoellner *et al*., 2010). There are several types of pedometer with the two main styles being spring-levered and piezo-electric (Foster *et al*., 2005). Piezo-electric pedometers are less susceptible to errors that occur due to tilt, which is especially important for use in overweight and obese populations, and so have generally been found to be more accurate (Crouter *et al*., 2005). These pedometers are further recommended for obese individuals since they are more accurate at slower walking speeds (<80 m.min^{-1}) (Crouter *et al*., 2005). Use of these devices can be crucial in walking intervention strategies such as the common set goal of 10000 steps.day^{-1} and individual goals involving baseline measures, both of which have been found to be successful techniques (Kang *et al*., 2009).

Table 11: Summary of several walking intervention studies and their effect on blood pressure

STUDY	'FIND' PRINCIPLE	SAMPLE	FINDINGS
Iwane *et al*., (2000)	≥10000 steps.day^{-1} 12 weeks	Male hypertensive adults EXC = 30; CON = 17	Walking ≥10000 steps.day^{-1}, irrespective of intensity or duration, is effective in lowering BP (average reduction of 10.2/8.4 mmHg) in hypertensive patients.
Moreau *et al*., (2001)	Extra 3 km.day^{-1} 24 weeks	Post-menopausal women with mild-to-moderate hypertension EXC = 15; CON = 9	SBP significantly decreased after week 12 by 6 mmHg and was further reduced by 5 mmHg at week 24. There was no change in DBP.
Murtagh *et al*., (2005)	20 mins (1/2 bouts) for 3 days.week^{-1} Brisk walking 12 weeks	Sedentary adults EXC 1 = 12; EXC 2 = 11; CON = 8	No effect on BP.
Tully *et al*., (2005)	30 mins for 5 days.week^{-1} Brisk walking 12 weeks	Healthy, sedentary adults EXC = 17; CON = 9	Significant decrease in SBP (11.82 mmHg) and DBP (4 mmHg).
Tully *et al*. (2007)	30 mins for 3 or 5 days.week^{-1} Brisk walking 12 weeks	Healthy, sedentary adults EXC 1 = 44; EXC 2 = 42; CON = 20	SBP fell significantly in the three day group (5 mmHg) and the five day group (6 mmHg). DBP only fell in the five day group (3.4 mmHg).
Sohn *et al*., (2007)	30 mins for 5-7 days week^{-1} Walking pace not specified 26 weeks	African-American hypertensive adults EXC = 8; CON = 10	SBP dropped by 9% and DBP by 7.42%.
Miyazaki *et al*., (2013)	Individualised step goals 59 weeks	Physically active older people EXC = 36; No CON group	SBP significantly reduced (9.1 mmHg)

* EXC = exercise group; CON = control group; BP = blood pressure; DBP = diastolic blood pressure; SBP = systolic blood pressure

As regards the duration of a walking intervention, a longer intervention period may lessen the adherence to the programme (Lee *et al*., 2010). This is supported by Kang *et al*. (2009) who found that due to the difficulties in having an intervention length >15 weeks, an intervention length ≤15 weeks may be a feasible, effective and inexpensive alternative to longer interventions (Kang *et al*., 2009). Several RCTs have specifically found walking interventions with a 12 week duration to be effective in decreasing both SBP and DBP in sedentary individuals (Higashi *et al*., 1999; Murtagh *et al*., 2005; Tully *et al*., 2005; Tully *et al*., 2007).

2.9. SUMMARY OF THE LITERATURE

It is evident that NCDs are rapidly increasing in South Africa, and, together with the burden of CDs, are placing huge strain on the already limited health resources. CVD in particular has reached epidemic proportions largely as a result of urbanisation. This process, and associated adoption of unhealthy lifestyles, is primarily being experienced by the majority black African group who are also the most impoverished, and are at high risk of CVD morbidity and mortality. There is a wealth of information on the multiple risk factors for CVD, with these having been identified quite accurately in many population groups within South Africa. Blood pressure is the primary risk factor, and is well-recognised both on a national and global scale. Despite this, hypertension prevalence remains high in South Africa and there is a need to address the problem. Black African females have been identified as an 'at risk' population due to a host of genetic, environmental and social factors. However, there is relatively little information on this largely uneducated and impoverished group within the Eastern Cape. As a result they have limited access to health services and require cost-effective approaches to combat this burden. One such cost-effective approach is an increase in physical activity which has specifically been recommended as an important lifestyle modification for the prevention and management of hypertension. This can be achieved through the adoption of brisk walking as a form of physical activity as it is low-cost and low-risk. Further, several unsupervised walking programmes (the nature of which would make the programme more feasible in this population group) have proven effective with the aid of pedometers, functioning as both a monitoring and motivational tool.

CHAPTER 3

METHODOLOGY

3.1. RESEARCH AIM

The primary goal of this research was to assess the cardiovascular disease (CVD) risk in a sample of black African females in the Makana region of the Eastern Cape Province of South Africa. The research also aimed to assess the efficacy of a 12 week walking programme on blood pressure (BP) in a mild-to-moderately hypertensive sub-sample from the same area.

3.2. RESEARCH DESIGN

This first part of the study was a comparative descriptive design. Baseline measures of specific CVD risk factors were collected from all participants and the data were compared with existing norms from a similar population.

The intervention part of the study was designed as a quasi-experimental study. Two conditions were tested: an experimental exercise (EXC) condition where participants underwent a 12 week progressive walking programme, and a control (CON) condition where participants received no treatment and were instructed to maintain their usual lifestyle which included no exercise participation. Participants were specifically allocated to either the EXC or the CON condition based on their shift schedules within the Rhodes University (Grahamstown, South Africa) dining hall or residence in which they worked. These participants were exposed to a battery of measurements at weeks 0, 4, 8, and 12 (Table 12) at the same selected time of the day to minimise circadian factors influencing the results. The principal researcher and research assistants were allocated to specific stations and performed the same measurements on each occasion so as to reduce inter-individual variability. At the end of the 12 week study members of the CON group were advised on a walking

programme so as to afford the benefits of the exercise intervention to all participants; however, this was not monitored and no data were collected or used.

Table 12: Design matrix

	Week 0	Week 4	Week 8	Week 12
Exercise Group (EXC)				
Control Group (CON)				

3.2.1. Dependent and Independent Variables

At week 0 all participants had various anthropometric, morphological, cardiovascular, physical activity, biochemical and behaviour factors measured. These included stature, body mass, and body mass index (BMI) – anthropometric variables; waist circumference, fat mass and lean mass – morphological variables; heart rate and BP – cardiovascular variables; step count and energy expenditure – physical activity variables; glycated haemoglobin and full blood lipid profile – representing biochemical variables; and alcohol and tobacco use – as behavioural variables. The EXC and CON group participants further had these measurements repeated at weeks 4, 8, and 12 to assess the effect of the exercise intervention. In addition to these measures, the intervention groups had their medication usage screened only at baseline, and monthly monitoring of dietary intake and fitness level. According to these criteria, exercising females were compared to non-exercising females against the before mentioned dependent variables as seen in Table 13.

Table 13: Dependent variables

	DEPENDENT VARIABLES				
	Anthropology	Morphology	Cardiovascular	Physical Activity	Other
EXC	Stature, BM, BMI	WC, FM, LM	HR, BP	Step count, fitness, EE	Diet, HbA_{1c}, FBLP
CON	Stature, BM, BMI	WC, FM, LM	HR, BP	Step count, fitness, EE	Diet, HbA_{1c}, FBLP

* BM = body mass; BMI = body mass index; WC = waist circumference; FM = fat mass; LM = lean mass; HR = heart rate; BP = blood pressure; EE = energy expenditure; HbA_{1c} = glycated haemoglobin; FBLP = full blood lipid profile

3.3. ETHICS

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

3.4. PILOT TESTING

Prior to the start of the research, the initial requirements for the study, parts of the screening process and the test procedures were performed on a random sample to ensure accuracy and precision of techniques and methods. The initial requirements for the study included an introductory meeting and the signing of consent forms. The screening process included the completion of three different questionnaires, a Rockport walk test and a three-day dietary recall. Baseline step counts were obtained using individual pedometers and all testing procedures, including anthropometric, morphological, cardiovascular, physical activity and control measures (excluding biochemical analyses), were taken. The pilot testing procedure facilitated changes to the way the data sheets were structured, how the measurement procedures were explained and the replacement of the ‘Rockport walk test’ with the ‘six-minute walk test’ (6MWT).

3.5. PARTICIPANTS

3.5.1. Recruitment

Participants were recruited from the greater Makana region in the Eastern Cape, South Africa, specifically from the Rhodes University catering and housekeeping staff. Participants were recruited via formal and informal meetings, pamphlet distribution (Appendix 2) and poster advertisements (Appendix 3).

3.5.2. Inclusion Criteria

The following criteria were considered when recruiting individuals for participation:
Rhodes University catering and housekeeping staff: to allow for the recruitment of a homogenous sample who experience similar home and work lifestyles, therefore reducing confounding variables. The Grahamstown area has a limited sample pool and so participants were formed solely from this tertiary education staff population.

Female: the 2013 South African National Health and Nutrition Examination Survey (SANHANES-1) indicates hypertension prevalence (defined as self-reported hypertension or those being measured as hypertensive using cut-off points of 140/90 mmHg) to be identical across sexes (10.2%). However, data from the 2003 South African Demographic and Health Survey (SADHS) indicated that black African females specifically have a far greater prevalence of hypertension than their male counterparts (17.4% and 10.0% respectively). Sex was determined with the 'Participant Screening' form.

Black African race: of the different population groups within South Africa, black Africans are the most impoverished and have the worst treatment status for hypertension across sexes. Race was validated via the 'Participant Screening' form.

Aged 25 to 44 years: prevalence of hypertension increases steadily with increasing age in both males and females. The lower age limit of 25 years was selected to include adult females who were an 'at risk' population within the black African group (Steyn, 2005). Further, BP is affected by hormonal changes associated with menopause and thus the upper age limit of 44 years was chosen to exclude females

who were menopausal or post-menopausal. This was validated via the 'Participant Screening' form.

Able to read and write English: participants were required to communicate with the principal researcher and research assistants, who were English speaking, and to read and respond to English-based information letters, questionnaires and daily diaries. This was confirmed with the 'Participant Screening' form.

Mild (120-139/80-89 mmHg) or moderate (140-159/90-99 mmHg) hypertension: lifestyle modifications as opposed to pharmacological treatment options are recommended as initial therapy for individuals with mild-to-moderate hypertension. All participants therefore had elevated seated resting BP (confirmed via manual measurement) as this was the primary variable of interest.

Overweight (25-29.9 kg.m^2) or obese class I (30-34.9 kg.m^2): obese adults have a 3.5-fold increased likelihood of having hypertension. Given that a large proportion of South African black women are classified as overweight (24.9%) or obese (39.9%), it was important that this variable be measured and controlled for.

No uncontrolled diabetes, hypercholesterolemia, or major surgery in the last six months: to ensure safety to the individual as this study involved an exercise intervention as well as to prevent these conditions from affecting BP measures. This was ensured via the 'Participant Screening' form and biochemical analyses.

No prior or current heart, lung, liver or kidney disease: no self-reports of any heart, lung, liver or kidney disease for which participants are medicated, as medication for certain conditions could mask the effects of the exercise intervention. This was validated with the 'Participant Screening' form, a Medication questionnaire and a physical examination by a specialist physician.

Non-smokers (tobacco): smoking has been significantly associated with increased systolic and diastolic BP in South African females. Participants were all non-smokers, and if they had previously smoked, they had to have given up the habit at least 12 months prior to recruitment. This was ensured with the 'Participant Screening' form.

Not heavy drinkers: as defined as the consumption of alcohol in amounts ≥210 $g.wk^{-1}$ (approximately 3 $drinks.day^{-1}$ containing 10 g ethanol) since this is an independent risk factor for hypertension. Alcohol consumption was measured via the 'Participant Screening' form and the CAGE questionnaire and participants who were classified as 'heavy' drinkers were excluded.

Sedentary: defined as taking ≤5000 $steps.day^{-1}$. This was validated via the 'Participant Screening' form, the Global Physical Activity Questionnaire (GPAQ) and a one-week habituation period with a pedometer to determine baseline daily steps.

No musculoskeletal injury: this research adopted a walking intervention of moderate-intensity, so it was important that participants were fit to undertake exercise. This was ensured with the 'Participant Screening' form and a physical examination by a specialist physician.

Not currently using or having used antihypertensive medication within the past month: in order to isolate the effects of the exercise intervention on BP responses. This was validated via the 'Participant Screening' form and a 'Medication' questionnaire.

Menstruation: in general there is a significant fall in mean arterial pressure and total peripheral resistance during the mid-luteal phase of the menstrual cycle. Measurements were taken at monthly intervals and therefore the effects of the different phases of the menstrual cycle could be reduced.

Not pregnant and/or breast feeding: these conditions have been found to have an effect on BP (can either raise or lower it), so it was important that women who were pregnant and/or breast feeding be excluded from this study. This was ensured with the 'Participant Screening' form.

Not currently on any oral contraceptive medication: oral contraceptive medication (specifically oestrogen-based) increases systolic blood pressure (SBP), so this was controlled for by exclusion based on the 'Participant Screening' form and a 'Medication' questionnaire.

Not using drugs or other agents: participants on any prescription drugs, over-the-counter agents, and/or bio-identical supplements that may affect BP were excluded

based on their response to the ‘Participant Screening’ form and a ‘Medication’ questionnaire.

3.5.3. Initial Requirements for the Study

After recruitment an introductory meeting was held where the study protocol was explained in detail, both verbally and in written form (Appendix 4), and questions from potential candidates were encouraged. An interpreter (English/isiXhosa) was present to assist with any communication issues. Each participant was then required to sign a written consent form (Appendix 5) to participate in this study.

3.5.4. Pre-Screening

Prior to the start of the study there were five levels of pre-screening which all participants were required to undergo as this formed the baseline measures and the descriptive part the study. Further, meeting the inclusion criteria at each of these stages was necessary for acceptance into the intervention part of the study.

The first level of screening involved completing a ‘Participant Screening’ form (Appendix 6), which outlined some of the basic inclusion criteria. Participants then had anthropometric, morphological and cardiovascular variables measured and recorded in a monthly data sheet (Appendix 7).

The second level of pre-screening saw each participant complete three different questionnaires under the supervision and aid of the principal researcher or research assistant. The questionnaires included an alcohol use questionnaire (CAGE questionnaire) (Appendix 8) to determine habitual use of this substance, the GPAQv2 (Appendix 9) to monitor energy expenditure (metabolic equivalent (METS)) and a medication questionnaire (Appendix 10) to reduce confounding variables.

The third level of pre-screening was a physical examination by a specialist physician, which was necessary to ensure safety, as individuals who may have been at risk of a cardiovascular complication if they undertook physical activity were excluded from the study. This was important because based on the inclusion criteria of this research all participants were classified as a 'high risk' group according to the ACSM's guidelines for exercise testing and prescription. This 'high risk' classification was due to the participants being sedentary, overweight/obese class I, and having mild-to-moderate hypertension, which were all necessary characteristics to be accepted into this study. This meant that for moderate-intensity exercise, current medical examination and exercise testing prior to participation, as well as physician supervision of exercise tests, was recommended (Balady *et al*., 2000; Armstrong, 2006).

The fourth stage involved the 6MWT (Appendix 11) for each participant. Following this exercise, each participant was required to wear their pedometer and record their daily step count in a diary (Appendix 12) for a one-week period so as to determine baseline step counts and therefore sedentarism.

The fifth and final level of pre-screening involved each participant being given a date and time in which to visit 'Du Buisson & Partners' situated in Peppergrove Mall, Grahamstown. This is an accredited medical testing laboratory (Appendix 13). Blood work was done in a fasted state to measure glycated haemoglobin (HbA_{1c}) and full blood lipid profile (FBLP). If the results indicated diabetes and/or high cholesterol the participant was excluded from the intervention part of the study.

This study attempted to isolate the effects of exercise on BP, so participants were advised accordingly regarding weight gain and energy expenditure. Participants were instructed to maintain their 'normal' habitual diet throughout the exercise intervention so as not to lose or gain weight. This was monitored via a three-day dietary recall (Appendix 14). Additionally, participants were required to complete the

GPAQv2 to track their energy expenditure. These measures were repeated at regular intervals (at weeks 0, 4, 8 and 12).

3.5.4.1. OUTCOME OF PRE-SCREENING ASSESSMENTS

The following flow diagram represents the pre-screening process and shows the number of participants (as an entire group so before participants were allocated to either the exercise or the control groups) successfully fulfilling each criterion (Figure 6). Based on the low numbers of participants' fulfilling specific criteria, several variables from the inclusion criteria list had to be removed to make it possible to recruit large enough cohorts for the intervention aspect of this study. The following variables, showing the percentage of participants who fulfilled the specified criteria, were removed: age (83%), presence of menstruation (68%), weight (23%), sedentary behaviour (3%), breast feeding (95%) and the use of contraceptive medication (85%).

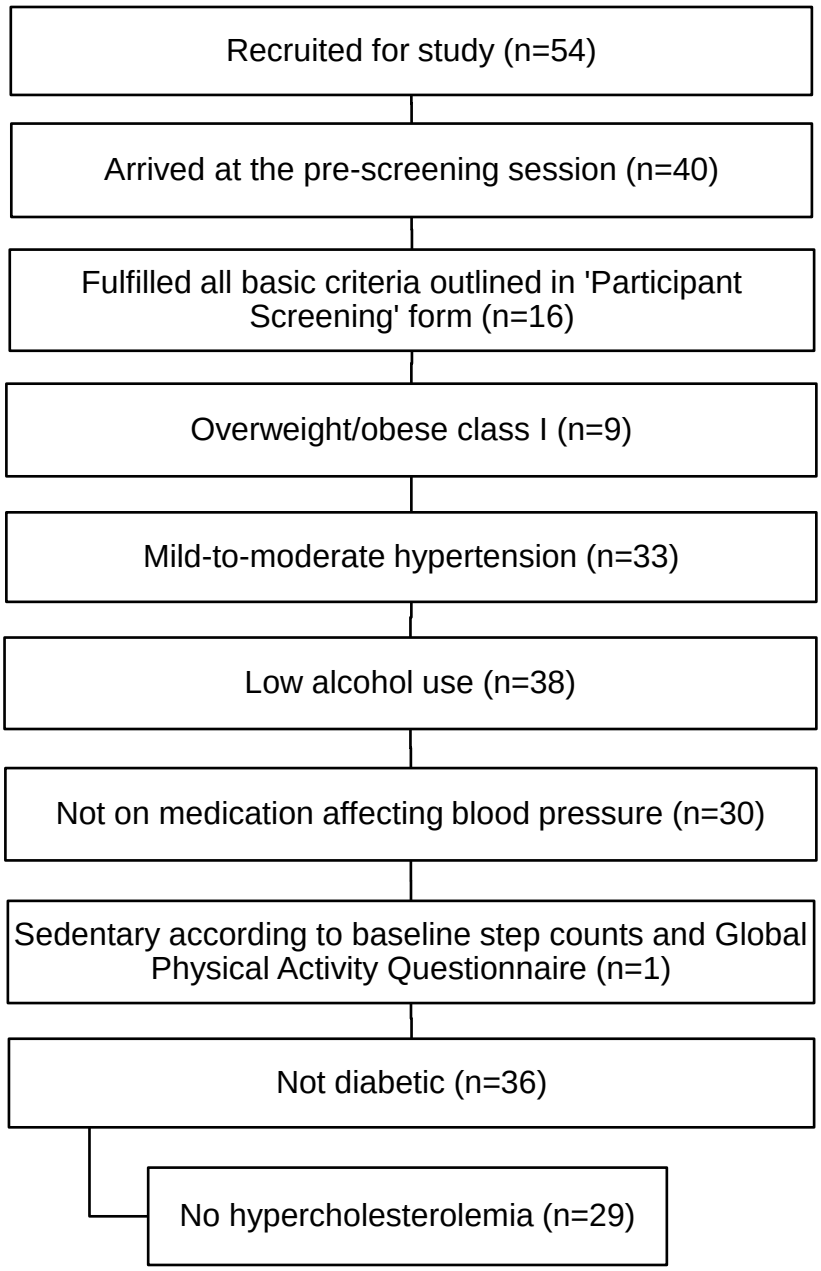

Figure 6: Flow diagram of the pre-screening process

Following this process, the remaining eligible participants included 25 women who were then allocated into either the EXC or the CON group based on their work schedules and thus availability (Figure 7).

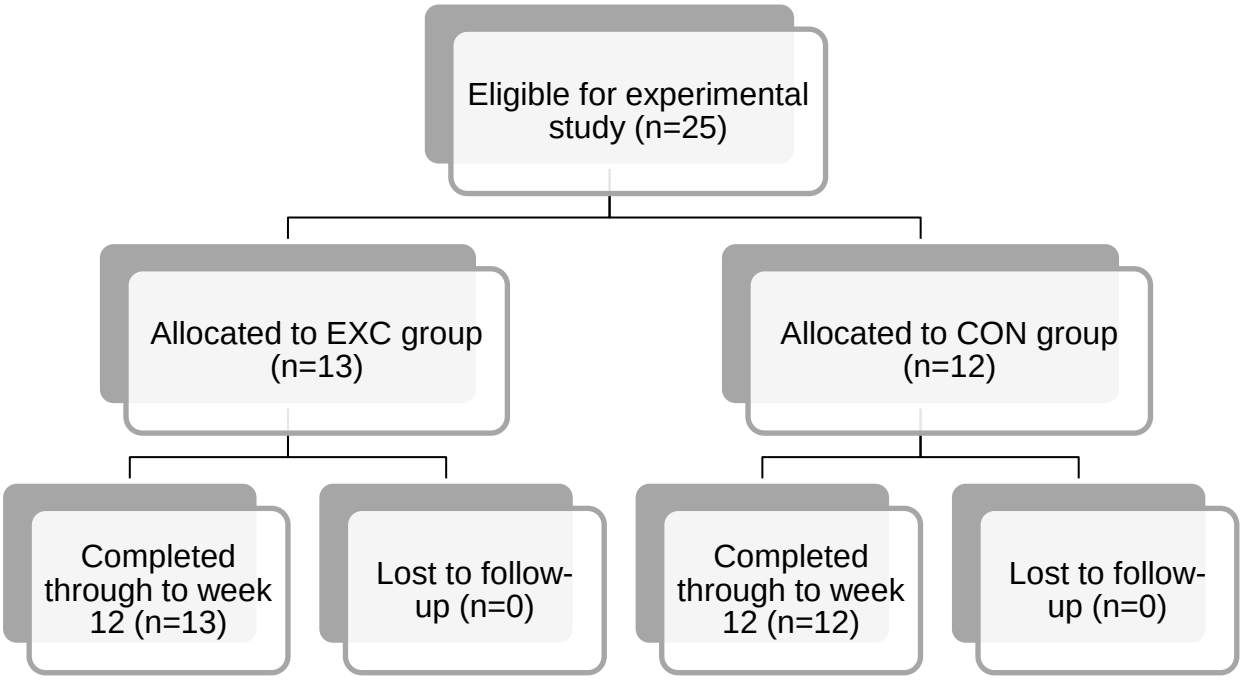

Figure 7: Flow diagram of the intervention study process (allocation to either EXC or CON groups)

All participants (in both the EXC group and the CON group) were measured at each time point throughout the 12 weeks and thus none were lost to follow-up.

3.6. MEASUREMENTS

3.6.1. Physical Examination

Prior to being selected for this research programme each potential candidate was examined by a specialist physician to ensure safety during exercise. The checklist used by the specialist physician is included (Appendix 15).

3.6.2. Anthropometry

3.6.2.1. STATURE

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

3.6.2.2. 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 to wear minimal clothing at the time of measurement so as to obtain accurate results.

3.6.2.3. BODY MASS INDEX (BMI)

BMI ($kg.m^{-2}$) is a well-known measurement in the assessment of fatness in relation to height, and despite the limitation of being unable to distinguish between fat and lean mass, it is accepted as one of the standard measures of obesity (James *et al*., 2001). BMI was determined from measurements of stature and body mass using the following formula: body mass (kg) / stature (m^2).

3.6.3. Morphology

3.6.3.1. WAIST CIRCUMFERENCE

Waist circumference, as a criterion for central fatness, is one of the strongest predictors of disease within population groups and is a convenient indicator of abdominal adipose tissue. Iliac waist circumference was measured with a standard

dressmaker's tape measure and was recorded to the nearest millimetre. This measurement was performed with the participant in a standing position, without heavy outer garments and at the end of normal expiration (Janssen *et al*., 2004). The iliac waist circumference measurement has been found an accurate technique when measuring changes in central adiposity and is suitable for people of all sizes, although palpating bony landmarks can be challenging (Fogli, 2005).

3.6.3.2. BIOELECTRICAL IMPEDANCE ANALYSIS (BIA)

In obese populations, dual-energy X-ray absorptiometry cannot measure individuals who weigh >113 kg; hydrostatic weighing requires large participants to climb into a tank that may cause claustrophobia, embarrassment, or be too physically challenging, and studies measuring subcutaneous fat with callipers have failed to validate the method once the fat fold exceeds the calliper's capacity (Fogli, 2005). Within controlled clinical conditions, BIA is a reliable measure of body composition and although it is not the most accurate technique available, its appeal lies in being non-invasive, simple, relatively inexpensive and portable (Dehghan and Merchant, 2008; Aleman-Mateo *et al*., 2010; Leahy *et al*., 2012; Saladino, 2012). The technique has previously been used in exercise intervention studies to assess body composition in sedentary women (Murtagh *et al*., 2005; Smith *et al*., 2007).

The Lipotrak® BIA equipment (Marulatech, Cape Town, South Africa) was used to estimate body composition. The specific measures obtained from the BIA technique were lean mass (kg and %) and fat mass (kg and %). According to Saladino (2012) this whole-body technique introduces a current (800µA) into the body at a single-frequency (50kHz) to determine the electrical impedance of body tissues based on 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 the length of the conductor)

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

Participants were required to remove all jewellery, not to have exercised 24 hours before testing, and having had no food or fluid intake two hours prior to this measure. Participants were required to remove their right shoe and sock and to lie in a supine position. The participant's legs were extended (knees straight) and spread so that the thighs did not touch (ankles at least 20 cm apart). The arms were extended next to the body in a relaxed state and not touching the body. The appropriate areas were cleaned with sterile alcohol swabs (Kendall Webcol™, TycoHealthcare, Midrand, South Africa) and then self-adhesive electrode pads (Skintact® Low Cost Multipurpose ECG Electrodes, Leonhard Lang GmbH, Innsbruck, Austria) were attached to the right hand (red cable was placed between the styloid processes of the radius and ulna; the black cable was placed just above the metacarpal-phalangeal joint line) and right foot (red cable was placed bisecting the medial and lateral malleoli; and the black cable was placed just above the metatarsal-phalangeal joint line). Once this had been completed, the appropriate information was entered into the BIA machine and then the small electric signal was circulated.

3.6.4. Cardiovascular

3.6.4.1. HEART RATE

Heart rate ($bt.min^{-1}$) was measured in triplicate by the same research assistant using the manual palpation method. Participants were required to rest quietly in a seated position for at least five minutes prior to the measurement. Heart rate was taken by placing two fingers on the carotid artery and counting the number of beats for 15 seconds and then multiplying this value by four. The two closest readings were averaged and this served as the resting value.

3.6.4.2. BLOOD PRESSURE (BP)

BP (mmHg) was measured in triplicate by the principal researcher using a manual sphygmomanometer (Baumanometer® Sphygmomanometer, W.A. Baum Co., Inc., New York) with at least three minutes separating each measurement. Participants were required to rest quietly in a seated position for at least five minutes prior to the measurement. The BP measurements were taken according to the auscultatory method (Joint National Committee, 2004) and by applying a sphygmomanometer cuff of appropriate size (cuff bladder encircling at least 80% of the arm) to the left brachial artery (Tortora and Derrickson, 2006; Sohn *et al*., 2007). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded at the first and fifth Korotkoff sounds respectively and then the two closest readings were averaged and recorded.

3.6.5. Physical Activity

3.6.5.1. STEP COUNT

Piezo-electric pedometers (NL-1000; New Lifestyles, Kansas City, Missouri, USA), with a seven-day memory function, moderate-to-vigorous physical activity (MVPA) time accumulation and not affected by pedometer tilt or adiposity level, were used to objectively measure physical activity. Participants were instructed on the procedures for wearing the pedometer, which included its being worn on the right hand side of the body, at the midline of the thigh, securely attached to a belt or waistband (Figure 8). Participants were told to wear the pedometer for the entire day beginning as soon as they woke up in the morning and to remove it only for sleeping, swimming and bathing purposes by unclipping. At the end of each day the pedometer was removed and the total step count and MVPA time displayed was recorded in individual step count diaries. These diaries were regularly checked by the principal researcher and then collected at monthly intervals to ensure the walking programme was being adhered to.

Figure 8: Piezo-electric pedometer (NL-1000)

3.6.5.2. FITNESS

The 6MWT has been found easier to administer, better tolerated and better reflects activities of daily living than other walk tests (Enright, 2003; Dourado, 2011). It is now widely used to assess functional exercise capacity and prognosis since it is inexpensive, quick, safe and corresponds to sub-maximal moderate exercise (Gremeaux *et al*., 2011). Furthermore the 6MWT has been found highly reproducible in obese participants and thus is recommended for use as a fitness indicator in clinical studies in these populations (Beriault *et al*., 2009).

Participants were instructed to wear appropriate clothing and shoes for this test and to perform five minutes of light stretching before commencing the walk. The procedure was as follows: a level outdoor grass field was used, where a 50-metre straight section was measured out and marked with cones. This 50 m section was then sub-divided into 10 m points marked out with cones which were placed along the side of the field. Participants were instructed to walk at a self-selected pace from one end of the field to the other and back in order to cover as much distance as they could during the allotted time (Gremeaux *et al*., 2011). The test was monitored and the time was called out every two minutes, with standard encouragement at 30-second intervals. Slowing down and stopping to rest were permitted. Participants were required to stop dead as soon as the six minute time was up so to allow total distance walked to be measured.

3.6.5.3. GLOBAL PHYSICAL ACTIVITY QUESTIONNAIRE (GPAQ)

The GPAQ was developed by the WHO, with the aim of having a tool that would produce valid and reliable estimates of physical activity especially relevant to developing countries where patterns of energy expenditure differ from those in developed countries (Armstrong and Bull, 2006). Once the GPAQ had undergone validation studies and qualitative feedback, the final GPAQ version 2 instrument (GPAQv2) was developed, which is a suitable physical activity surveillance instrument in developing countries (Armstrong and Bull, 2006). The GPAQv2 comprises 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 (high, moderate and low); and a continuous variable of total physical activity within each domain (reported as median METmins.week^{-1}) (Armstrong and Bull, 2006) (Appendix 9). For this study, participants were required to have ‘low’ total physical activity to begin with, which can be equated to <600 METmins.week^{-1} (Bergman *et al*., 2009).

3.6.6. Biochemical

A blood test, whereby a blood sample is drawn from the participant, represents the most accurate means of assessment for both type II diabetes and hypercholesterolemia (Du Plessis *et al*., 2000). Blood tests for the full blood lipid profile were conducted at baseline, and repeated every four weeks throughout the 12 week protocol. Blood tests for glycated haemoglobin were performed only pre- and post- the 12 week walking intervention. The initial tests functioned as a screening tool, and individuals presenting with diabetic conditions and/or hypercholesterolemia were excluded from the EXC and CON cohorts. During the exercise intervention, these measures acted as controlled variables.

3.6.6.1. HYPERCHOLESTEROLEMIA

See Appendix 16 for a comprehensive methodology on the collection and analysis of samples for total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C).

3.6.6.2. TYPE II DIABETES

See Appendix 17 for a comprehensive methodology on the collection and analysis of glycated haemoglobin (HbA_{1c}).

3.6.7. Behavioural

3.6.7.1. ALCOHOL USE (CAGE QUESTIONNAIRE)

The CAGE questionnaire is composed of four questions and is known to serve as a screening tool for possible alcohol dependence (Appendix 8). CAGE is an acronym representing “Cutting down”, “Annoyance by criticism”, “Guilty feeling” and “Eye-openers”. The CAGE questionnaire was used in the 2003 SADHS and therefore has been validated not only nationally, but within different population groups in the

Eastern Cape. Individuals who answered positively to two or more questions were classified as being alcohol dependent and were therefore excluded from the intervention part of the study (Schneider *et al*., 2007).

3.6.7.2. TOBACCO USE

Tobacco use was determined in a simple questionnaire asking participants if they currently smoked or have smoked in the previous 12 months.

3.6.8. Other

3.6.8.1. MEDICATION QUESTIONNAIRE

The medication questionnaire used in the current study was adapted from one used in the 2003 SADHS and has therefore been validated within different population groups in the Eastern Cape. The aim of the questionnaire in this study was to determine the specific type and amount of medication used both at the time of the study and on a regular basis. The difference between the two questionnaires was that the one used in the current study omitted the 'payment for medication' section as this was of no interest.

3.6.8.2. DIETARY RECALL

Participants were asked to maintain their habitual diets for the duration of the study. To monitor food intake each participant completed a three day (Tuesday, Thursday and Saturday) dietary recall at baseline and every four weeks thereafter. Instruction on how to complete the dietary recall form was given at the commencement of the study by the principal researcher. Dietary intake and macronutrient composition for each participant was analysed using the Medical Research Council (MRC) FoodFinder 3™ software programme for Windows® (Mircrosoft Corporation). This contains the latest version of the South African Food Composition Database (SAFOODS) which is the official database on the nutrient composition of foods eaten

in South Africa. The data recorded were entered into the programme, following which dietary intake was analysed and total daily energy (kcal), protein (g), carbohydrate (g) and fats (g) intake was documented.

3.7. EXERCISE INTERVENTION

This sub-study engaged a pedometer-based progressive walking intervention on at least five days of the week for 12 weeks (Table 14). Participants from the EXC group were instructed to walk at a moderate intensity, which was demonstrated during the 6MWT as well as having the pedometer record MVPA time accumulation to ensure the correct intensity was being used. For the current study it was important to link the walking intensity required to a time duration which allowed the participants to easily monitor their own walking pace. In the final 9-12 weeks of the intervention, participants were walking an extra 3000 steps.day^{-1} or performing an extra 30 minutes of physical activity.

Table 14: Design of EXC walking programme

WEEK (INCLUSIVE)	EXTRA STEPS.DAY^{-1}	EXTRA TIME.DAY^{-1}	DAYS.WEEK^{-1}
0	0	0	0
1-4	1000	10 minutes	≥5
5-8	2000	20 minutes	≥5
9-12	3000	30 minutes	≥5

This walking programme was completed unsupervised and in the participants' own locality and time. The EXC group members were motivated and held accountable through individual step goals which were measured and recorded using piezo-electric pedometers and step count diaries. The 'daily' aspect of this study was stressed to ensure participants wrote down their step count at the end of each day, allowing for accurate monitoring of daily walking. Due to the characteristics of the sample population, specifically the exercise inexperience and high BMIs, it was decided that a progressive intervention which slowly increased extra steps per day

would be the most feasible technique. It should also be noted that the participants were told that the extra step counts could be accumulated via one ‘long-bout’ of walking or several ‘short-bouts’ of walking of at least ten minutes..

A walking intervention was selected because this type of exercise is seen as a lifestyle activity. For this research to be plausible participants walked without supervision and this may have resulted in both inter- and intra-individual variability due to environmental conditions (weather, terrain and gradient). Other authors conducting similar walking research on CVD risk do not address this issue at all (Moreau *et al*., 2001; Tully *et al*., 2005; Sohn *et al*., 2007; Tully *et al*., 2007). In an effort to focus on research with high compliance as well as an intervention with long-term sustainability, the current study advised participants about these environmental factors and acknowledged this as a limitation of an unsupervised programme.

3.8. EXPERIMENTAL PROCEDURES

3.8.1. Phase 1 – Recruitment

The initial stage of the research involved recruiting willing and able participants who fulfilled the basic requirements for inclusion. This process began in February 2013 and involved contacting the Rhodes University catering and housekeeping managers with a proposal for the study. This was followed up with a formal meeting at which the background to the research and the requirements of the participants was explained in detail. Permission to approach the staffs’ supervisors was given. The principal researcher then held two separate meetings with the catering and housekeeping supervisors to explain the study and encourage them to get their staff involved. Recruitment posters and pamphlets where given to the supervisors who were instructed to distribute these to their staff to allow them to read about the study before the next meeting. Four further meetings were then held, one with the housekeeping staff and three with the catering staff (who were split into different halls). The study was very briefly explained and interested individuals signed up with their names and contact numbers to be included in the pre-screening phase.

3.8.2. Phase 2 – Baseline Measures

Four separate pre-screening sessions were held, one for the potential CON group participants and three for the potential EXC group participants. Participants were allocated to one of these groups based on their working schedule and therefore available times. Each participant was then contacted via a phone call/text message and invited to attend one of these sessions. They were instructed to not eat or drink anything for at least two hours before the session commenced.

At the start of the group-specific session a detailed introductory meeting was held and the walking programme and research in general was carefully explained, accompanied by information letters. An interpreter (English/isiXhosa) was present at these sessions to assist with any communication issues. Questions were encouraged to ensure that all participants were fully aware of what would be expected of them throughout the 12 week study duration, as well as during the monthly testing sessions. Written consent forms where signed by those individuals interested in taking part in the study.

The individuals were then required to complete a ‘Participant Screening’ form which formed the initial stages of screening. The variables included in this form were sex, race, age, language, health status, physical activity, medication usage, presence of menstruation, and smoking and drinking habits. A research assistant was available to help individuals with this process and to clarify any questions that were not fully understood.

The next three stations involved having stature, body mass, waist circumference, fat mass and lean mass, heart rate and BP measured. These measures allowed for further screening of the inclusion criteria and served as baseline measures for data analyses. Stature was recorded only at baseline as this measure was not expected to change throughout the course of the study.

The next station involved the completion of three questionnaires. A research assistant administered these questionnaires to avoid any misunderstandings and therefore inaccurate data.

Following this, each participant underwent a physical examination by a specialist physician to make sure each individual was fit to safely participant in an exercise intervention of this nature.

At the next station each participant was given a specific day and time to visit 'Du Buisson & Partners', in Peppergrove Mall, where blood work was done in a fasted state to measure glycated haemoglobin and full blood lipid profile levels.

The next station involved one-on-one time with the principal researcher. Each participant was given a folder with their participant code, step count diary and dietary recall forms. Detailed instruction was given on how to complete the three day dietary recall form which was required each month. Each participant was then given an individual pedometer following careful instruction on how to operate the device, as well as what information was to be recorded in the daily diary. Stride length was measured with a standard dressmaker's tape measure and these data were put into the pedometer to provide the most accurate results. Participants were required to place the pedometer on their belt/waistband to check that the device was being positioned correctly and to ensure that each participant was comfortable with putting on the pedometer and removing it themselves. Both groups were to record total daily steps according to the pedometer display screen. Participants in the CON group were instructed not to alter their lifestyle and to continue with their lives as normally as possible. Participants in the EXC group were instructed to walk unsupervised at a moderate-intensity for at least five days of the week for the 12 week study duration. The number of extra steps to be taken was individually-specific based on their baseline measures and increased by 1000 steps.day^{-1} at monthly intervals. This was explained to them and also illustrated in their diaries to ensure they were following the programme correctly.

The whole group was then taken to a grass field next to the building to perform the 6MWT. This fitness test used a stopwatch and a 50 m flat section sub-divided into 10 m segments which had been measured out and marked with large orange cones. Participants were instructed to walk at a self-selected pace up and down the 50 m field as many times as possible within six minutes.

The fitness test marked the end of the pre-screening session. Participants took home a pedometer and wore it for one week without altering their normal physical activity or lifestyle, to determine baseline step counts. The pedometer was to be worn for the entire day, and to be removed only for bathing and sleeping. At the end of each day the individual was to record the date, their total daily step count (displayed on the pedometer screen) and MVPA time accumulation.

At the end of this habituation week a meeting was held to collect the dietary recall forms and step count diaries. At this point all the data from the pre-screening sessions had been analysed. Only those individuals fulfilling the following criteria were invited to be a part of the intervention study for the full 12 week duration: female; black African; able to read and write English; mild/moderate hypertension; no diabetes, hypercholesterolemia or major surgery in the past six months; no prior or current heart, lung, liver or kidney disease; non-smokers; not heavy drinkers; no musculoskeletal injury; not currently using or having used antihypertensive medication in the past month; not pregnant; and not using drugs or other agents that may affect BP. The remaining individuals were informed of their failure to meet the standards for the intervention part of the study and thanked for their participation in the baseline measurements.

3.8.3. Phase 3 – Exercise and Control Interventions

Each month a meeting was held separately for the EXC and CON groups to keep the groups isolated. All the meetings, however, involved the same process, whereby step count diaries were brought to the meeting so that the principal researcher could

check them and ensure the programme was being adhered to and the data were being filled in appropriately. Dietary recall forms were given out to be completed and participants were given the chance to talk to each other and to the principal researcher and research assistants and ask questions. Following this, data collection occurred. Anthropometric, morphological, cardiovascular, physical activity and control variables were measured for each participant. At the end of each session the participants were given a specific date and time to visit 'Du Buisson's & Partners' to have their routine blood test done in a fasting state. These formal meetings were held monthly as it marked the transition from one timeframe to the next (for example from week 1-4 to week 5-8) of the walking programme. However, several informal interactions took place in the interim to provide motivation and knowledge to all participants.

At the end of the 12 week duration all the data from the baseline measurements, EXC and CON groups were collected, analysed and interpreted. Results were formed for each individual and were presented as feedback to the participants, along with a small thank-you gift. Participants were given the opportunity to ask questions and seek advice at this feedback session.

3.9. DATA ANALYSES

Statistical analyses were conducted using STATISTICA© (Version 11.0). Descriptive statistics were initially conducted to obtain mean and standard error values of all response variables. Further, each response variable was checked for normality using the Shapiro-Wilk test with a level of significance at 5%, and Levene's test for homogeneity of variances was performed.

Comparisons were then made between the baseline data and the existing norms using a single sample t-test. Following this, the dependent variables were analysed using a two lists correlation matrix to see if any significant relationships existed which were expressed when $p<.05$.

Using the EXC and CON group data, comparisons were made between each response variable over the 12 weeks of the walking programme, using repeated measures ANOVA with the response variable at each month as the repeated measure and group as a factor. The hypotheses were tested using F statistics and level of significance at 5%. When a significant difference was identified a Tukeys Post-Hoc analysis test was conducted to identify where the difference(s) were. Additionally with the EXC group, for those variables which demonstrated a trend with time, but were not statistically significant – possibly due to the small sample size – a simple correlation was used to identify any worthwhile relationships.

CHAPTER 4

RESULTS

4.1. COMPARATIVE STUDY

The current study was compared against the 2003 South African Demographic and Health Survey (SADHS) and the 2013 South African National Health and Nutrition Examination Survey (SANHANES-1). The latter does not always differentiate between race and sex, making certain variables difficult to compare hence the need to use the SADHS which provides information on black African females specifically. It is important to note that the 2003 SADHS had issues with their blood pressure (BP) data collection in the field and so results are reported as inaccurate. Where there is no South African specific data, general guidelines are applied. The SANHANES-1 does not report on standard error (SE) values and so this information is missing. All significant differences were determined at $p \leq .05$. The '*' on the figures denotes this significant difference, but, in some instances where the differences are intricate, the significant changes are not shown on the figure but rather highlighted in a subsequent table. See Appendix 18 (page 210) for detailed statistical analyses.

4.1.1. Risk 1: Overweight/Obesity

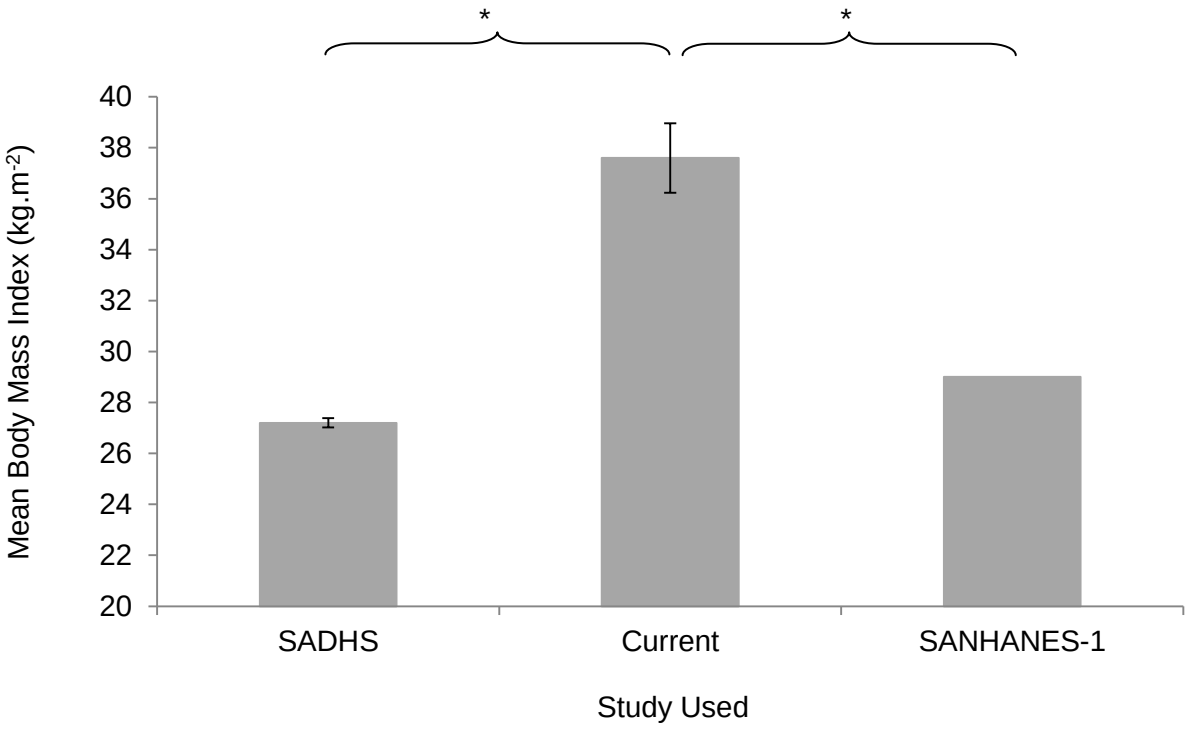

Figure 9: Comparison of mean (±SE) body mass index ($kg.m^{-2}$)

At a significance of $p<0.0001$, black African females in the current study were found to be heavier than their population-matched group from both the SADHS and the SANHANES-1 (Figure 9). The average body mass index (BMI) found in the current sample was 37.6 $kg.m^{-2}$ (±8.64) which, according to the World Health Organisation (WHO), is classified as 'obese class II' and associated with 'severe risk of co-morbidities'.

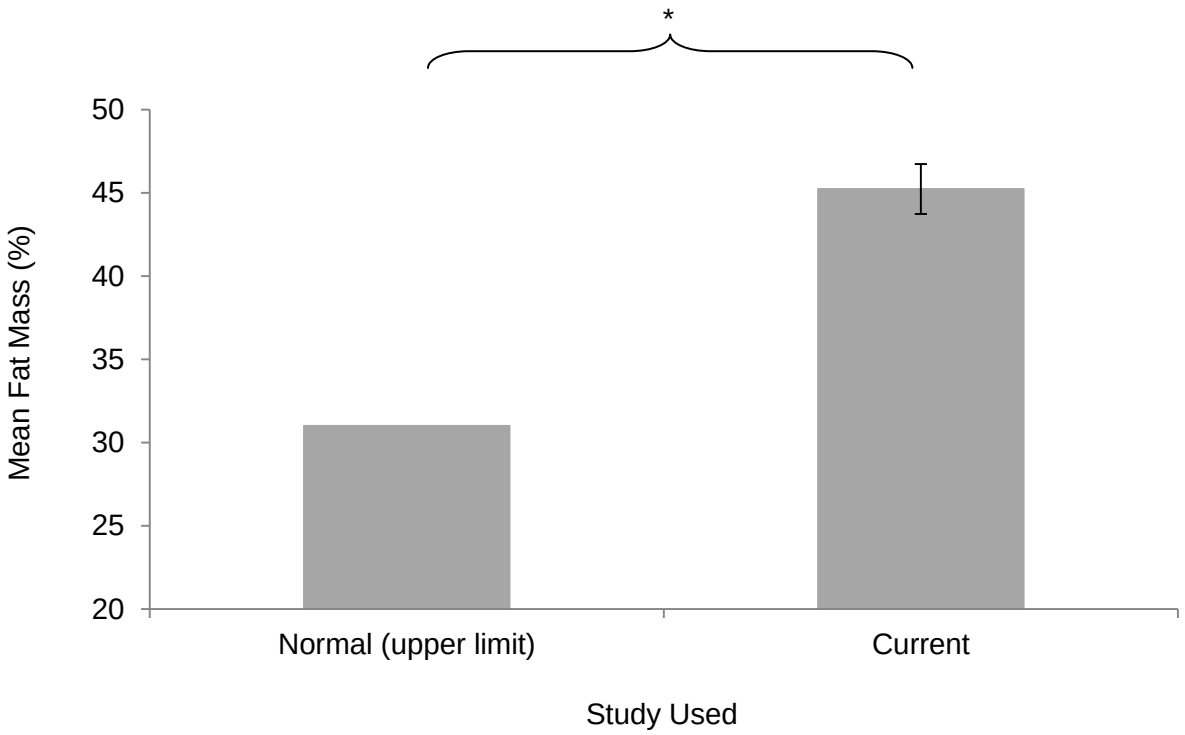

Figure 10: Comparison of mean (±SE) fat mass (%)

With respect to fat mass, there was a significant difference ($p<0.0001$) between the upper limit of the general guidelines and the results from the current study (Figure 10). The current study result of a mean fat mass of 45.23% (±9.47) is well above the normal upper limit (31%) and, for females, is classified as 'obese'.

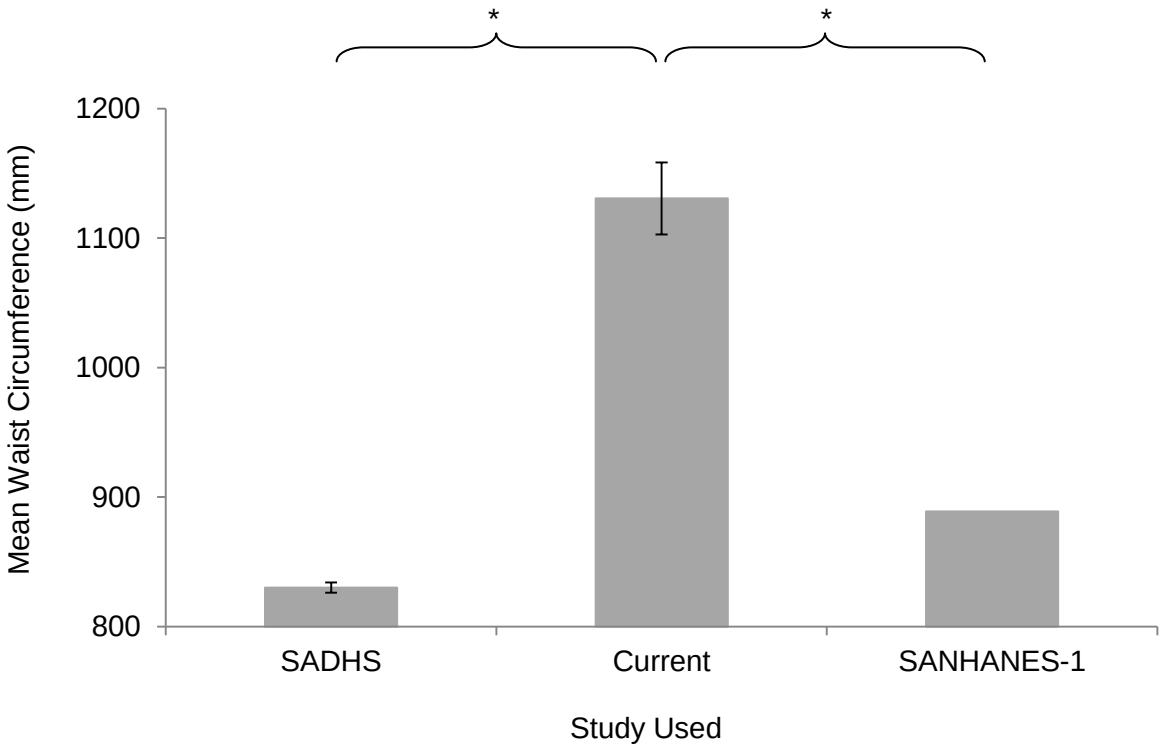

Figure 11: Comparison of mean (±SE) waist circumference (mm)

Figure 11 illustrates the significant differences ($p<0.0001$) when comparing both the SADHS and the SANHANES-1 to the current study for waist circumference (WC). The average WC was greater, with a value of 1130.58 mm (±176.29), which, according to the WHO, is a risk classification of 'above action level 2'.

4.1.2. Risk 2: High Blood Pressure

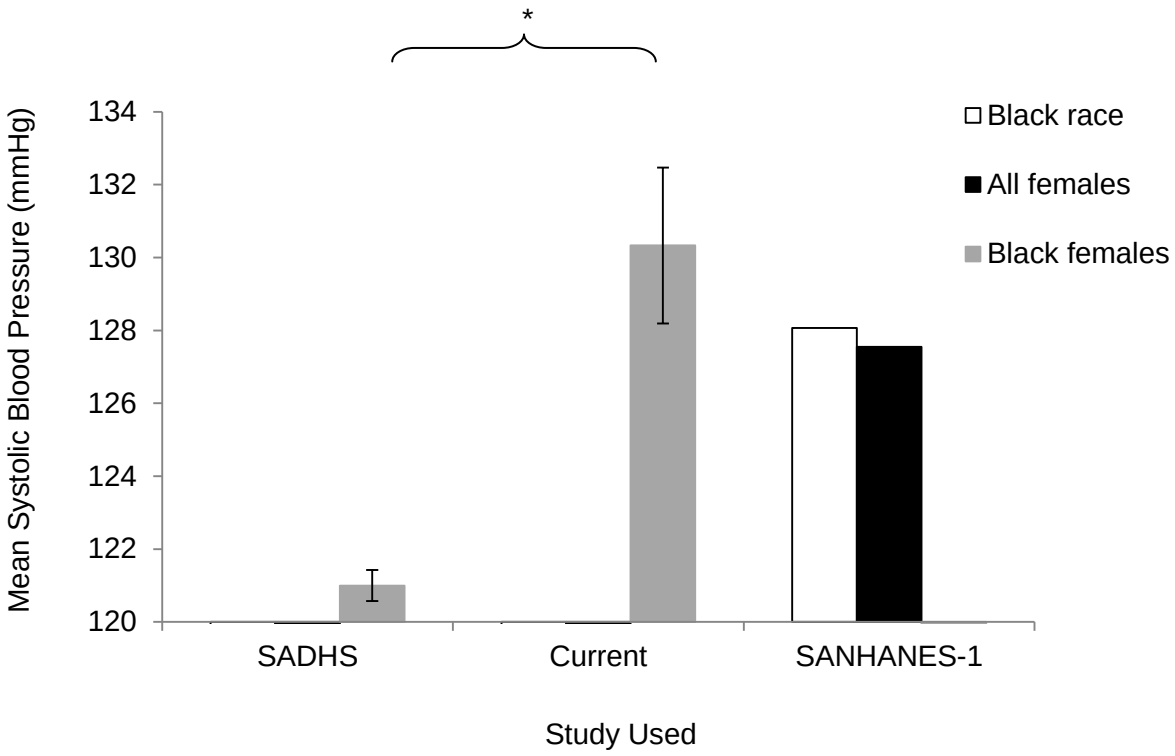

Figure 12: Comparison of mean (±SE) systolic blood pressure (mmHg)

At a significance level of p=0.0001, the mean systolic blood pressure (SBP) for the current sample (130.33 ±13.56 mmHg) was higher than that found in the SADHS (121 mmHg) (Figure 12). No differences were found between the SANHANES-1 'black race' and 'all female' group data and the current study.

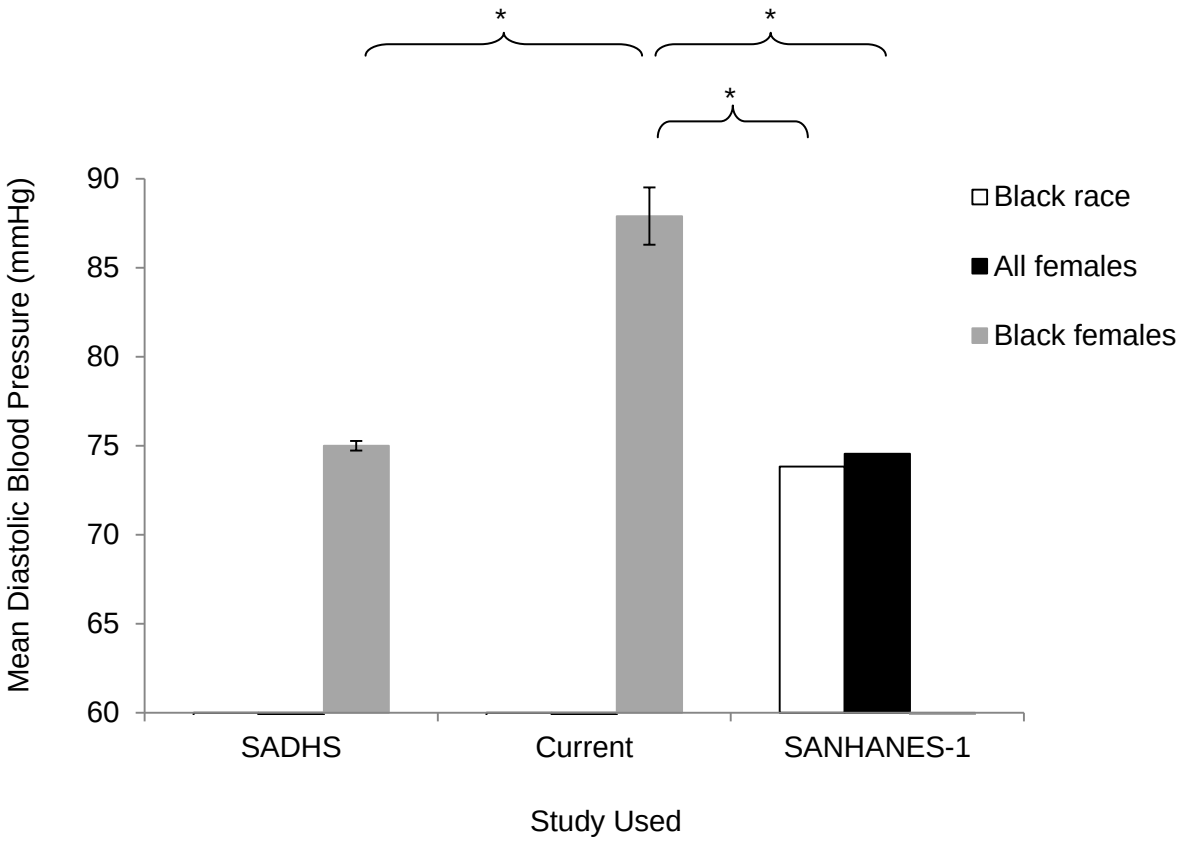

Figure 13: Comparison of mean (±SE) diastolic blood pressure (mmHg)

The current results for diastolic blood pressure (DBP) showed a significantly greater ($p<0.0001$) mean value compared to both previous national surveys (regardless of group) (Figure 13). The mean result of 87.90 mmHg (±10.21) in the current study is paired with the SBP result and thus indicates pre-hypertension.

4.1.3. Risk 3: Physical Inactivity

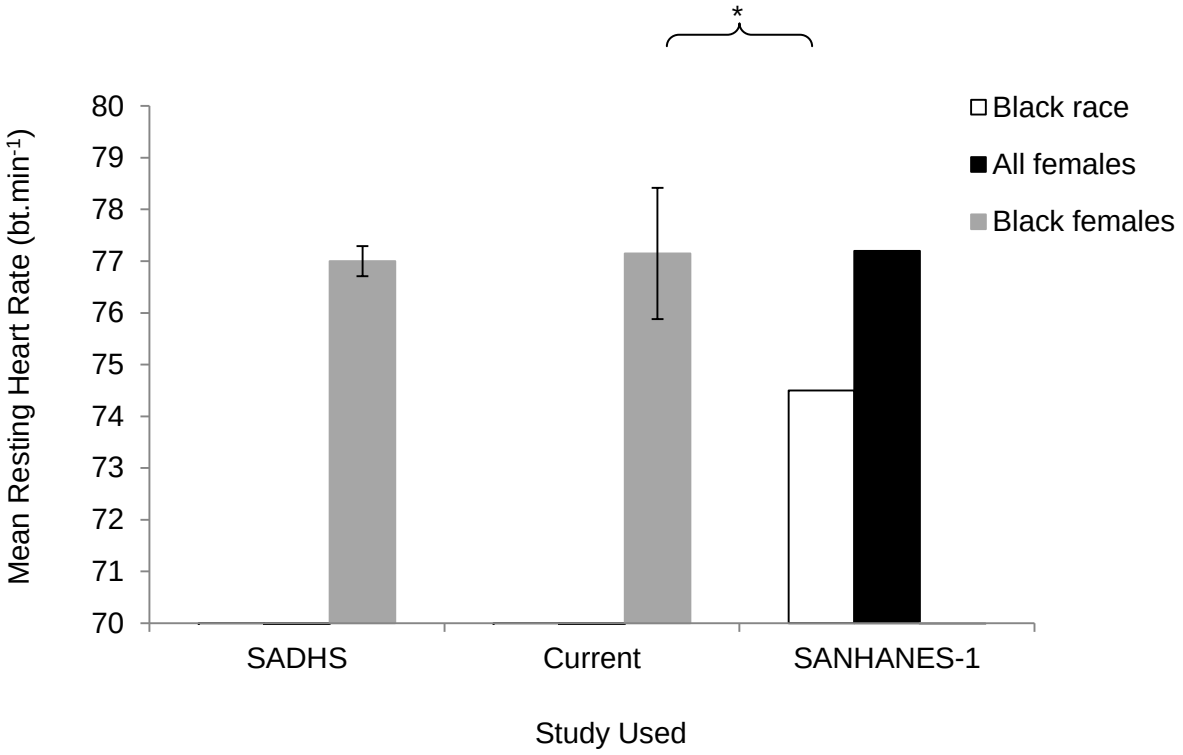

Figure 14: Comparison of mean (±SE) resting heart rate (bt.min^{-1})

Referring to Figure 14 it can be seen that, on average, a significantly higher (p=0.0441) resting heart rate was found in the current study compared to the SANHANES-1 'black race' group. However, clinically, the average resting heart rate of 77 bt.min^{-1} (±8) is considered to be normal. Also, no significant differences were identified between the population-matched group from the SADHS and the 'all female' group from the SANHANES-1.

The clinical mean daily step count of 10162 steps.day^{-1} (±3090) places this cohort into the 'active' category according to accepted guidelines (as highlighted in Table 15). The large variance in this response shows that some participants fell into the lower 'somewhat active' group while others were classified as 'highly active'. Previous literature by Chan and Tudor-Locke (2008) says that it is difficult to get

people with initially high levels of steps per day to increase this activity further, which is consistent with later findings.

Table 15: Comparison of daily steps (steps.day^{-1}) (Tudor-Locke and Bassett, 2004)

GUIDELINE STEPS.DAY^{-1} (WHOLE POPULATION)	CATEGORY	CURRENT STUDY MEAN STEPS.DAY^{-1}
<5000	Sedentary	
5000-7499	Low Active	
7500-9999	Somewhat Active	
10000-12499	Active	10162
≥12500	Highly Active	

At baseline, the moderate-to-vigorous physical activity (MVPA) time accumulation was an average of 19.42 mins.day^{-1} (±13.02). This is below the recommended 30 mins.day^{-1} which shows that, although the participants were classified as 'active' according to the step guidelines (Table 15), they were not habitually walking at an intensity necessary to illicit health benefits. Only baseline MVPA data is reported on as during this intervention this variable was not accurately provided by the participants and the pedometer memory did not bank this information long enough for the researcher to access it.

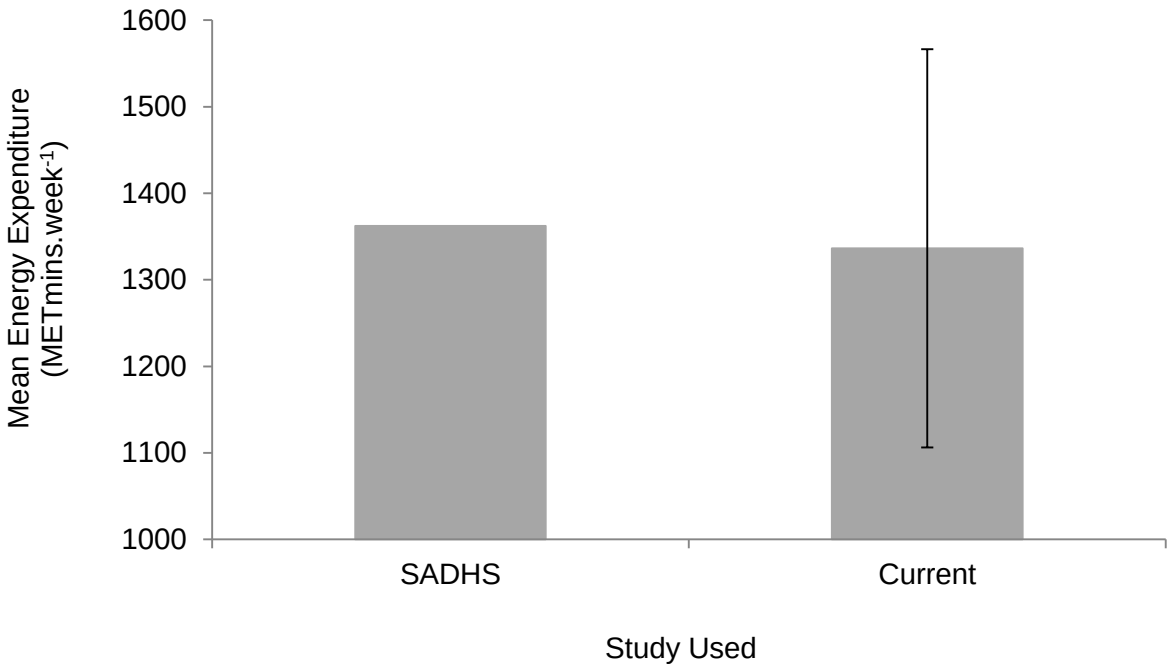

Figure 15: Comparison of mean (±SE) energy expenditure (METmins.week^{-1})

No significant differences existed for weekly energy expenditure between the SADHS (1362.33 METmins.week^{-1}) and the current study (1336.4 METmins.week^{-1}) (Figure 15). Noteworthy is the large standard error in the current study (±1455.66 METmins.week^{-1}) which represents the broad physical activity range of the participants at baseline.

4.1.4. Risk 4 and 5: High Cholesterol and Type II Diabetes

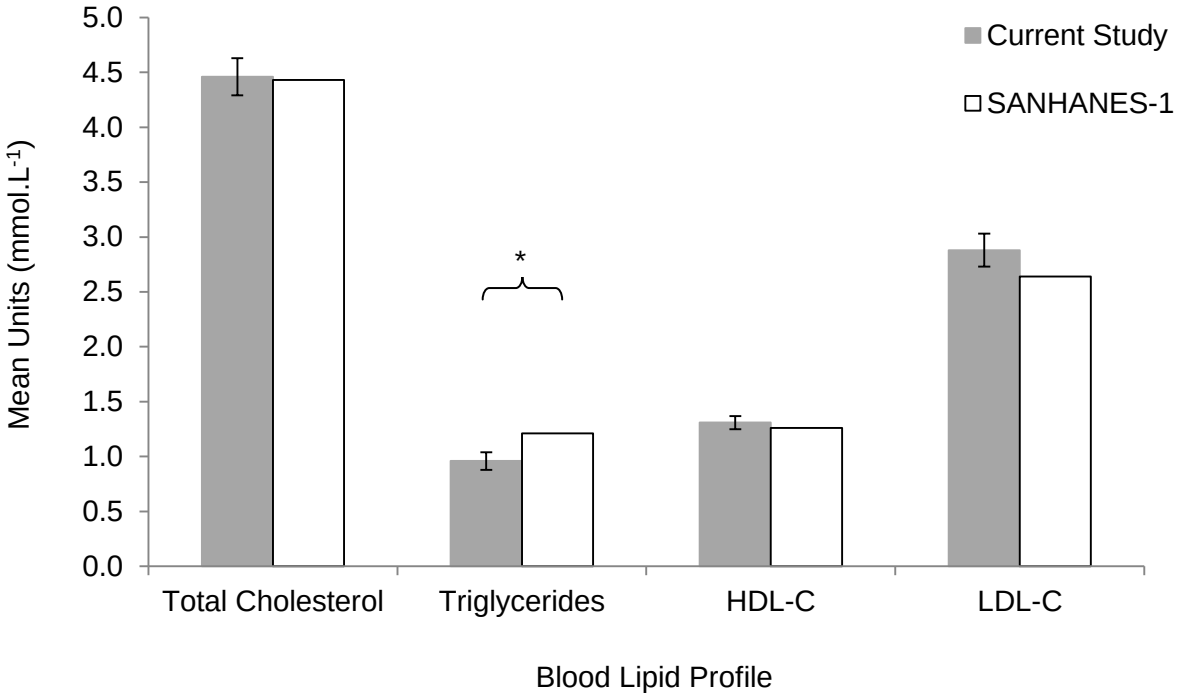

Figure 16: Comparison of mean (±SE) lipid profile values (mmol.L^{-1})

All of the lipid profile components were classified as 'low risk' except for high-density lipoprotein cholesterol (HDL-C) which fell into the 'borderline high risk' group. A significant difference (p=0.0037) was found only with triglycerides (TGs) between the SANHANES-1 and the current study, with the current study revealing a lower and therefore more beneficial result (0.96 mmol.L^{-1} compared to 1.21 mmol.L^{-1}) (Figure 16).

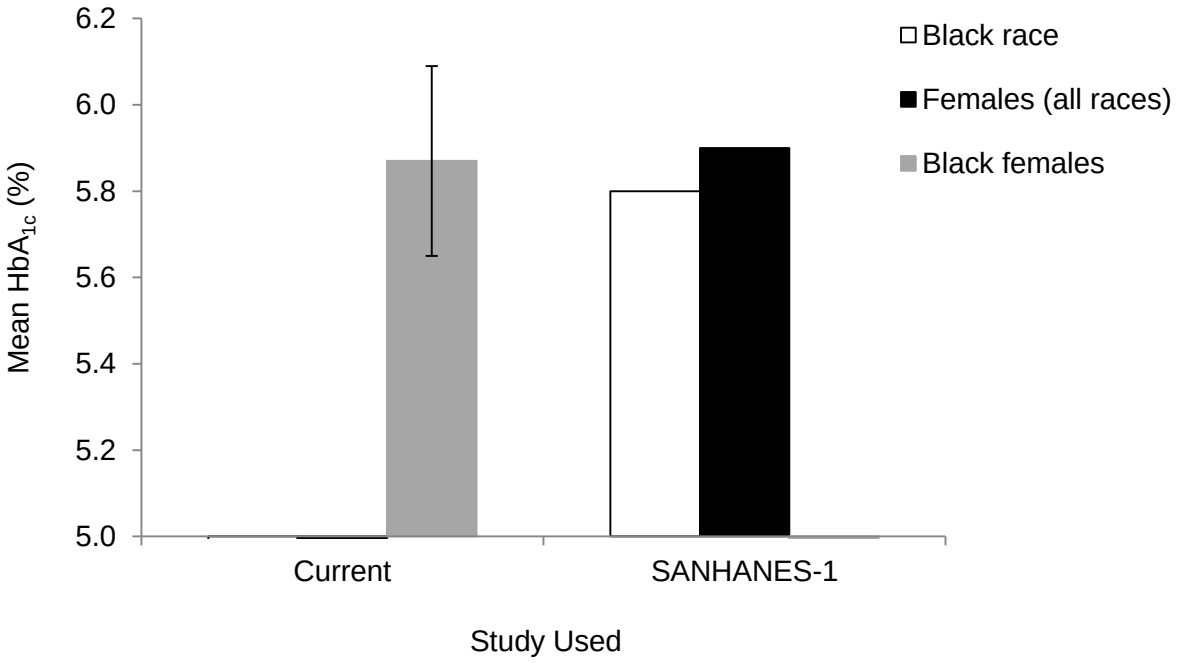

Figure 17: Comparison of mean (±SE) glycated haemoglobin (HbA_{1c}) values (%)

There were no significant differences in glycated haemoglobin levels (Figure 17). However, mean results from both the SANHANES-1 (5.8% in black race and 5.9% in female group) and the current study (5.87%) indicate an 'increased risk of diabetes (pre-diabetes)' in the population groups tested.

4.1.5. Risk 6: Tobacco Use

The smoking statistics in the current study were positive with 0% reportedly being 'non-smokers' defined as not currently a smoker or having smoked in the previous 12 months. This is compared to the most recent national survey indicating a prevalence of 4.8% in black African females (Table 16).

Table 16: Comparison of smoking prevalence (%)

STUDY	SMOKERS (%)
SANHANES-1	4.8
Current	0

4.1.6. Risk 7: Alcohol Abuse

The alcohol abuse statistics were also positive as only 5% of the cohort was classified as being 'heavy drinkers' based on the CAGE questionnaire, compared to the national 6.6% prevalence reported in the SADHS (Table 17).

Table 17: Comparison of drinking rates (%)

STUDY	HEAVY DRINKERS BASED ON CAGE QUESTIONNAIRE (%)
SADHS	6.6
Current	5

4.2. BASELINE CORRELATIONS

Using the baseline data, BMI demonstrated a significant and very strong, positive relationship with fat mass (p=0.000, r=0.8967) and WC (p=0.00, r=0.9522) and thus, WC was also significantly and very strongly associated with fat mass (p=0.000, r=0.8701).

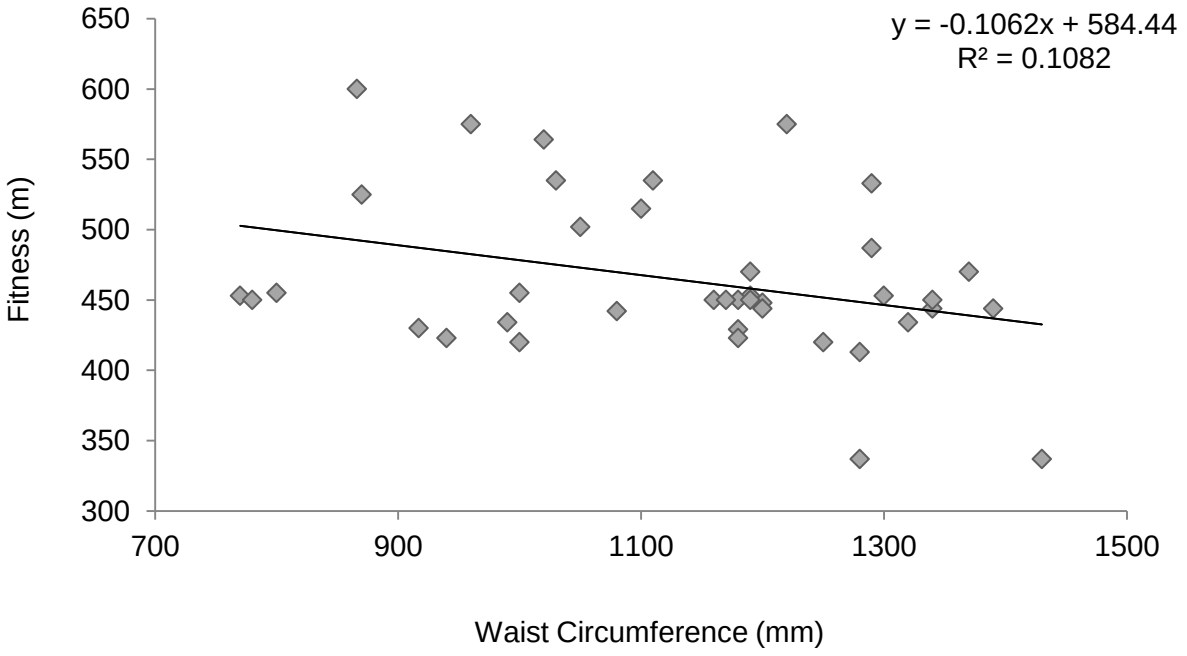

Figure 18: Negative correlation between waist circumference (mm) and fitness (m)

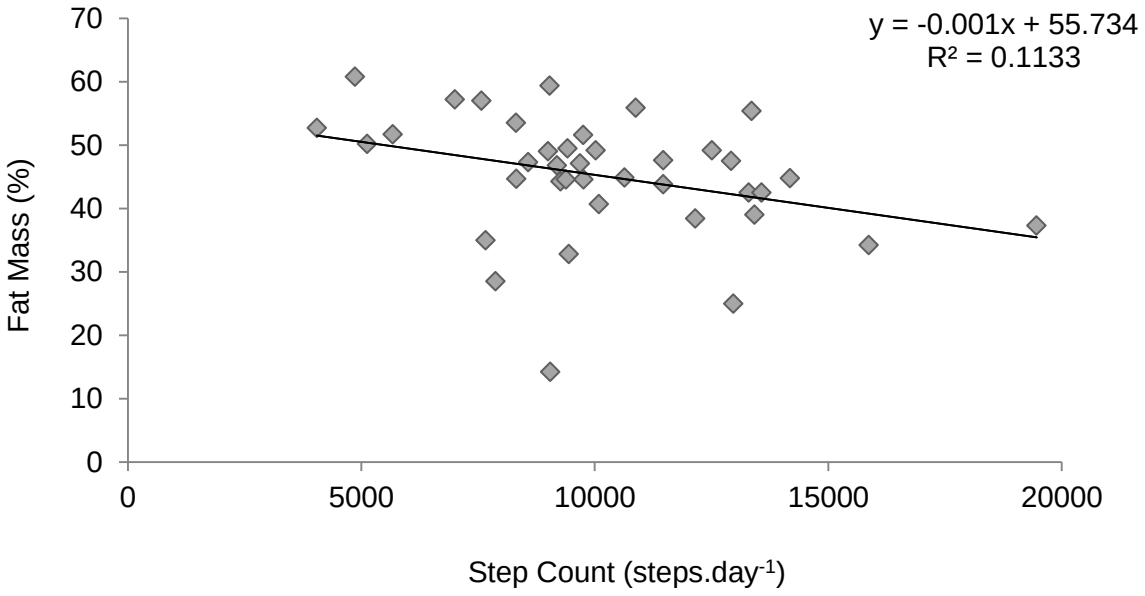

Figure 19: Negative correlation between step count (steps.day^{-1}) and fat mass (%)

There was a significant, weak, negative relationship between WC and fitness (p=0.038, r=-0.3289) (Figure 18), and step count and fat mass (p=0.036, r=-0.3366) (Figure 19). These relationships justify the need for increased physical activity in overweight/obese populations.

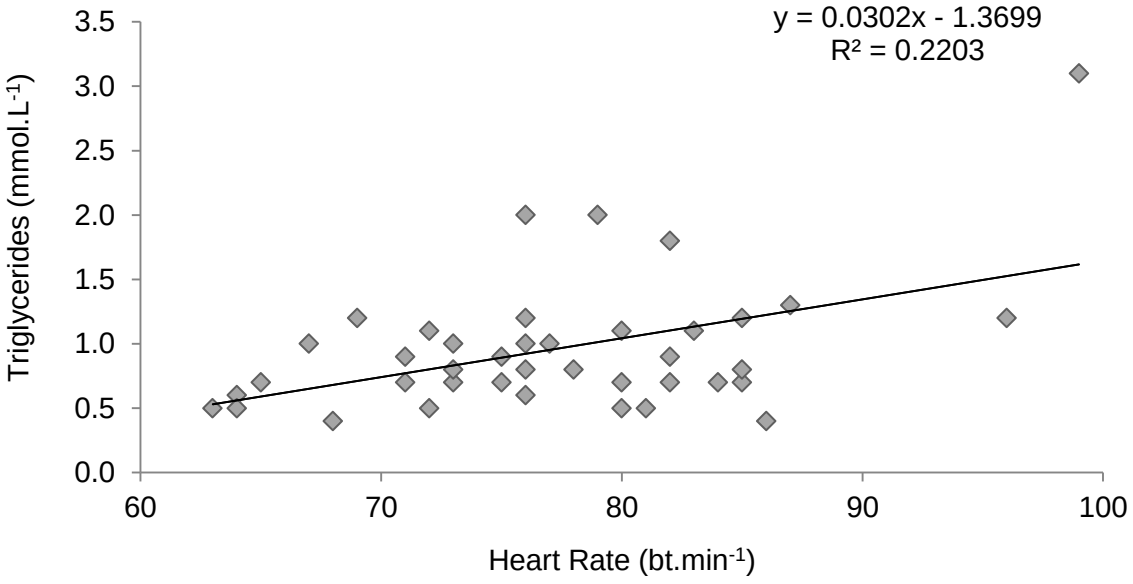

Figure 20: Positive correlation between heart rate (bt.min^{-1}) and triglycerides (mmol.L^{-1})

A significant, moderate, positive relationship was observed between heart rate and TG levels (p=0.002, r=0.4693) (Figure 20).

4.3. INTERVENTION STUDY

4.3.1. Group Results

The walking programme was poorly adhered to in terms of reaching both individual step goals and walking intensity (Table 18) which may, in part, be due to the participants already being classified as 'active' at the start of the programme. Only 15.4% of the participants met the recommended step goal on five days of the week, and 23.1% met their step goal on three days of the week. Adherence to the walking intensity was 0% and, as such, was a limitation.

Table 18: Step goal and walking intensity adherence

	100% ADHERANCE (5 DAYS.WEEK^{-1})		60% ADHERANCE (3 DAYS.WEEK^{-1})	
	Step Goal	Walking Intensity	Step Goal	Walking Intensity
NUMBER OF PARTICIPANTS	2	0	3	0

Although the programme was not well adhered to, the EXC group, on average, did increase their physical activity. Based on these results and observations of the researcher, it was decided that monthly average step count values would be used for analysis and, if significant group differences existed, the response variables would be further analysed.

4.3.1.1. STEP COUNT

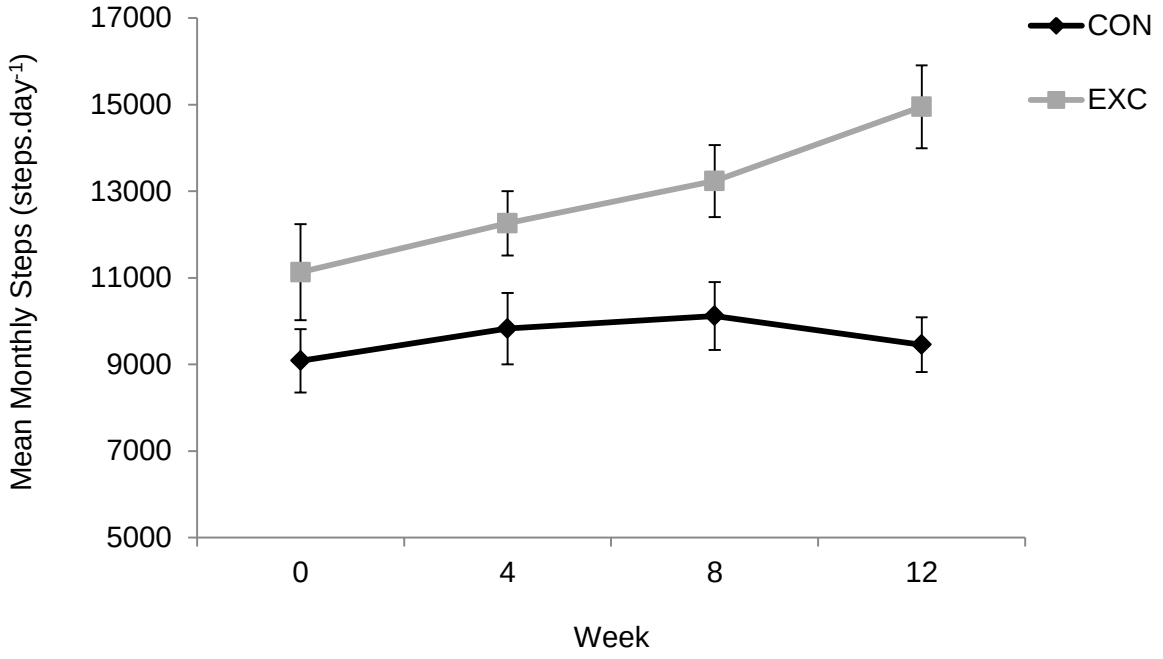

Figure 21: Mean (±SE) monthly steps (steps.day^{-1}) over the 12 week study

The EXC group had a higher mean step count at each time interval compared to the CON group. As expected, the EXC group step counts increased progressively over the 12 weeks. These were significant with respect to group effect (F=11.24, df=(1, 23), p=0.0028), time effect (F=5.09, df=(3, 69), p=0.0031) and interaction effect (F=3.74, df=(3, 69), p=0.0150) (Figure 21). The significant interaction effects are highlighted in Table 19.

Table 19: Significant interaction effects for the step count data

	CON 0	CON 4	CON 8	CON 12	EXC 0	EXC 4	EXC 8	EXC 12
CON 0								
CON 4								
CON 8								
CON 12								
EXC 0								
EXC 4								
EXC 8								
EXC 12								

4.3.1.2. ENERGY EXPENDITURE

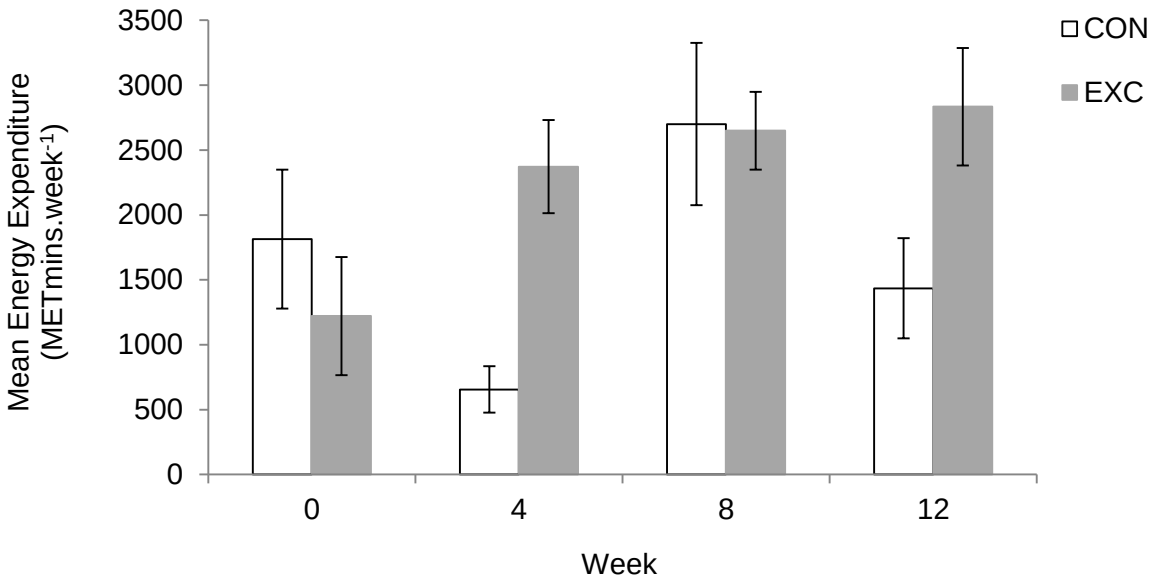

Figure 22: Mean (±SE) energy expenditure (METmins.week^{-1}) over the 12 week study

There was a significant time effect (F=5.19, df=(3, 69), p=0.0027) and interaction effect (F=5.17, df=(3, 69), p=0.0028) for energy expenditure (Figure 22). There was a general trend for increasing energy expenditure in the EXC group which complements the monthly increments in step goals, with a significant increase

(p=0.0267) between the starting and ending mean energy expenditure. There was a significant decrease (p=0.0029) in the energy expenditure of the CON group, between week 4 and week 8. The interaction effects, which were significant, are highlighted in Table 20.

Table 20: Significant interaction effects for the energy expenditure data

	CON 0	CON 4	CON 8	CON 12	EXC 0	EXC 4	EXC 8	EXC 12
CON 0								
CON 4								
CON 8								
CON 12								
EXC 0								
EXC 4								
EXC 8								
EXC 12								

4.3.1.3. DIET

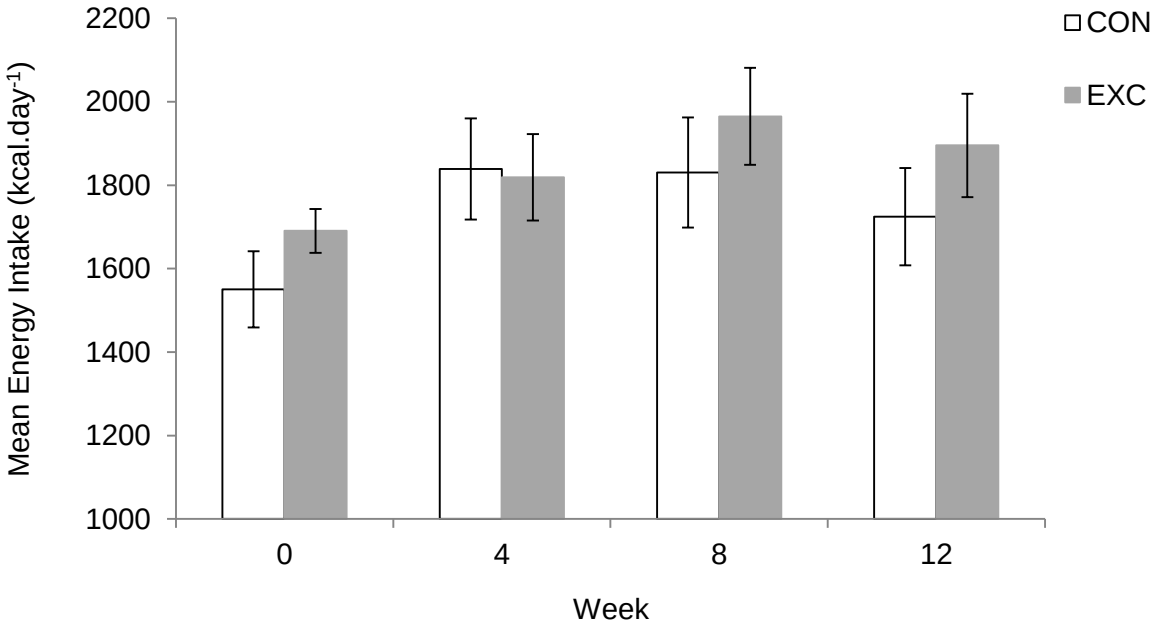

Figure 23: Mean (±SE) energy intake (kcal.day^{-1}) over the 12 week study

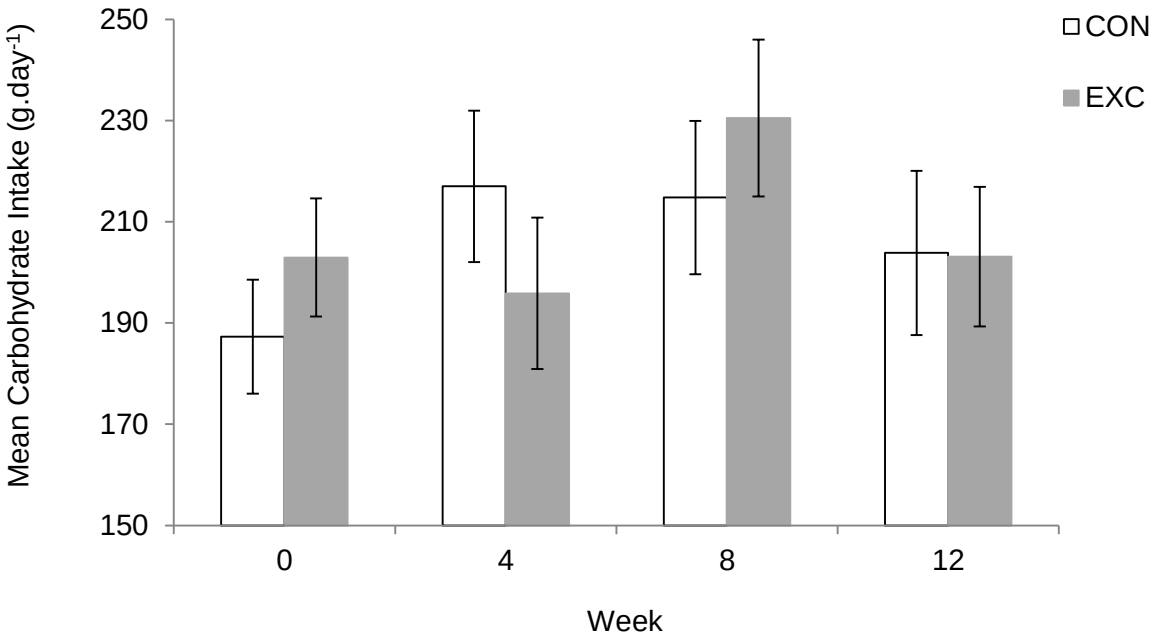

Figure 24: Mean (±SE) carbohydrate intake (g.day^{-1}) over the 12 week study

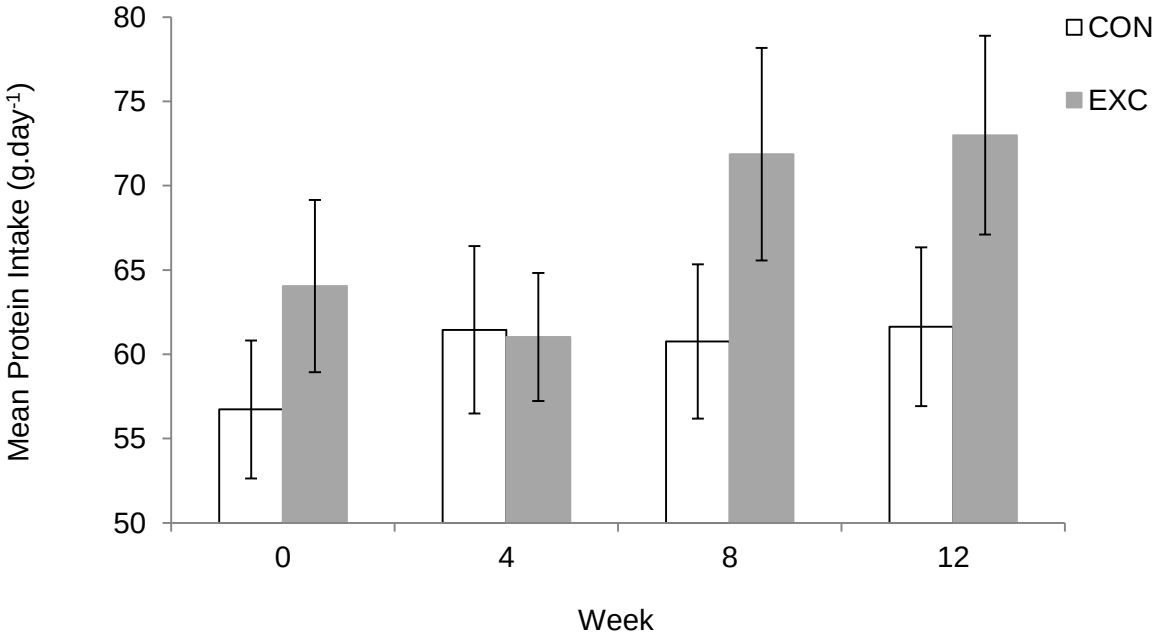

Figure 25: Mean (±SE) protein intake (g.day^{-1}) over the 12 week study

It was important that participants did not alter their 'normal' dietary habits so as to isolate the effects of exercise alone. This criterion was adhered to as there were no

significant differences present in mean total energy intake (Figure 23), carbohydrate intake (Figure 24) and protein intake (Figure 25).

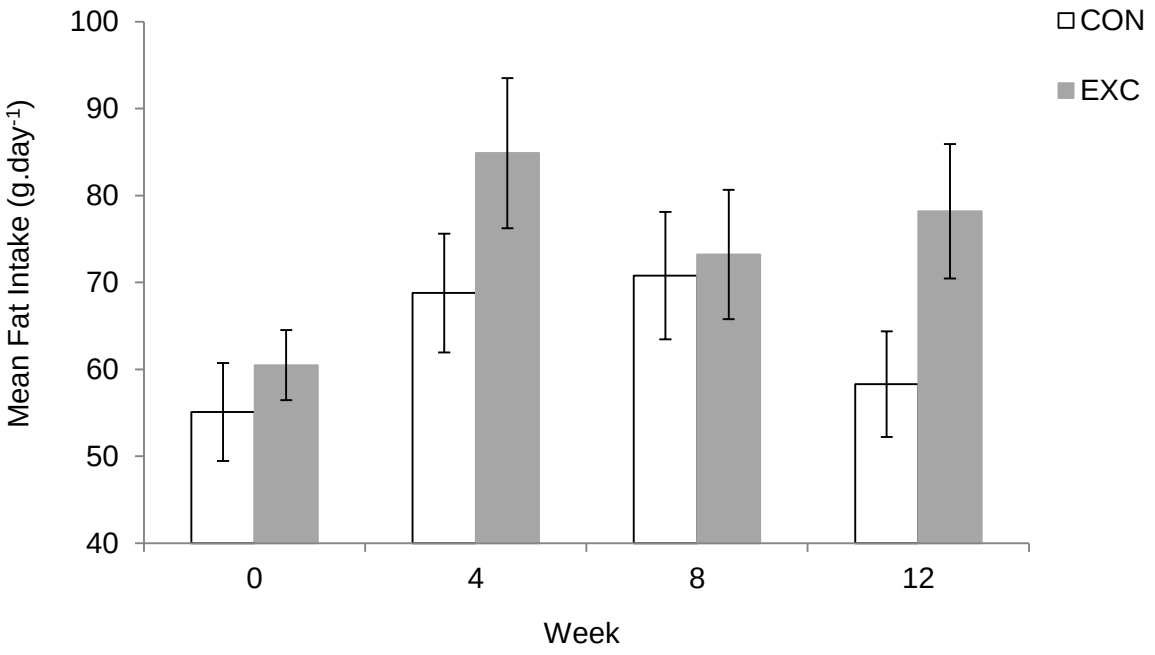

Figure 26: Mean (±SE) fat intake ($g.day^{-1}$) over the 12 week study

There was a significant time effect (F=3.18, df=(3, 66), p=0.0295) for mean fat intake. Both groups combined saw a significant increase (p=0.0149) in fat intake between baseline and week 4 (Figure 26). Importantly, this change did not affect the overall average energy intake of the group.

4.3.1.4. FULL BLOOD LIPID PROFILE

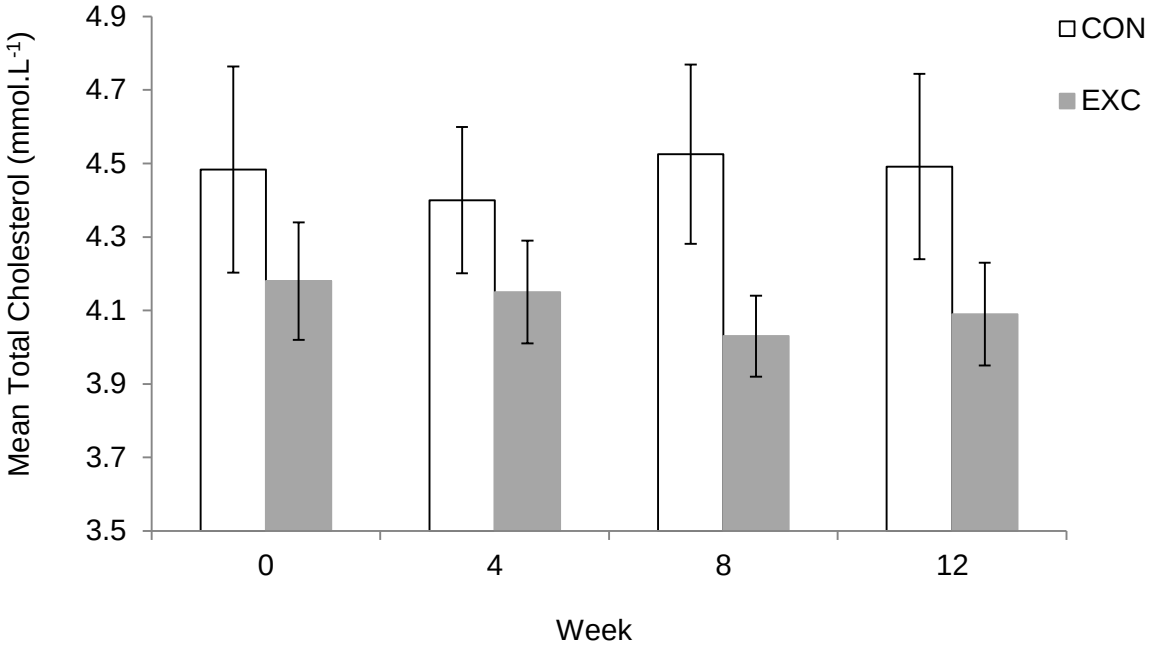

Figure 27: Mean (±SE) total cholesterol (mmol.L^{-1}) over the 12 week study

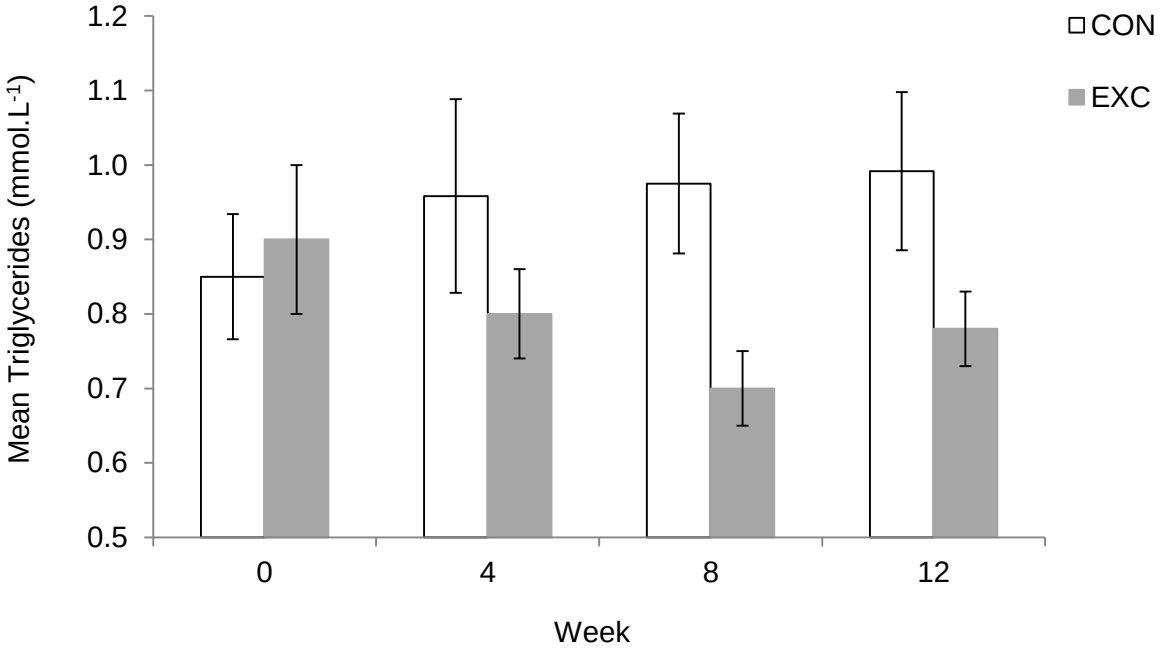

Figure 28: Mean (±SE) triglycerides (mmol.L^{-1}) over the 12 week study

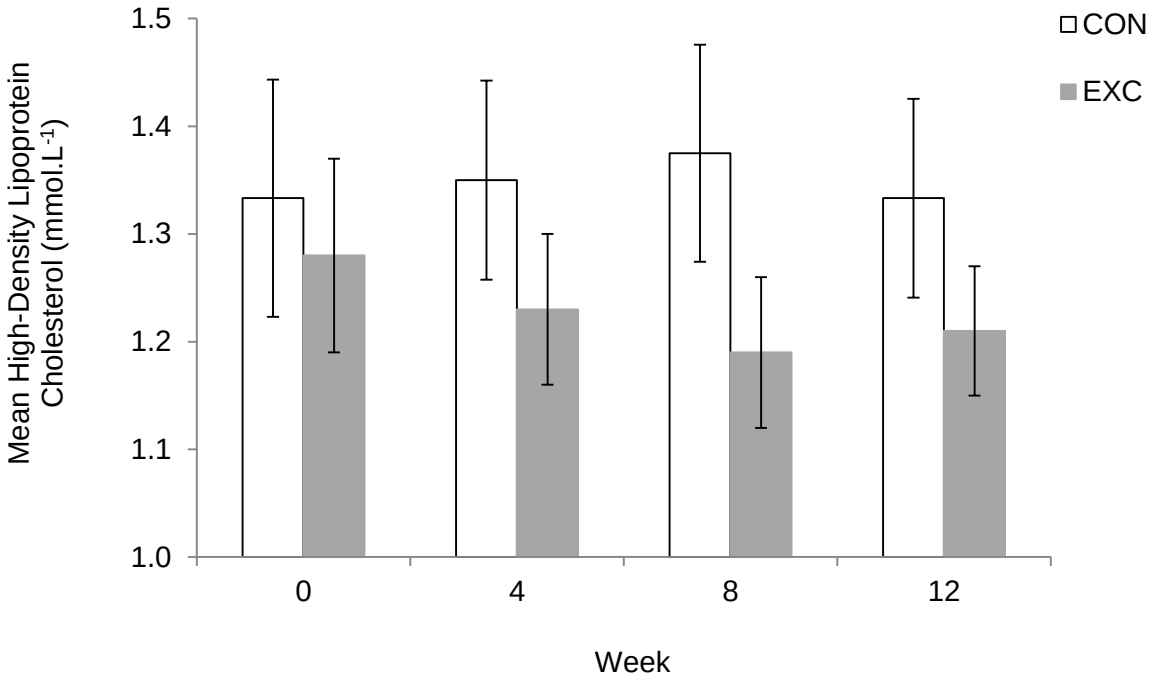

Figure 29: Mean (±SE) high-density lipoprotein cholesterol (mmol.L^{-1}) over the 12 week study

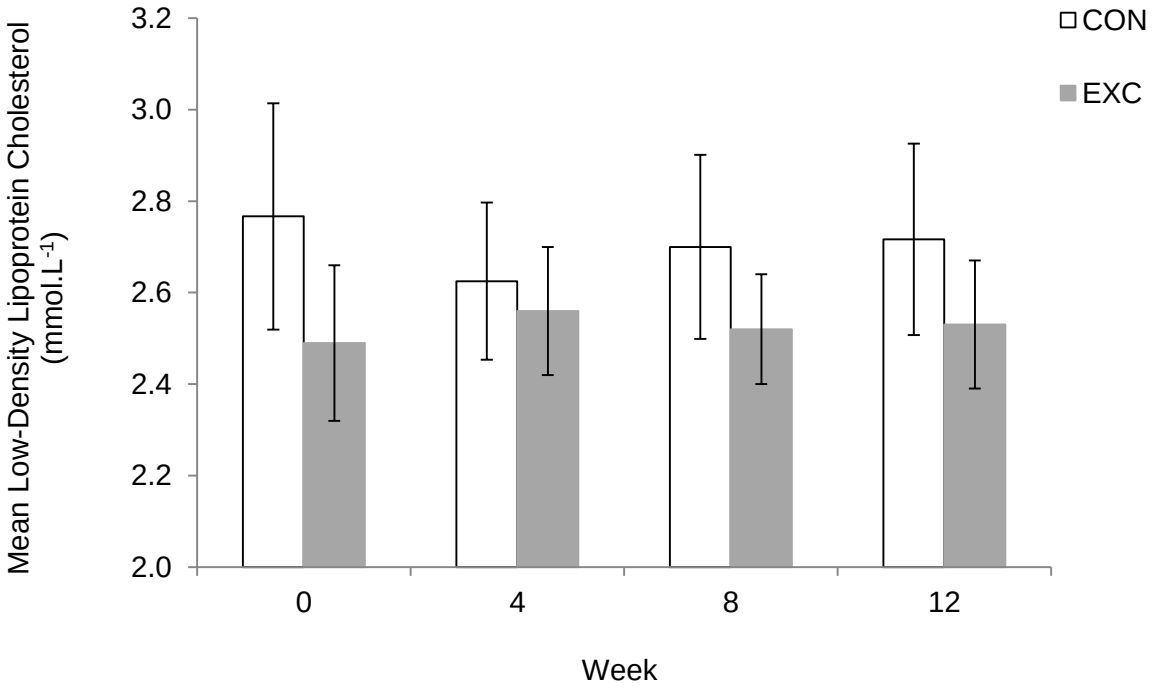

Figure 30: Mean (±SE) low-density lipoprotein cholesterol (mmol.L^{-1}) over the 12 week study

There were no significant differences for mean total cholesterol (Figure 27), HDL-C (Figure 29) and low-density lipoprotein cholesterol (LDL-C) (Figure 30). There was a significant interaction effect (F=3.06, df=(3, 69), p=0.0338) for mean TG levels (Figure 28).

4.3.1.5. GLYCATED HAEMOGLOBIN

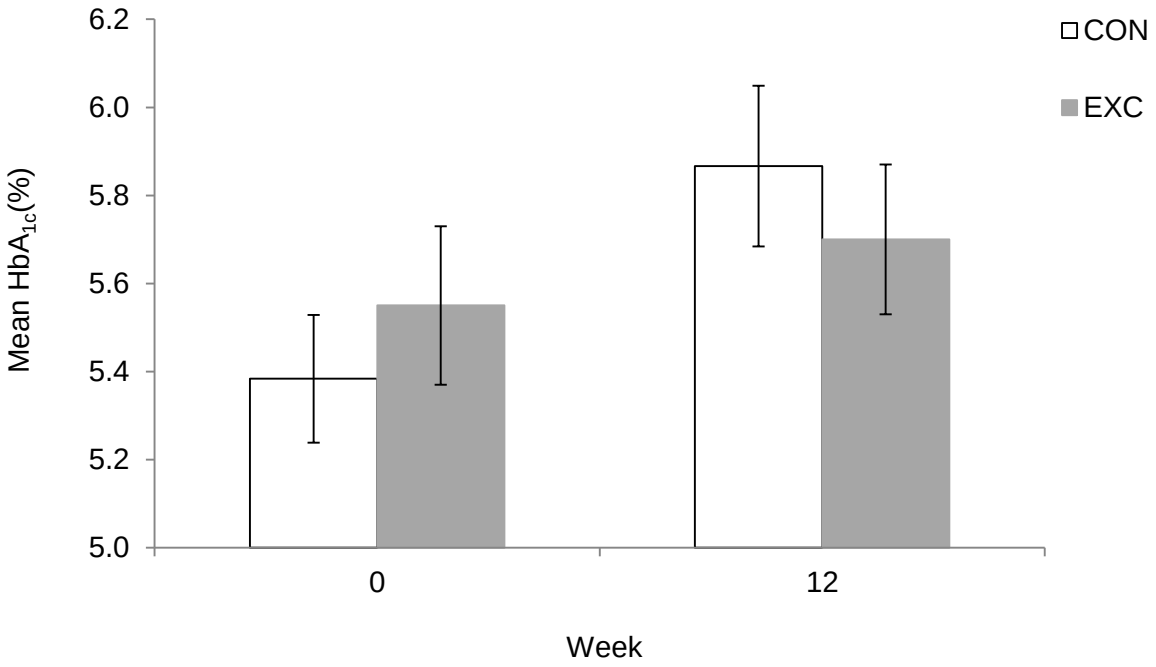

Figure 31: Mean (±SE) glycated haemoglobin (HbA_{1c}) levels (%) pre- and post- the 12 week study

In general, glycated haemoglobin levels significantly increased (F=33.70, df=(1, 23), p<0.0001) from baseline to week 12 (Figure 31). On average both groups independently started the study as 'non-diabetic' and ended with an 'increased risk of diabetes' according to the American Diabetes Association classification. Further, there was a significant interaction effect (F=9.01, df=(1, 23), p=0.0064) between the CON group mean at baseline (5.38% ±0.5) compared to week 12 (5.87% ±0.63) as is highlighted in Table 21.

Table 21: Significant interaction effects for glycated haemoglobin

	CON 0	CON 12	EXC 0	EXC 12
CON 0				
CON 12				
EXC 0				
EXC 12				

4.3.1.6. BODY MASS INDEX

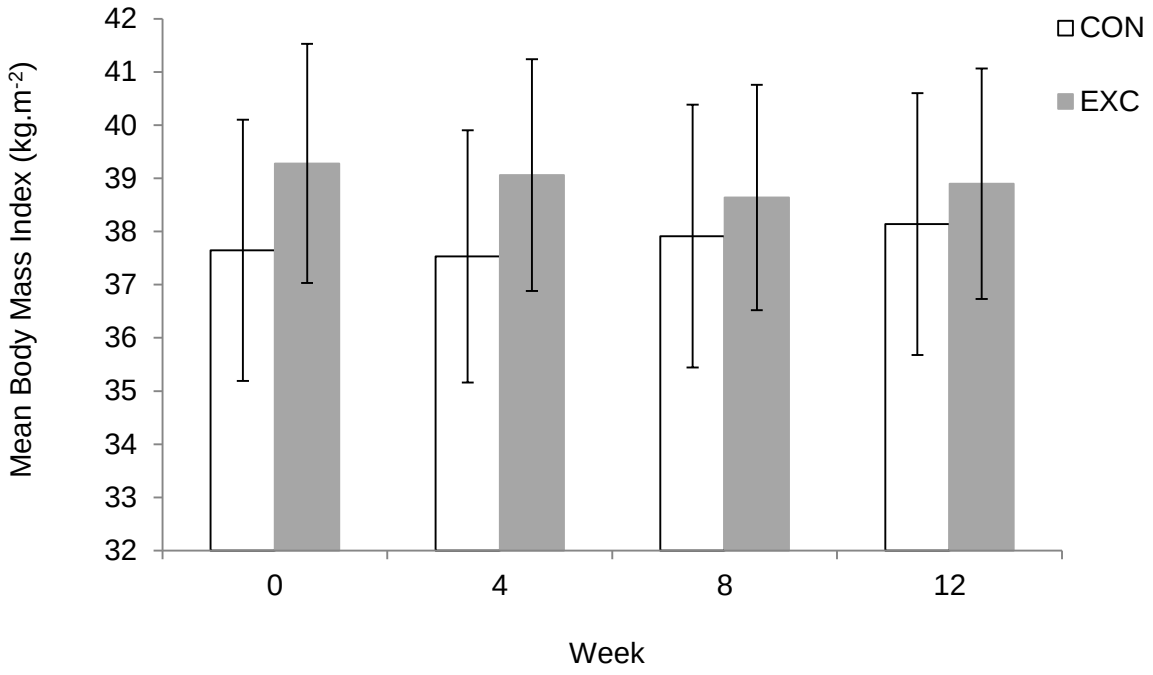

Figure 32: Mean (±SE) body mass index (kg.m^{-2}) over the 12 week study

Linked to the dietary information, it was important that participants maintained their starting body mass to isolate the exercise effect. BMI results showed a significant interaction effect (F=4.56, df=(3, 69), p=0.0057) (Figure 32). The CON group saw an average increase of 0.49 kg.m^{-2} while the EXC group saw an average decrease of 0.92 kg.m^{-2}. However, for both groups, these weight changes did not alter their starting risk category.

4.3.1.7. WAIST CIRCUMFERENCE

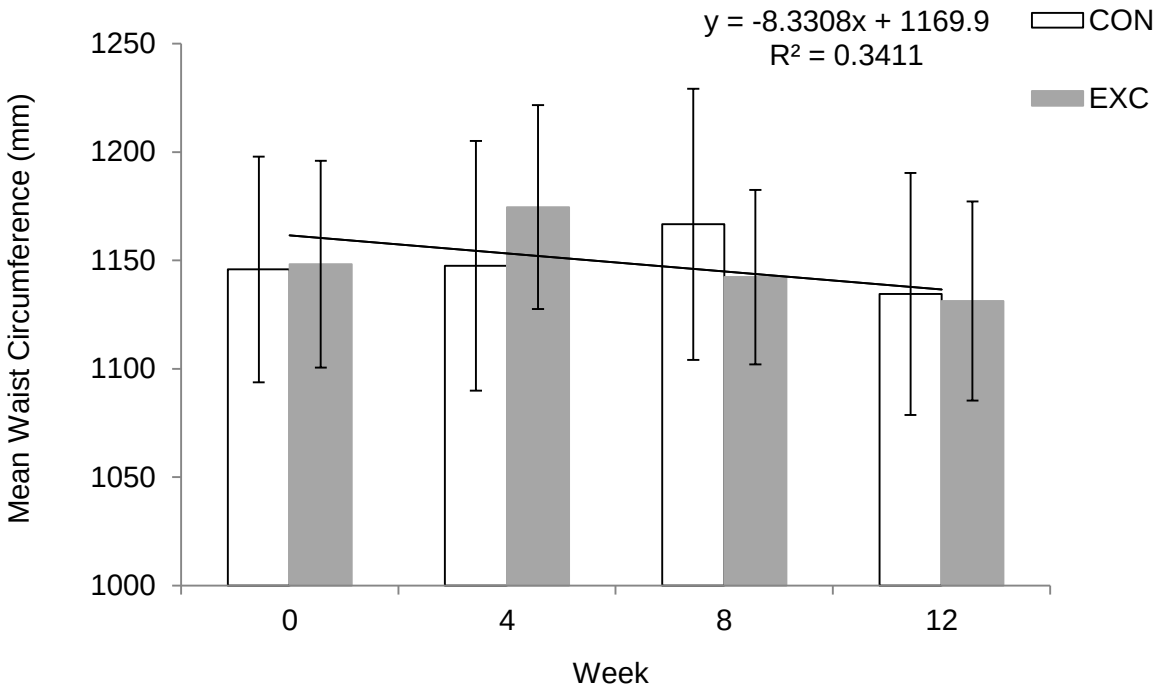

Figure 33: Mean (±SE) waist circumference (mm) over the 12 week study

There were no significant differences between the groups for WC (Figure 33). However, a moderate trend (r=0.5840) for decreasing WC over time existed for the EXC group as is illustrated in Figure 33, while the CON group showed little to no relationship (r=0.1414).

4.3.1.8. BODY COMPOSITION

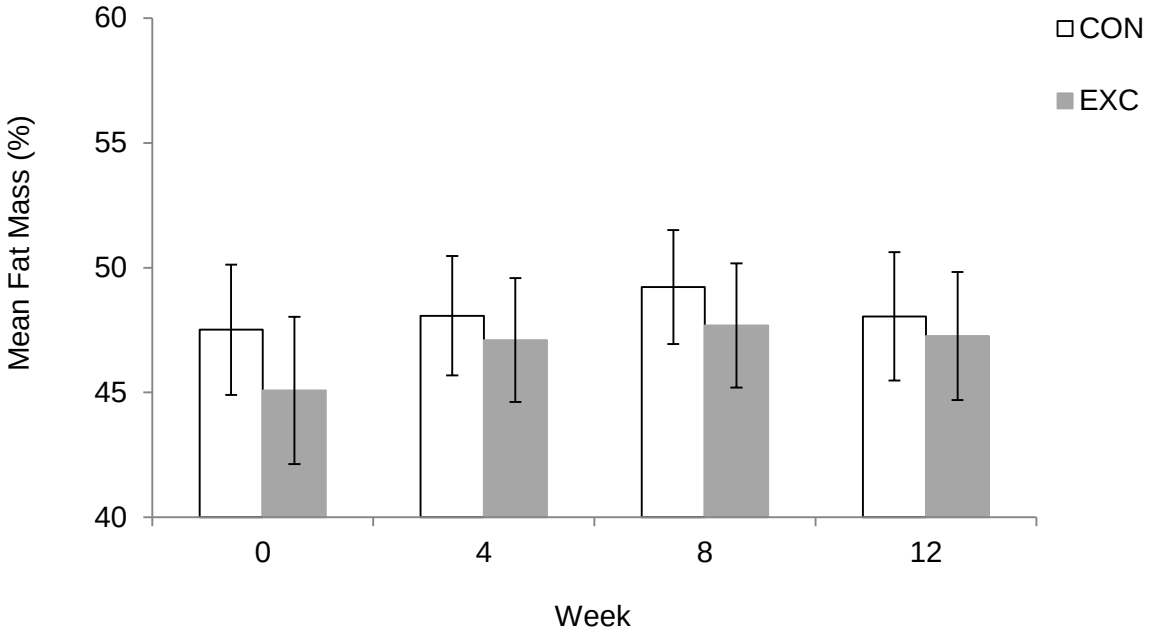

Figure 34: Mean (±SE) fat mass (%) over the 12 week study

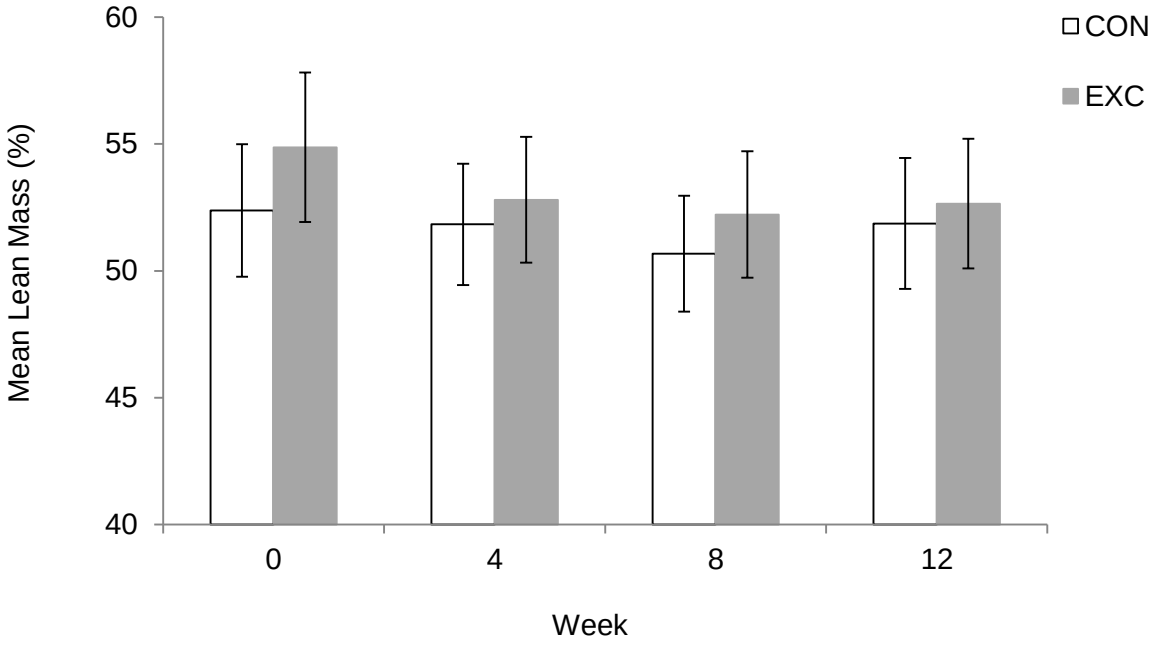

Figure 35: Mean (±SE) lean mass (%) over the 12 week study

There was a significant time effect for fat mass (F=7.50, df=(3, 69), p=0.0002) (Figure 34) and lean mass (F=7.54, df=(3,69), p=0.0002) (Figure 35). The group data combined saw significant differences from baseline through all the measurement intervals, with fat mass increasing and lean mass decreasing over time.

4.3.1.9. HEART RATE

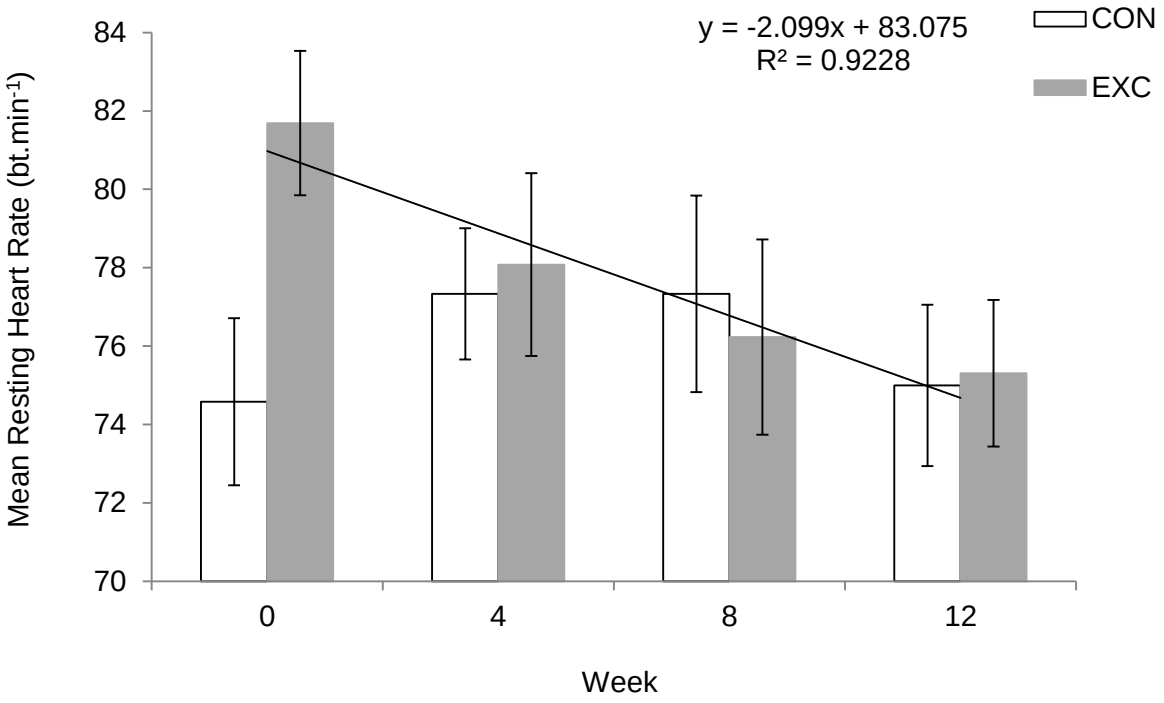

Figure 36: Mean (±SE) resting heart rate (bt.min^{-1}) over the 12 week study

There were no significant differences for mean resting heart rate (Figure 36). However, a very strong trend (r=0.9606) existed for decreasing resting heart over time for the EXC group, as can be seen in Figure 36, but little to no relationship (r=0.1091) in the CON group.

4.3.1.10. BLOOD PRESSURE

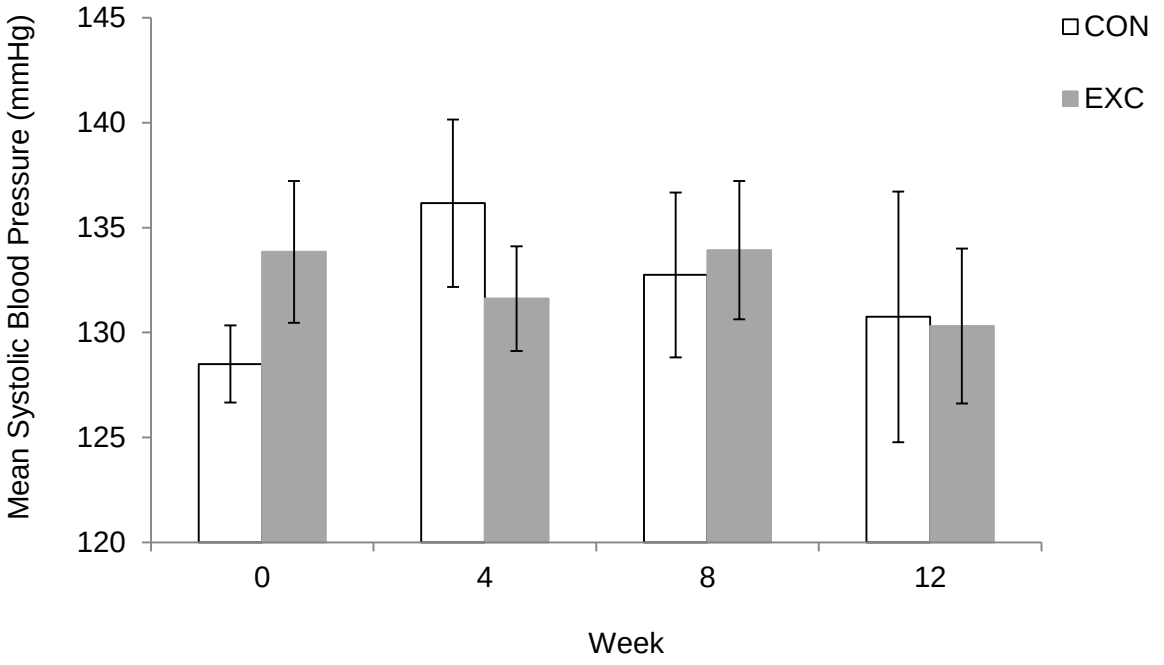

Figure 37: Mean (±SE) systolic blood pressure (mmHg) over the 12 week study

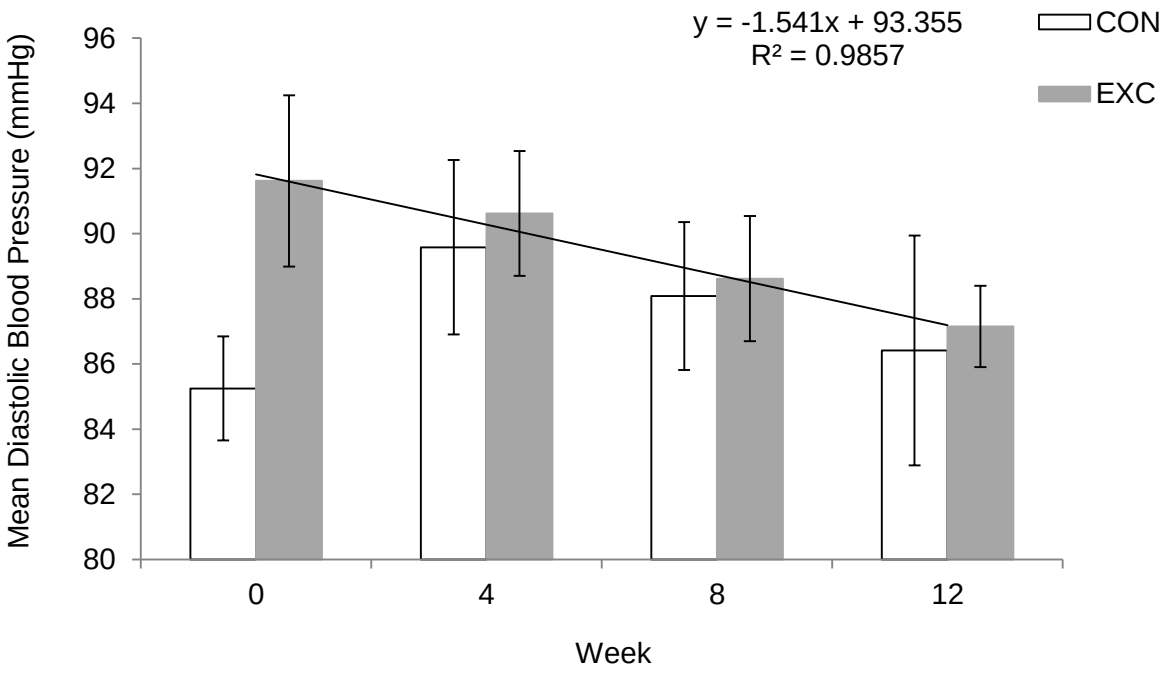

Figure 38: Mean (±SE) diastolic blood pressure (mmHg) over the 12 week study

For both SBP (Figure 37) and DBP (Figure 38) there were no significant differences present. However, there was a very strong relationship (r=0.9928) for decreasing DBP over time for the EXC group (Figure 38) but little to no relationship (r=0.1360) for the CON group.

4.3.1.11. FITNESS

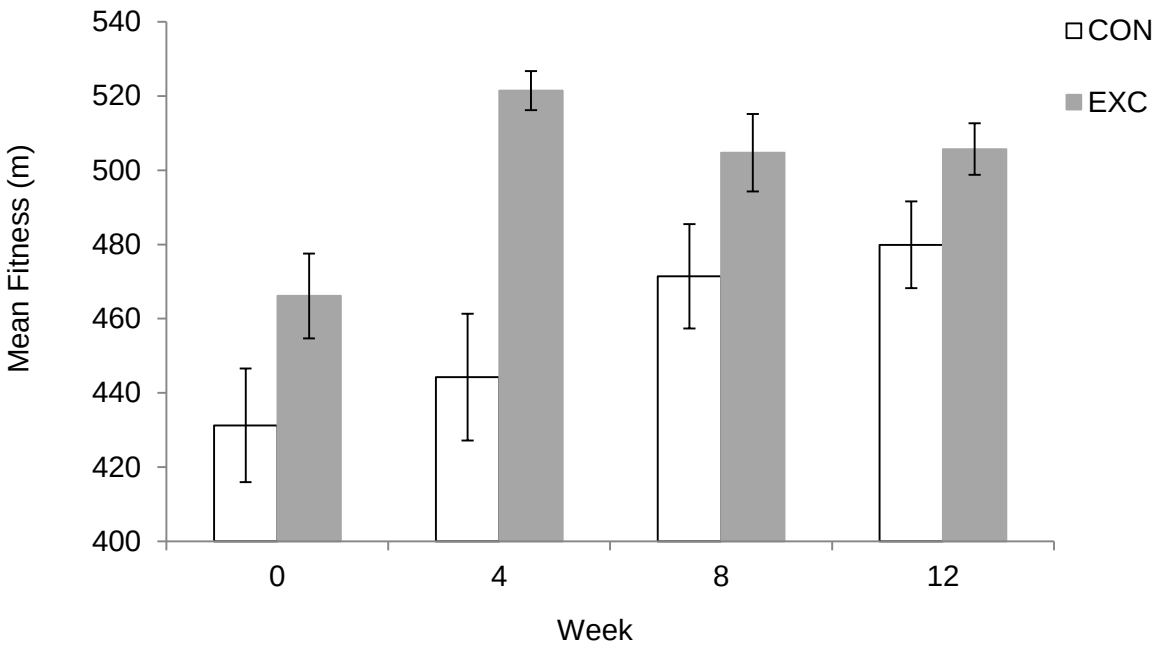

Figure 39: Mean (±SE) fitness (m) over the 12 week study

There was a significant group effect (F=10.96, df=(1, 23), p=0.0031), time effect (F=10.28, df=(3, 69), p<0.0001) and interaction effect (F=3.48, df=(3, 69), p=0.0204) for the fitness results (Figure 39). The EXC group data started at, and continued to have, slightly higher fitness levels than the CON group throughout the study. At week 4, the EXC group had a very large increase from baseline (55.31 m) which was found to be significantly greater (p=0.007) than the CON group who only had a 13 m increase at this point. Thereafter, the CON group continued to progressively increase their fitness while the EXC group data showed a marginal decrease and plateau in fitness level. The significant interaction effects are highlighted in Table 22.

Table 22: Significant interaction effects for the fitness data

	CON 0	CON 4	CON 8	CON 12	EXC 0	EXC 4	EXC 8	EXC 12
CON 0								
CON 4								
CON 8								
CON 12								
EXC 0								
EXC 4								
EXC 8								
EXC 12								

4.3.2. Individual Results

4.3.2.1. COMPLIANCE

The group results may be misinterpreted due to the poor overall compliance of some individuals to the walking programme guidelines. These compliance rates were calculated based on actual vs. optimum adherence at each week which produced a result relative to the individual's baseline values and step goals. The compliance rates were then calculated and expressed as a single percentage taking into consideration both the adherence to the step goals and to the walking intensity (Table 23). Both the CON and EXC groups were then examined side-by-side. The dependent variables which were looked at more closely included the two primary risk factors for CVD which were initially identified from the comparative study: overweight/obesity and high BP.

Table 23: Overall compliance to the walking programme

CODE	OVERALL COMPLIANCE (%)
E 05	68.3
E 19	64.2
E 12	58.3
E 28	52.5
E 10	51.7
E 30	47.5
E 04	46.7
E 02	45.0
E 29	35.0
E 18	30.0
E 14	30.0
E 27	26.7
E 13	11.7

4.3.2.2. RISK FACTOR 1: OVERWEIGHT/OBESITY

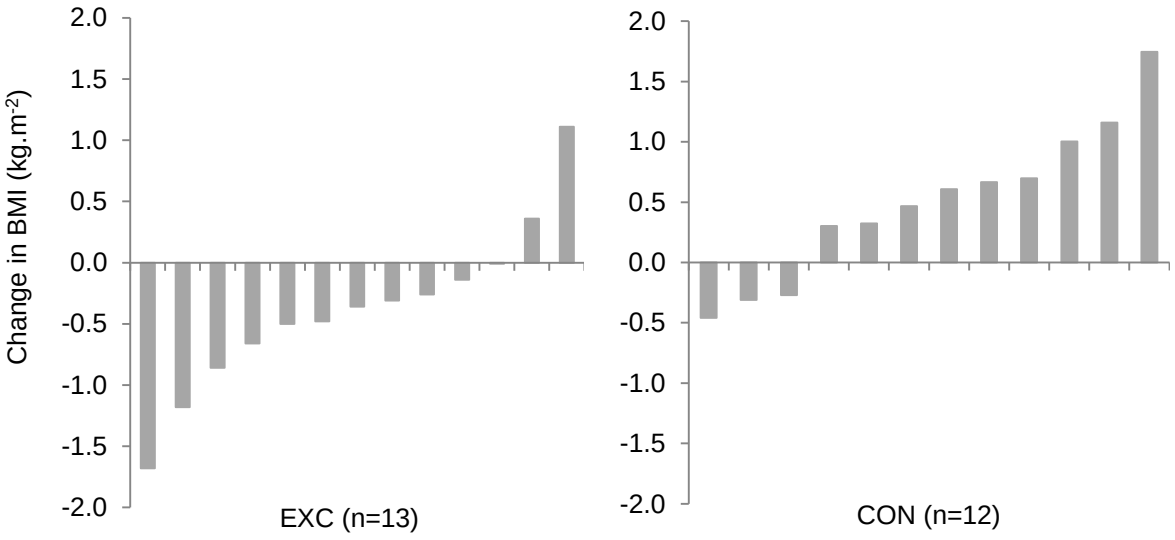

Figure 40: Individual changes (pre- and post-walking programme) in body mass index ($kg.m^{-2}$) in the EXC and CON groups

A beneficial decrease in BMI occurred in more participants, and to a greater extent, in the EXC group compared to the CON group (Figure 40). However, two of the EXC group participants (E19 with 64.2% compliance and E02 with 45% compliance) increased their BMI; therefore, no uniform pattern can be identified between the walking intervention and BMI change.

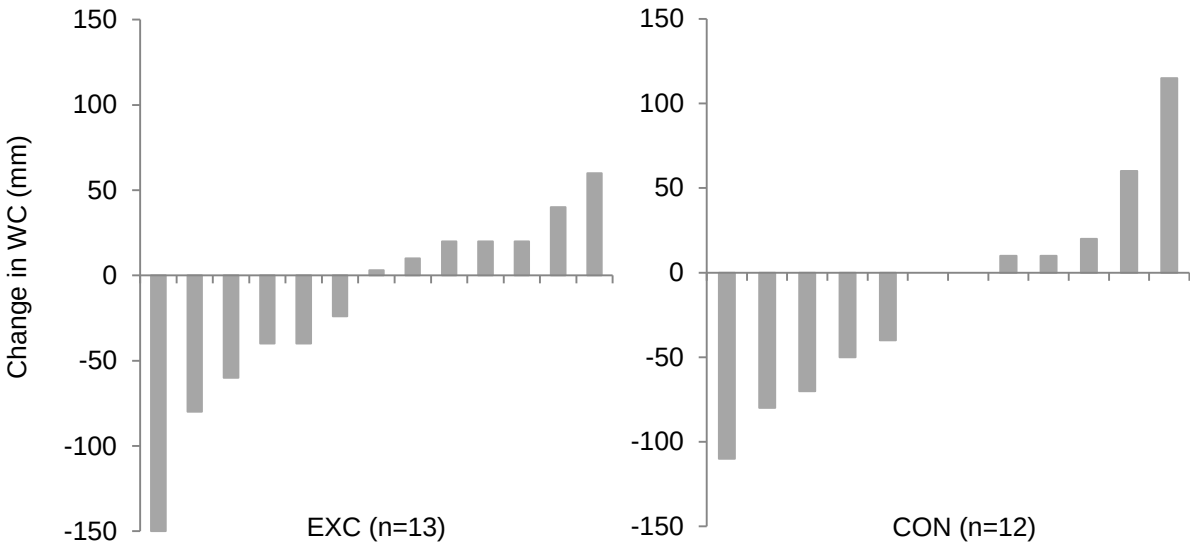

Figure 41: Individual changes (pre- and post-walking programme) in waist circumference (mm) in the EXC and CON groups

Waist circumference changes were very similar in the EXC and CON groups with approximately half the sample in each group increasing and the other half decreasing their WC (Figure 41). The biggest reduction was seen in participant E02 (overall compliance of 45%) from the EXC group with a 150 mm decrease.

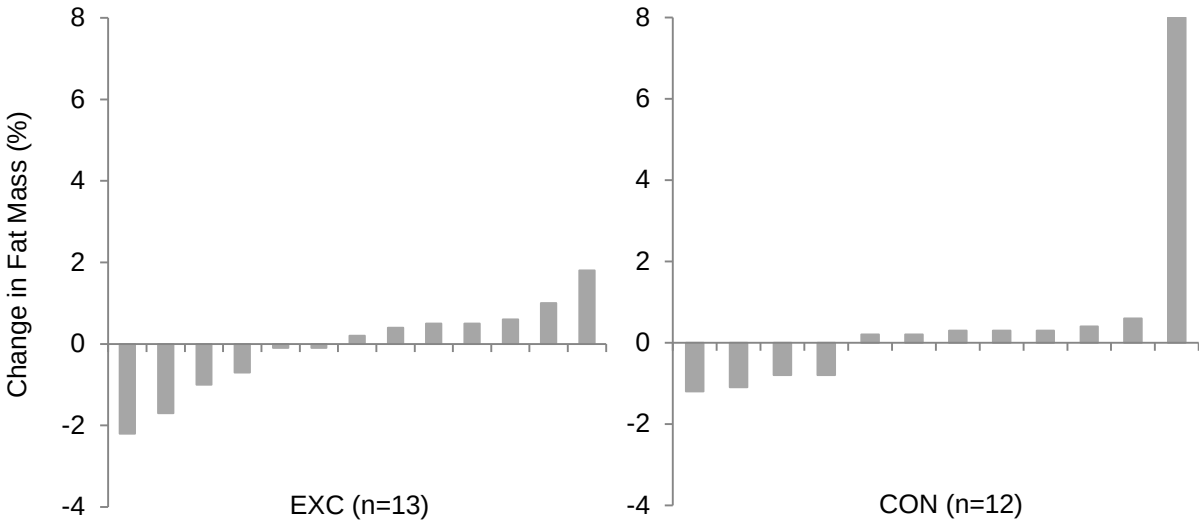

Figure 42: Individual changes (pre- and post-walking programme) in fat mass (%) in the EXC and CON groups

Fat mass changes were similar across groups (Figure 42). Noteworthy is the large fat mass increase (8%) in participant C07 from the CON group which may have been equipment error. The largest loss in fat mass occurred in participants E05 and E10 (-2.2 and -1.7% respectively) from the EXC group who had high compliance rates. However, many participants who engaged in the walking programme had an adverse increase in fat mass, and participant E13 who had the lowest compliance rate (11.7%) achieved a reduction in fat mass, -1%.

4.3.2.3. RISK FACTOR 2: HIGH BLOOD PRESSURE

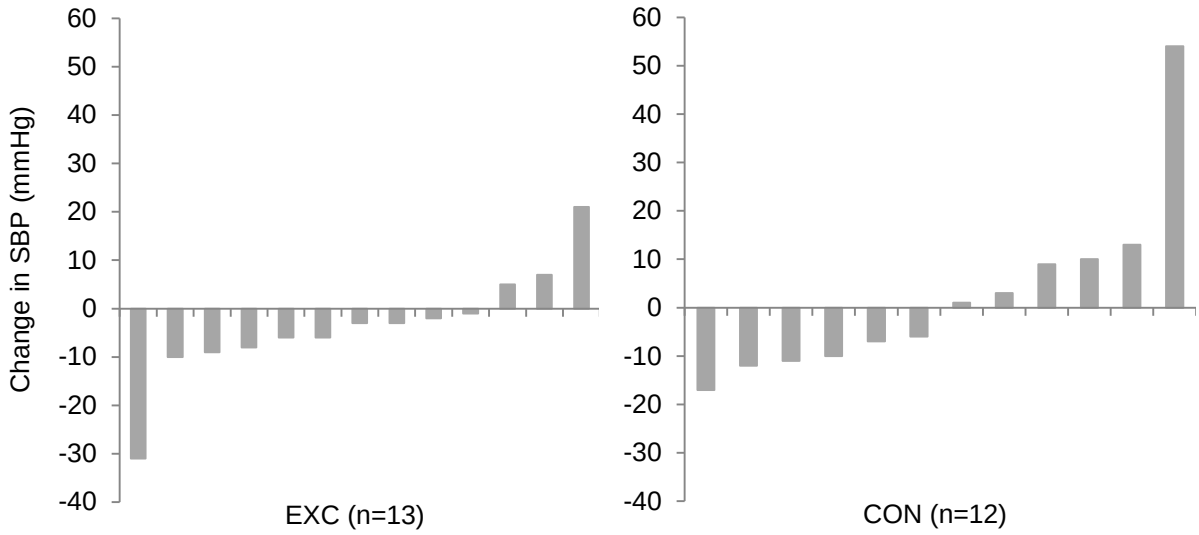

Figure 43: Individual changes (pre- and post-walking programme) in systolic blood pressure (mmHg) in the EXC and CON groups

The majority of the EXC group participants decreased their SBP (10 of the 13 participants) although half of the CON group participants also decreased in this response (Figure 43) and so no exercise effect can be concluded. The greatest decrease in SBP was seen in E27 (-31 mmHg) from the EXC group who had a very low compliance rate (26.7%) while an adverse increase in SBP was seen in participant E19 (+21 mmHg) from the EXC group who had the second highest compliance rate (64.2%).

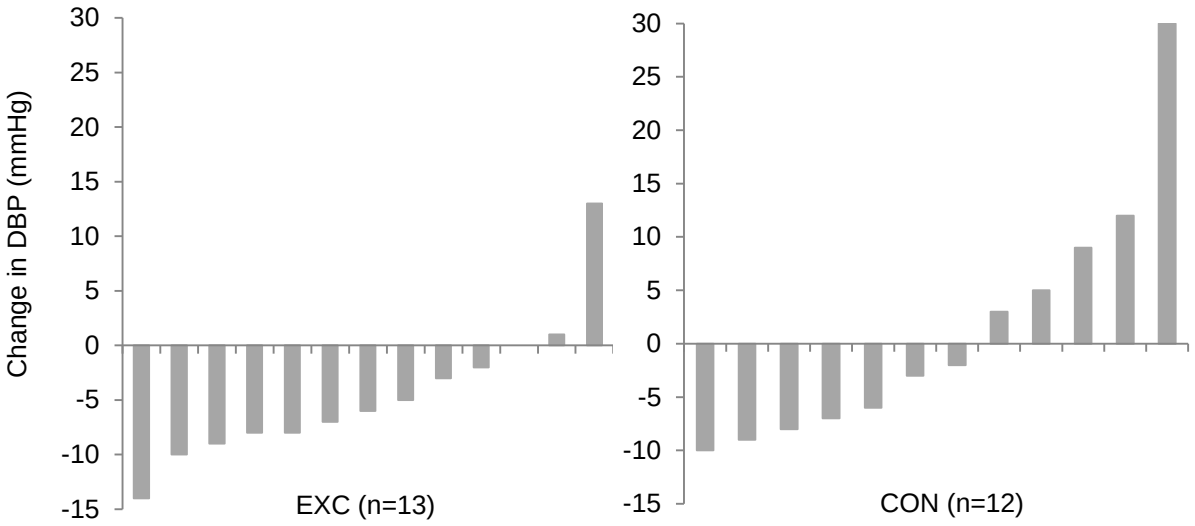

Figure 44: Individual changes (pre- and post-walking programme) in diastolic blood pressure (mmHg) in the EXC and CON groups

The majority of the exercising participants (10 of the 13) responded to the walking programme with a decrease in DBP (Figure 44). This finding was mimicked in the CON group where 7 of the 12 participants also had a decrease in DBP. The anomaly in the EXC group was participant E04 (compliance of 46.7%) who had a very large and unexplained increase in DBP (+13 mmHg). Participant E13, who did not adhere to the intervention (11.7%), showed an increase in DBP (+1 mmHg).

4.4. LIMITATIONS TO THE RESULTS

For both the initial comparative part of this study and the exercise intervention the biggest limitation was the sample size. This sample size was smaller than ideal, 40 and 25 participants respectively, due to the inclusion criteria being specific and making recruitment within such a small community difficult. Recruitment was of a passive nature making the process slower. Further, the length of the intervention study was long (12 weeks) and this required a high level of commitment from the participants.

The walking intervention was designed as a quasi-experimental study rather than a randomised controlled trial since accommodating participant work schedules was a big priority. In comparison, a quasi-experimental study is less preferable as it lacks the element of random assignment to treatment which may have negatively affected the results.

Another limitation for the intervention was the unsupervised nature of the walking programme. This may have resulted in both inter- and intra-individual variability due to the environmental conditions which the participants walked in. This limitation was unavoidable as making the walking programme supervised wouldn't have been feasible within the scope of this research and has previously been found to increase attrition rates.

Although pedometers have been found effective tools in physical activity programmes, their ease of use in populations such as that under investigation is not well-known. At the beginning of the study there were major issues encountered regarding the functionality of the pedometer despite specific instructions and demonstrations. Some participants forgot to wear the pedometer on certain days, particularly during their off-duty weekends as many associated the study with the university schedule which resulted in missing data.

To avoid possible contamination of data it is advised that condition groups be kept isolated from each other throughout the course of an intervention. This was not adhered to in the current study since the primary concern in this research was the participants' work schedules rather than their respective work places. This may have lead to groups interacting and influencing each other and therefore the data.

This study relied on the honesty and compliance of the participants specifically when completing the pre-screening forms and the monthly questionnaires. Any dishonesty regarding medication usage would reduce the accuracy of the results obtained. To

further increase the reliability and validity of the study, the calibration of equipment and machines was guaranteed by the primary researcher and the same equipment and machines were used for each participant at each testing session. If, however, the equipment and/or machines were faulty and incorrect readings were given, the accuracy of the results could be questionable.

A manual sphygmomanometer was used as opposed to an electronic one which may have led to less accurate results. However, the same manual device was used at each session (by the principal researcher) but this device should have been calibrated prior to the first testing session.

The iliac WC measurement was challenging to obtain in this population group as most participants were overweight/obese making the palpation of anatomical landmarks difficult. The heart rate and BP measurements were taken manually by the primary researcher and so there may have been intra-individual variability.

For standardisation purposes, when participants underwent body composition tests in the form of bioelectrical impedance analysis, it was requested that they not have any fluid or food intake 2-3 hours prior to testing, no exercise 24 hours prior to testing and to remove all jewellery prior to testing. This was dependent on participants' cooperation and any deviation from these instructions may reduce the validity and reliability of the results. Additionally, there may have been intra-individual variability in terms of placement of the electrodes by the research assistant (even though the placement points were standardised) and this could result in inaccurate results.

For the fitness test, instructions were given for the participants to walk at their own pace and to personally challenge themselves to cover as much distance as possible within six minutes. Due to personnel and time constraints, the participants completed this test in groups and so this may have affected individual performance.

CHAPTER 5

DISCUSSION

5.1. COMPARATIVE STUDY

The first phase of this study identified overweight/obesity and high blood pressure (BP) as the two most prevalent and concerning cardiovascular disease (CVD) risk factors. These two risk factors were not only common, but were also measured as being significantly greater than one, or both, of the previous national surveys. Overweight/obesity and high BP are both independently associated with an increased mortality and morbidity of CVD (Donnelly *et al*., 2000; Joint National Committee, 2004; Chan and Woo, 2010; Wang and Nakayama, 2010; Bentley and Dezorzi, 2012; Després, 2012; Jiang *et al*., 2013; van Baak, 2013) which highlights the importance of addressing these two issues in particular.

The current study revealed a very high body mass index (BMI) of 37.6 $kg.m^{-2}$ (Figure 9, page 84) and fat mass of 45.23% (Figure 10, page 85). The implication of this elevated BMI value is shown in a prospective study on a large United States (US) cohort which found that having a BMI value in the range of 35.0-39.9 $kg.m^{-2}$, as was found in the current study, was associated with excess CVD mortality with a hazard ratio of 1.87. This association was independent of sex, smoking status and education level (Jiang *et al*., 2013). More specifically, a Framingham Heart Study found that for every 1 $kg.m^{-2}$ increase in BMI, the risk of heart failure increased by 7% in women (Lavie *et al*., 2010). However, although the most common method of defining overweight and obesity, BMI does not necessarily reflect true body fatness (Lavie *et al*., 2010). The World Health Organisation (WHO) recommends, where possible, that abdominal obesity be measured and used in conjunction with BMI to assess and predict disease risk (Shields *et al*., 2012). Results yielded an average waist circumference (WC) of 1130.58 mm (Figure 11, page 86) which indicates that the risk of metabolic complications is substantially increased (Chan and Woo, 2010). Another large prospective study in US men and women showed that when WC and

BMI were mutually adjusted in a model, WC was related to a 1.82-fold increase in death from CVD (Leitzmann *et al*., 2011). Obesity assessment via WC is difficult to interpret though as optimal cut-off values are affected by sex, age and race. As it stands, the general guidelines specified by the WHO in 2000 are from a Caucasian cohort only whilst several other population groups have also been validated (James, 2005; Chan and Woo, 2010). However, Sub-Saharan Africans are not included in these groups and are rather advised to "use European data until more specific data are available" (James, 2005; Crowther and Norris, 2012) which surfaces the need for specific reference for different African populations. A study in urban African females recommends the WC cut-point to increase from 800 mm to 915 mm (Crowther and Norris, 2012) although there are slight differences in the literature (Botha, 2012; Botha *et al*., 2012). Despite these proposed changes to WC cut-points, the current result still indicates high risk within this sample. Paired with the other obesity measures, these results indicate that overweight/obesity is continuing to rise far beyond the acceptable limits. It can confidently be proposed that, in this population group, this risk factor is being driven primarily by adverse lifestyle behaviours associated with urbanisation. This is supported by the THUSA (Transition and Health during Urbanisation of South Africans) study which measured BMI in five different strata ranging from deep rural areas to affluent, Westernised areas. It was found that BMI values of female black Africans progressively increased from the most rural (25.6 $kg.m^{-2}$) to the most urban (28.1 $kg.m^{-2}$) areas (Vorster, 2002). Further, urbanisation in South Africa is mainly due to the rapid migration of black Africans (Vorster *et al*., 1999).

A prospective study on black South Africans found that 24% of the participants with optimal BP developed hypertension during the five years, and that one of the two strongest contributors to this development was abdominal obesity (WC) (Schutte *et al*., 2012). The Joint National Committee (2004) positioned that BP values between 130-139/85-89 mmHg are associated with a more than twofold increase in relative risk from CVD as compared with levels below 120/80 mmHg. This is of concern as the result from the current study for systolic blood pressure (SBP) was 130.33 mmHg (Figure 12, page 87) and diastolic blood pressure (DBP) was 87.90 mmHg (Figure 13, page 88). Since high BP is the primary risk factor for CVD (Forrester, 2004;

Gaziano *et al*., 2005; Cheung *et al*., 2005; Kearney *et al*., 2005; Chockalingam *et al*., 2006; Kaplan and Opie, 2006; Kemp *et al*., 2011), these findings support the urgent need for BP prevention and treatment therapies within South African black females.

Exercise has been specifically recommended as a lifestyle modification for hypertension, yet, it was recently shown that physical inactivity is still very prevalent (>50%) in South African females (Bravata *et al*., 2007; Sohn *et al*., 2007; Lee *et al*., 2010; Hallal *et al*., 2012). The physical activity measures in the current study were of interest even though results pointed towards 'normal' values. Looking in more detail at these risk factors, some discrepancies were observed. Daily walking was measured continuously for a one week period with results averaging 10162 steps.day^{-1} which is classified as 'active' (Table 15, page 90). Le-Masurier *et al*. (2003) studied 59 women aged 20-65 years and found that individuals who accumulated 10000 steps.day^{-1} were more likely to meet the current physical activity guidelines, but the data also revealed that this relationship was not guaranteed. Further, a cross-sectional analysis found that physical activity intensity, and more specifically higher intensity, is the main characteristic of physical activity determining its effect on CVD risk factors (Sassen *et al*., 2009). This information highlights the importance of intensity in walking programmes in order to confer health benefits. Even though the daily step count in this cohort was high, moderate-to-vigorous physical activity (MVPA) time accumulation was only 19.42 mins.day^{-1} as opposed to the ACSM recommendation of 30 mins.day^{-1} (Haskell *et al*., 2007; Murphy *et al*., 2007; Whitt-Glover *et al*., 2012). Adding to this, many participants did not understand the significance of, and relationship between, walking intensity and CVD risk. This finding identifies a 'gap' which can be positively manipulated to produce beneficial results in this sub-population.

Another measure of physical activity, energy expenditure, was recorded at 1336.4 METmins.week^{-1} which is associated with 'moderate' activity and is supported by the SADHS findings (Figure 15, page 91). However, this variable was measured via the Global Physical Activity Questionnaire (GPAQ version 2), and although found to be an accurate self-report tool, it was observed that participants responded inaccurately

as they struggled to understand moderate- and vigorous-intensity activity. Huge effort and heavy breathing for this population group was justified as climbing stairs (perceived as vigorous) but is actually a moderate-intensity activity. This is supported by Cook *et al*. (2010) who suggest that the overestimation of reported physical activity may be the result of misclassification of sedentary and light activities as moderate. Also, the participants may have wanted to make their job requirements seem more taxing than they really are, possibly for workplace credit, which may have contributed to the skewing of the data. Further, it was observed that energy was expended primarily for transport reasons and to a lesser degree for work, with little to no physical activity occurring for recreational purposes again highlighting an area for possible intervention. This is in line with previous literature which found that black South Africans in more rural settlements had a high ambulation level (12471 steps.day^{-1}) which was linked to increased non-leisure, subsistence demands as well as active transport (Cook *et al*., 2010). Ross *et al*. (2009) state that "increasing physical activity is associated with a marked reduction in waist circumference, visceral fat and cardiometabolic risk factors, concurrent with an increase in cardiorespiratory fitness despite minimal or no change in body weight". This was supported by two correlations which found a weak negative relationship between both WC and fitness (Figure 18, page 94) and daily step count and fat mass (Figure 19, page 95). The importance of these inter-connected relationships within the scope of this research is the positive effect physical activity has on certain CVD risk factors. A recent study in obese participants found that decreasing WC to healthy levels (<940mm in males and <800 in females), favourably altered certain CVD risk factors including BP, hyperglycaemia, triglycerides (TGs) and Framingham risk at ten years (Fanghänel *et al*., 2011).

The biochemical analyses for diabetes highlights another area of concern in this population group. In South Africa, type II diabetes is quickly emerging as a serious health problem, specifically in the black African population as they become more urbanised (Ntyintyane *et al*., 2010; Goedecke *et al*., 2013). Adding to this burden, women of African ancestry have been found to experience a disproportionately higher prevalence of type II diabetes compared to their white counterparts. Similarly, obesity and insulin resistance, which are major risk factors for type II diabetes, are

greater in black compared to white women (Knight *et al*., 2011; Goedecke *et al*., 2013). The HbA_{1c} result from the current study was 5.87% which is classified as 'pre-diabetic' and supports the findings from the SANHANES-1 (Figure 17, page 93). Noteworthy is that 87% of all type II diabetes in South Africa is attributable to elevated BMI (Goedecke *et al*., 2013) routing back to the obesity epidemic that black African females are experiencing. This suggests that addressing the overweight/obesity issue in this population group could positively affect their risk of diabetes. The second biochemical analysis test looking at lipid profiles was also an issue even though results revealed 'low risk' for total cholesterol, TG and low-density lipoprotein cholesterol (LDL-C). The red flag existed for high-density lipoprotein cholesterol (HDL-C) which, at 1.31 $mmol.L^{-1}$, is classified as 'borderline high risk' (Figure 16, page 92). This supports previous literature in the black population, and specifically black South Africans, which shows insulin resistance in this racial group to present with a characteristic lipid profile of normal TG levels and low HDL-C which is referred to as the 'TG paradox' (Kannel and Vasan, 2009; Knight *et al*., 2011; Yu *et al*., 2012). This differs from the general population where the dyslipidemia of insulin resistance follows the classic pattern of elevated TG and low HDL-C. Therefore although at face value these results, which were similar to those in the SANHANES-1, present as 'low risk', they indicate a predisposition to diabetes. Correlation analysis revealed a moderate positive correlation between TGs and resting heart rate (Figure 20, page 95). In the general population, targeting TGs as a vascular risk factor is justified because of the role of TC-rich lipoproteins in atherogenesis (Kannel and Vasan, 2009). However, given the 'TG paradox' that the black African population experiences, this risk factor is of less concern to their overall CVD risk profile and rather approaches, perhaps dietary, to increase HDL-C should be investigated.

The remaining CVD risk factors were of little concern. A high resting heart rate shares an independent and strong association with an increase in cardiovascular and all-cause mortality in the general population (Hsia *et al*., 2009; Perret-Guillaume *et al*., 2009; Cooney *et al*., 2010) as well as in hypertensive patients (Cook and Hess, 2010), but the result of 77 $bt.min^{-1}$ places this group in a safe range for this variable (Figure 14, page 89). However, it is important to continue to monitor resting

heart rate since Cooney *et al*. (2010) reported that for each 15 bts.min^{-1} increase in resting heart rate there was an increased CVD mortality with hazard ratios of 1.32 in women. The data from the CAGE questionnaire illustrated that 5% of the sample were 'heavy drinkers' (Table 17, page 94), but this was based on a self-report which may have been inaccurate. Given this limitation, the result is still in line with the 2003 SADHS where black African females were reported to have the lowest levels of alcohol consumption (6.6%), although, given the social pressures of women in certain cultures not to drink alcohol, it is possible that under-reporting may be even greater in this sub-population (Schneider *et al*., 2007). The data from the tobacco use questionnaire was even more positive as 0% of the cohort reported as 'non-smokers' (Table 16, page 93). Again, this was from a self-report questionnaire which allows for a larger margin of error. Further, this result may have been inaccurate as the questionnaire did not address the use of smokeless tobacco. A study conducted on the rural black African population in Limpopo, South Africa, found that these women markedly smoked more frequently than the population-matched national sample (1998) and also used smokeless tobacco (snuff and chewing tobacco) far more frequently than their black counterparts (Alberts *et al*., 2005). The use of smokeless tobacco is widely accepted and is even considered to have beneficial health effects. These findings highlighted the urgent need to address this traditional form of tobacco use in black African women (Alberts *et al*., 2005).

5.2. INTERVENTION STUDY

The current walking intervention was completed unsupervised so to represent a sustainable lifestyle behaviour. This approach is conversely supported in a study by Murtagh *et al*. (2005) who supervised participants as they "walked briskly" on indoor treadmills throughout a 12 week programme. The author acknowledged this supervised technique as a limitation and recommended the exercise prescription be repeated in free-living conditions so that attrition rates could be clarified. Rowe *et al*. (2013) found that when asked to walk for ten minutes at a 'brisk' pace, overweight and obese inactive adults walked at a cadence and speed that are above moderate-intensity and were able to maintain this pace for ten minutes without slowing down. Further, in sedentary overweight and obese women the use of step goals and

immediate feedback from using a pedometer has proven effective in increasing physical activity levels (Pal *et al*., 2009). Despite this evidence to support the implementation of an unsupervised walking programme, the results from the current study showed that this type of programme was mostly ineffective. Of the possible 13 participants only two reached their step goal and zero the walking intensity, on at least five days of the week for the full duration of the study (Table 18, page 96). The main reasons provided by the participants for the poor adherence was “not having enough time” and “being too tired”. There were several other observed factors which may have contributed to these overall poor compliance rates. Primarily the lack of knowledge about the benefits of physical activity engagement hindered the success of this study. Although these benefits were repeatedly explained, both in written and verbal formats, it was observed that the participants were more concerned about weight loss than any cardiovascular benefits. This may have been because of researcher-participant communication barriers or a general lack of information around the potential cost-effective advantages of activity. Secondly, at the beginning of the study there were major issues encountered regarding the functionality of the pedometer. Participants were confused with how to access the data despite having this demonstrated several times and this meant that many data fields were incomplete. Thirdly, it was observed that the participants would arrive promptly for their blood tests (and in a fasted state as instructed) but many would arrive late/not at all to the measurement sessions therefore affecting results. This may be because they perceived the blood tests as being more of a financial benefit and were also more interested in those specific results. Contributing to this may be the perception that the pathologist was a more professional situation compared to attending the measurement sessions hosted by a young researcher. Lastly, time constraints played a role in participant compliance, specifically during the Grahamstown National Arts Festival. This ten day festival altered participants’ work schedules by extending working hours and making the days both more demanding and more exhausting. Many participants failed to meet the walking guidelines during this time period as a result.

Going forward, an alternative option for an unsupervised walking program is to make the intervention group-based. Kassavou *et al*. (2013) conducted a meta-analysis and

concluded that, despite low homogeneity of results and other limitations, interventions to promote walking in groups are efficacious at increasing physical activity. Another option, either for a supervised or unsupervised intervention, is the use of rhythmic auditory feedback (such as a metronome or music) which is an effective method for regulating walking at a prescribed intensity in inactive adults (Rowe *et al*., 2013). This could accommodate the prescription of progressive walking programs in which walking intensity slowly increases as the program goes on (Fitzsimons *et al*., 2008). It could further enable the use of higher intensity walking in a shorter time, in order to meet current guidelines, especially for individuals who have limited time to incorporate physical activity into their daily schedules (Rowe *et al*., 2013) as was voiced in the current study. Despite the failure to meet the guidelines, based on the recorded step counts, the EXC group significantly increased their physical activity when compared to the CON group (Figure 21, page 97). The GPAQ version 2 further supports this finding as energy expenditure in the CON group followed no trend while that in the EXC group gradually increased at monthly intervals with a significant increase existing between the starting and ending mean value (Figure 22, page 98), although this was a self-report measure and so could be inaccurate. So, although adherence to the initial study requirements were not met, the EXC group still showed a significant increase in physical activity and so the discussion pertaining to the exercise effect must be considered within this frame.

5.2.1. Group Results

In order to isolate the exercise effect on BP, and therefore draw accurate conclusions, it was important that certain variables be controlled. From baseline through to week 12 the results confirmed that diet and body weight remained constant which allows the findings to be attributed to the walking intervention. Although total energy intake was maintained (Figure 23, page 99), it was noted that under-reporting occurred across all participants. Participants did not monitor all food intake (usually wrote down breakfast, lunch and dinner but not any snacks or foods in-between) and did not always include all the components of a certain dish. This error was noted at the monthly measurement sessions when the principal researcher and the participants privately discussed the food intake sheets for clarity. This may

be because, as caterers in the University dining halls, they are not allowed to 'pick' at the food that they were preparing so they didn't want to get in trouble by reporting this. Also, portion sizes were reported as being much smaller than they really were which may be a perception issue as they do not want to portray a certain image about themselves. The BMI result (Figure 32, page 105) was expected since it has been found that physical activity is not closely associated with alterations in body mass, unless accompanied by specific dietary restrictions on reducing caloric intake (Behall *et al*., 2003; McArdle, 2007).

The primary measure of this intervention study was the exercise effect on BP, both systolic and diastolic. Two previous meta-analyses investigated the same issue by looking at studies with brisk walking programmes lasting at least four weeks in sedentary, but healthy, adult populations. The one meta-analysis found significant decreases in both SBP and DBP (Kelley *et al*., 2001) while the other only found a significant reduction in DBP (-1.54 mmHg ±0.79) (Murphy *et al*., 2007). Looking more specifically at walking interventions which were 12 weeks long, a frequency of five days.week^{-1} has proven effective in significantly reducing both SBP and DBP (Iwane *et al*., 2000; Tully *et al*., 2005; Tully *et al*., 2007), while three days.week^{-1} has proven less beneficial with either no effect on BP (Murtagh *et al*., 2005) or a decrease in SBP only (Tully *et al*., 2007). The current study does not agree with these findings as no significant changes in BP were identified (Figure 37 and Figure 38, page 109). This result may be due to the poor compliance to the exercise guidelines or the small sample size (n=25). However, an almost perfect (r^2=0.9857), although non-significant, relationship for decreasing DBP over time was found showing that even modest participation in physical activity is beneficial. A meta-analysis by Hamer and Chida (2008) found that walking pace was a stronger independent predictor of overall CVD risk compared with walking volume (48% versus 26% risk reduction respectively) and therefore current results may not have been strong due to the low walking intensity which was adopted and is something which needs to be investigated further and especially in this population group. Of interest are the findings of Carnethon *et al*. (2010) who showed both BMI and WC to be significantly inversely associated with categories of fitness and activity. They conclude that the mechanism by which fitness and activity are associated with a lower rate of

developing hypertension, is through lowered BMI (Carnethon *et al*., 2010). Since the BMI in this current study was controlled, this may have represented one of the barriers to BP reduction. A previous study found hunger and energy intake to be unchanged by a bout of walking suggesting that overweight/obese individuals do not acutely compensate for the energy cost of the exercise bout through increased caloric consumption. This allows for an energy deficit to exist post-exercise, having potentially favourable implications for weight control (Unick *et al*., 2010).

The other obesity measures, namely WC and body composition, did not show any beneficial changes as a result of the exercise. Results showed no change in WC over time (Figure 33, page 106) while there was a significant increase in fat mass (Figure 34, page 107) and decrease in lean mass (Figure 35, page 107). These adverse body composition changes are largely unexplained and are more-than-likely due to the inaccuracy of the measurement technique used paired with participant deviation in food and fluid intake prior to the measurement session. Some of the decrease in lean mass may also be due, in part, to the age-related loss of muscle mass known as sarcopenia (Lang *et al*., 2010). Although this is unlikely as the process usually only occurs from the age of 50 years onwards and is slowly progressive (1-2% muscle mass loss per annum) (Bijlsma *et al*., 2013). In support of the WC finding, a pedometer-based walking programme investigated the effect of ≥10000 steps.day^{-1} for eight weeks in overweight women at risk of type II diabetes. Results showed changes in glucose tolerance and BP but no changes in body mass, body fat percentage or WC. These findings highlight the importance of accumulated walking, despite no weight loss, in benefiting this population (Swartz *et al*., 2003). Hornbuckle *et al*. (2012) conducted a similar study in overweight African-American women but extended the study length to 12 weeks. Again, they found no changes in body composition variables. However, when walking plus resistance training (twice per week) was compared to walking only, the former was found to be more effective in increasing strength and decreasing WC, body fat and HbA_{1c}. This prescription may be an important addition to a lifestyle intervention aiming to facilitate reductions in CVD risk factors in overweight and obese African-American women (Hornbuckle *et al*., 2012).

The exercise effect on glycated haemoglobin, the measure for type II diabetes, is important. Results showed an adverse increase in HbA_{1c} (Figure 31, page 104) which was significant only in the CON group. This result is supported by previous studies in individuals with pre-diabetes since exercise training, often in combination with other lifestyle strategies, has beneficial effects on preventing the onset of type II diabetes and improving glycaemic control (Colberg *et al*., 2010; Hordern *et al*., 2012). This suggests that the increase in HbA_{1c} was ameliorated in the EXC group tentatively as a result of their physical activity participation.

Aerobic training increases parasympathetic activity and decreases sympathetic activity in the human heart at rest. These two training-induced autonomic effects, along with a possible reduction in intrinsic heart rate, decreases resting heart rate (Carter *et al*., 2003). The current study did not support this, however, as there were no significant differences in mean resting heart rate (Figure 36, page 108). But, the EXC group did demonstrate a very strong decreasing trend over time which may have been insignificant due to the small sample size and poor compliance. Also, contributing to this non-significant result, this group had a baseline resting heart rate of 82 $bts.min^{-1}$ which is considered 'normal' and so alteration from modest exercise alone may not have been possible. These current findings, showing a trend for decreasing resting heart rate, were matched with a significant increase in fitness level (Figure 39, page 110). This was expected since fitness is a physiological response to activity (Carnethon *et al*., 2010). The large, and significantly greater, increase in fitness by the EXC group in the first four weeks (55.31 m) corresponded with this groups greatest overall compliance to the walking programme; week 1-4 (49.6%), week 5-8 (41.9%) and week 9-12 (43.5%). Thereafter, the average fitness level marginally decreased and plateaued. The CON group progressively increased throughout the study which may be attributable to a 'learning curve' from practice of the six-minute walk test as opposed to a physiological adaptation. It is important to distinguish between aerobic fitness and physical activity since physical activity is a behaviour while aerobic fitness is a physiological measure that reflects a combination of physical activity, genetic potential and functional health of different organ systems (Carnethon *et al*., 2010). Carnethon *et al*. (2010) tested whether fitness and physical activity were independently associated with the 20-year

incidence of hypertension in black and white adults. The incidence of hypertension was inversely associated with fitness category in all of the race and sex groups. By contrast, patterns of association between activity and incident hypertension were less pronounced which highlights fitness as the more important risk factor. The estimated proportion of hypertension cases that could be prevented if participants moved to a higher fitness category was 34% (Carnethon *et al*., 2010). This shows the importance again of performing physical activity at the correct intensities to illicit the most optimal responses.

Finally, biochemical analyses revealed a relatively constant lipid profile (Figure 27, Figure 28, Figure 29 and Figure 30, pages 102 and 103). A meta-analysis of walking programmes lasting at least eight week found significantly reduced LDL-C and TC/HDL-C in adults independent of changes in body composition (Kelley *et al*., 2004). However, the literature around this issue is controversial (Durstine *et al*., 2001). In similarly designed studies, Lian *et al*. (2013) found a significant improvement in all four components of the lipid profile, whereas several other studies found that exercise alone was insufficient to stimulate changes in any lipid or lipoprotein measure (Rad and Gholami, 2010; Chaimongkol and Pratanaphon, 2011; Hornbuckle *et al*., 2012).

5.2.2. Individual Results

It has been reported that while exercise benefits the majority of the population, the response is not always uniform (Bouchard *et al*., 2012). Loenneke *et al*. (2013) in a recent publication entitled “Hypertension risk: exercise is medicine for most but not all” emphasises the point that group mean results often mask individual findings which are important for exercise/treatment prescription. To illustrate, the HERITAGE Family study reported a person-person variability in the BP adaptation to chronic aerobic exercise (Rice *et al*., 2002). Bouchard *et al*. (2012) retrospectively analysed data from 1687 adults from six separate studies and found that 12.2% of the sample studied had an adverse resting SBP response to exercise (+10 mmHg) training. The reason for this is unknown, but is likely attributable to genetics. They suggest that

this heterogeneous exercise response is important to study so that individuals at risk for adverse events can be identified and offered proper guidance for reducing any potential health risk to exercise (Bouchard *et al*., 2012). For this reason, the two main risk factors identified in the comparative study, overweight/obesity and high BP, were further examined from an individual perspective.

From an individual level, compliance in the current study ranged from a high of 68.3% to a low of 11.7% (Table 23, page 112). But, these compliance rates were not related to responses. Although this compliance-response relationship was missing, the individual results were still of interest in terms of comparing the EXC and CON groups despite the lack of significance. Looking at each measure in particular, the BP results from the EXC group revealed a beneficial response as both SBP (Figure 43, page 115) and DBP (Figure 44, page 116) were seen to decrease in the majority of the participants. However, similar findings were seen in the CON group and therefore cannot be attributed to the walking intervention. To a lesser degree, BMI, although controlled within the study, was seen to decrease in the majority of exercising participants (Figure 40, page 112). This decrease was more common and more pronounced in the EXC group compared to the CON group. Conversely, in the EXC group the participant with the lowest compliance had a greater decrease in BMI than the participant with the highest compliance. There were no group differences observed in the WC (Figure 41, page 113) and fat mass (Figure 42, page 114) data.

Buford *et al*. (2013) reported on a study highlighting the fact that some individuals may have a non-existent or negative response to an exercise intervention that is beneficial to other group members, thus supporting this ‘personalised medicine’ approach. In the same vein, individuals can potentially be classified as ‘responders’ or ‘non-responders’ to an exercise training programme which would have huge implications for treatment prescription. Supporting this notion are results from the EXC group in the current study. Participant E19 had the second highest compliance at 64.2% while participant E27 had the second lowest compliance at 26.7%, less than half that of the former. Yet, the health responses do not favour E19 and, in fact, this participant showed an adverse response in terms of BMI (greatest increase),

WC, fat mass and SBP (greatest increase). Conversely, E27 showed very beneficial responses for BMI (greatest reduction), WC, fat mass, SBP (greatest reduction) and DBP (greatest reduction). Since these changes cannot be explained by compliance to the exercise programme, it is likely that genetics may play a role as suggested by Bouchard *et al*. (2012).

5.3. SUMMARY OF THE STATISTICAL HYPOTHESES

1) The first null hypothesis for the comparison of CVD risk factors is mostly rejected. Compared to both the SADHS and the SANHANES-1, there were significant differences in BMI, WC and DBP, with all these variables being higher in the current study. SBP was significantly greater than the SAHDS alone, while heart rate and triglycerides were significantly different, but still considered normal, than the SANHANES-1. Finally, fat mass was significantly higher than the existing norms for this variable.

2) The second null hypothesis is accepted for group effect, time effect and interaction effect. There was no significant change in BP with the walking programme.

3) The third null hypothesis is accepted for group effect, time effect and interaction effect. There was no significant change in WC with the walking programme.

4) The fourth null hypothesis is accepted for group effect and interaction effect. But, there was a significant, although adverse, body composition change over time and therefore the time effect hypothesis is rejected.

5) The fifth null hypothesis is accepted for group effect, time effect and interaction effect. There was no significant change in resting heart rate with the walking programme.

6) The sixth null hypothesis is rejected for group effect, time effect and interaction effect. There was a significant increase in fitness levels, both between and within the groups, with the walking programme.

7) The seventh null hypothesis is mainly accepted for group effect, time effect and interaction effect. However, there was a significant interaction effect for TGs and so part (d) of the interaction effect hypothesis is rejected.

8) The eighth null hypothesis is accepted for the group effect. There was a significant time effect and interaction effect for glycated haemoglobin and so these hypotheses are rejected.

CHAPTER 6

CONCLUSION

6.1. MAIN FINDINGS AND RECOMMENDATIONS

6.1.1. Comparative Study

The comparative study exposed overweight/obesity and high blood pressure (BP) to be the two most concerning cardiovascular disease (CVD) risk factors in this population group. Body mass index (37.6 $kg.m^{-2}$ ±8.64) and waist circumference (1130.58 mm ±176.29) were significantly greater than both the 2003 South African Demographic and Health Survey (SADHS) and the 2013 South African National Health and Nutritional Examination Survey (SANHANES-1). The third measure for overweight/obesity, fat mass (45.23% ±9.47), was above the normal upper limit of 31%. This obesity epidemic is well-documented in black African females, but the higher levels reported in the current study for this Eastern Cape cohort demands attention. A knock-on effect of overweight/obesity, and other modifiable risk factors associated with urbanisation, is raised BP. This is shown in the current study with the mean BP being classified as pre-hypertension with systolic blood pressure (SBP) (130.33 mmHg ±13.56) being significantly higher than the 2003 SADHS, while diastolic blood pressure (DBP) (87.90 mmHg ±10.21) was significantly greater than both previous national surveys. This is of added concern for females whom have previously been thought of as protected against heart disease pre-menopause, leading to poor awareness, detection and treatment for high BP. Black Africans specifically have the second worst hypertension treatment status of all South African population groups. South Africa, and in particular the Eastern Cape, is a developing region which struggles to cope with its overburdened health situation and so these two CVD risk factors, overweight/obesity and high BP, require cost-effective approaches for their successful prevention and treatment. These approaches primarily involve lifestyle changes of which physical activity is highly recommended, although poorly researched in an African context. Baseline measures showed this cohort to be ‘active’ according to walking volume (10162 $steps.day^{-1}$) but the average

walking intensity of 19.42 mins.day^{-1} was below the recommended 30 mins.day^{-1} required to illicit health benefits.

6.1.2. Intervention Study

The 12 week progressive walking programme did not significantly ($p \leq 0.05$) alter BP in the sample of black African females. It did, however, positively affect DBP which showed a strong negative relationship ($r^2=0.9857$) with time with a total average decrease of 4.47 mmHg. The lack of significance could be due to the small sample size of the exercise (EXC) group (n=13) and/or the poor compliance to the individual walking guidelines. Recommendations for the future are therefore different walking strategies such as group-based sessions or supervised sessions to improve compliance. Future research may also want to focus on levels of compliance during the intervention to see if these improve, worsen or remain the same as the exercise programme progresses. It was observed (although not specifically analysed) through the GPAQ responses that energy was expended primarily for transport reasons which identifies a possible avenue for future exercise interventions to target. Further, a common reason for non-compliance was “lack of time” which can be addressed in subsequent walking interventions by adopting higher walking intensities which require less time. The World Health Organisation recommends the practice of both aerobic and resistance training to decrease high BP and so this combination technique should be researched in this population group. Lastly, since this group is on average obese class II, it is important that this obesity epidemic also be addressed. An exercise and diet programme for weight loss could be an important approach to combat this issue and at the same time decrease BP since these risk factors are directly related. Another beneficial effect of the walking programme was a delay in the rise of glycated haemoglobin. The control (CON) group saw a significant increase in HbA_{1c} of 0.49% while the EXC group increased by only 0.15%. This is an important finding as baseline measures from the comparative study placed this sub-population at an ‘increased risk for diabetes’ based on the HbA_{1c} measure. Lastly, although not significant, there was a strong negative trend ($r^2=0.9228$) for decreasing resting heart rate as the walking programme progressed. Resting heart rate is often used as a fitness indicator and so this is a positive finding of the intervention study.

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APPENDIX 1

ETHICAL APPROVAL

Human Kinetics and Ergonomics Ethics Committee Report

RHODES UNIVERSITY
Where leaders learn

Student Name: Tegan Crymble

Type of Research: Masters Research Project

Project Title: Efficacy of a 12 week walking programme on blood pressure in previously sedentary Black African females.

Supervisor: Dr Christie

Application received: 06 February 2013

Report Compiled: 11 March 2013

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

Remarks:

Only a few minor concerns remain as highlighted below:

No mention of research assistants in letters (privacy and confidentiality)
-Need to clarify that normal diet + exercise (in previously sedentary people) will probably result in weight loss. But require participants to try maintain weight?
.

I wish you luck with your research!

Signed

Al Todd
Chair: Human Kinetics and Ergonomics Ethics Committee

Confidential
HKE Ethical Committee Review Form

APPENDIX 2

RECRUITMENT PAMPHLET

MASTERS RESEARCH

Walking is a low cost and low risk exercise that most people can perform, either alone or in groups. It is often recommended for preventing health problems.

This research will look at the effects of a 12 week walking programme on blood pressure. You will be asked to walk *briskly* on at least 5 days of the week *without* supervision. You will be given step 'goals' to reach each day which will be monitored with a pedometer (small device that counts steps) that you wear on your belt/waistband.

Research Funding: Medical Research Council (MRC)
Researcher Funding: National Research Foundation (NRF); Ernst and Ethel Eriksen Trust

SOUTH AFRICAN MEDICAL RESEARCH COUNCIL

National Research Foundation

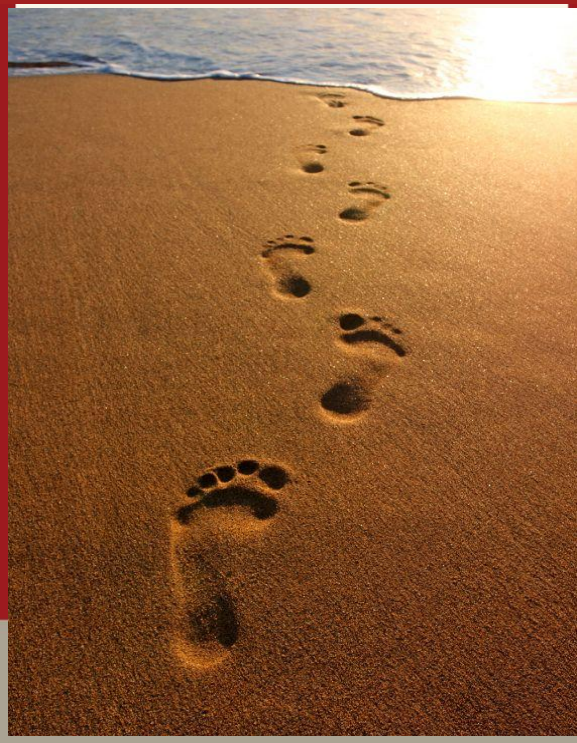
ERNST & ETHEL ERIKSEN TRUST

Principal Researcher:
Tegan Crymble
Phone | 079 482 2032
E-mail | tegan.777@gmail.com

Supervisor:
Dr Candice Christie
Phone | 046 603 8470
E-mail | c.christie@ru.ac.za

WALKING PROGRAMME

12 WEEKS LONG



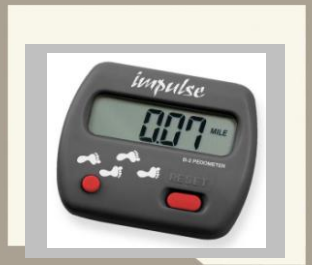
"WALKING GETS THE FEET MOVING, THE BLOOD MOVING, THE MIND MOVING. AND MOVEMENT IS LIFE…"

~ CARRIE LATET

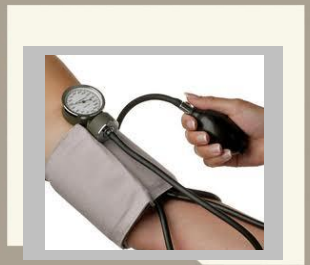
WALKING PROGRAMME
12 WEEKS LONG

Ethically approved by the Rhodes University Human Kinetics and Ergonomics Ethical Standards Committee

Department of Human Kinetics and Ergonomics



PEDOMETER



BP

HEALTHY

HAPPY

MEASUREMENTS:

- Stature and body mass
- Lean and fat mass, waist circumference
- Heart rate, blood pressure
- Step count, fitness, energy expenditure
- Diet, blood sugar, blood fats

CRITERIA

DO YOU WANT A *FREE* WALKING PROGRAMME AND HEALTH TESTING?

TO TAKE PART YOU MUST BE:

- Female
- Black African
- Age 25-44 years
- Able to read and write English
- Non-smokers (not smoked in 1 year)
- Not heavy drinkers (no more than 3 alcoholic drinks per day)
- Sedentary (you do not exercise a lot)
- No injury to bones and muscles
- Healthy (no heart, lung, liver, kidney disease)
- Still menstruating (you have your period)
- Not pregnant or breast feeding
- Not using drugs or other agents

BENEFITS OF TAKING PART

- Improved health and wellness
- Increased energy levels
- Decreased risk of heart disease
- Physical examination by a doctor
- Blood tests to see if your blood sugar and/or blood fat levels are too high
- Information about your own body
- Help with important research

RISKS OF TAKING PART

- Uncomfortable/embarrassed when walking and during the measurement sessions
- Injury or pain during walking
- Walking alone can be unsafe

SIGN UP IF INTERESTED

~ Let's get healthy!

APPENDIX 3

RECRUITMENT POSTER

WALKING PROGRAMME
12 WEEKS LONG

Ethically approved by the Rhodes University Human Kinetics and Ergonomics Ethical Standards Committee

Be part of a fun and easy exercise programme which will get you walking and keep you healthy!

Organised through the Human Kinetics and Ergonomics (HKE) Department

BACKGROUND

- Masters' research
- Exercise is a great way to improve your health
- Walking is a safe and simple exercise
- Walking is good for your heart, mind, and body
- Fun and social
- Doesn't cost anything!

TO TAKE PART YOU MUST BE:

- Female
- Black African
- 25-44 years old
- Able to read and write English
- Non-smokers (not smoked in 1 year)
- Not heavy drinkers (no more than 3 alcoholic drinks per day)
- You do not exercise a lot
- No injury to bones and muscles
- Healthy (no heart, lung, liver, kidney disease)
- You still have your period
- Not pregnant or breast feeding
- Not using drugs or other agents

WHAT YOU WILL DO

The walking programme is 12 weeks long and starts off very easy and gets a bit harder every 4 weeks.

You must follow this programme which will have step 'goals' which you must reach by walking *briskly* and *without* supervision. You will be able to see how much you walk with a small device which you will wear on your belt/waistband.

You must record your step count which will be shown on this device at the end of every day. You will have measurements done at the start of the programme and then every 4 weeks until the end.

MEASUREMENTS TAKEN AT WEEKS 0, 4, 8, 12:

- Stature and body mass
- Lean mass and fat mass
- Waist circumference
- Heart rate, blood pressure, fitness
- Step count
- Energy expenditure, diet, blood sugar, blood fats

WHAT YOU WILL GET

- Improved health
- Free physical exam by a doctor
- Free blood tests to see if your blood sugar and blood fats are too high
- Juice and biscuits at measurement sessions ☺

This research is funded by:

Medical Research Council

National Research Foundation

ERNST & ETHEL ERIKSEN TRUST

Contact Information: Tegan Crymble | phone: 079 482 2032 | email: tegan.777@gmail.com

APPENDIX 4

INFORMATION LETTER – CONTROL GROUP

Introduction:

Thank you for wanting to be part of this. You are at the introductory meeting where I (Tegan) will talk about the study and allow you to ask questions. Please ask if you do not understand. At the end you must sign a consent form if you want to take part.

Screening:

To do this study you must go through 6 levels to see that you fit the criteria.

1st level: fill in a 'Participant Screening' form which will ask you about age, race, health etc. You will have your height, weight, fat, muscle, waist, heart rate and blood pressure measured. This is to see your starting measures.

2nd level: fill in three questionnaires. The questionnaires are about alcohol use, exercise and medication use.

3rd level: have a physical exam by a doctor to make sure you are safe to exercise.

4th level: you will be given a day and time to go to 'Du Buisson & Partners' in Peppergrove Mall to have a blood test. You must not eat anything 12 hours before the blood test. This test will measure your blood fats and blood sugar.

5th level: do a small fitness test. You must walk up and down a 50 metre field as many times as possible in 6 minutes.

6th level: you must wear a device which counts your steps (called a 'pedometer') for 1 week so I can see your usual activity level. You must not change your usual activity - go about your day as normally as possible.

Control:

You are asked to keep the way you normally eat for the 12 week duration. You must try your best to not lose any weight or put on any weight during this time. To check this you will fill in a 3 day (Tuesday, Thursday and Saturday) dietary recall each month. This will not take you much time.

Acceptance into study:

After this, if you fit the criteria, you will be asked to take part. If so, you will be in the control (CON) group which is based on the res/hall you work in.

What does the CON group do?

The CON group does not take part in the walking. You must go about your life normally but your daily steps will be measured with a 'pedometer'. You must wear this pedometer every day and write down how many steps you take (shown on the 'pedometer' screen) in a diary at the end of each day. You will have measurements taken every 4 weeks.

Measurements:

Before you start the study, and every 4 weeks after, all of the listed measures will be taken as seen in the table below. These will be taken at meetings where you will have the chance to talk about the study and ask questions.

MEASURE	RESULT	HOW TO MEASURE
Anthropometry	Height	Stadiometer
	Weight	Scale
Body Composition	Body fat and muscle mass	Body composition machine
	Waist circumference	Tape measure
Cardiovascular	Heart rate	Feel and count
	Blood pressure	Blood pressure meter
	Fitness	Walking fitness test
Physical Activity	Step count	Pedometer
Control	Normal diet	3 day dietary recall
	Energy used	Questionnaire
	Blood sugar and blood fats	Fasting blood test

Risks:

Since you will not be doing the walking, the risks may occur during the measurements. All risks are small and will not last long. When looking at fat and muscle, the machine sends a very small electrical current through your body - this will not be felt. Alcohol swabs clean your skin and this may cause allergic reactions and rashes - I will ask you about any allergies before. Sticky pads will be put on your skin and may cause pain when I remove them.

For blood pressure a cuff goes around your left arm and I pump it up with air. This will be tight and may be sore for about 1 minute. This is a short pain and it will go away as soon as I let the air out. For the fitness test you will need to walk up and down a field as many times as possible in 6 minutes - this might make you feel sick and tired but you can stop and rest if you need to. Blood tests will be taken to see

your blood fats and blood sugar. Blood will be taken by a nurse and this may be sore.

Benefits:

You will have lots of measures done (free of charge) including a check-up with a doctor and blood tests. These tests will tell you about your health and own body. Also, you will be involved in a science environment which may help you learn about health in general.

More information:

To help with the measures there will be female assistants. They are HKE students and will make sure results are kept private. You may be asked to have your photograph taken to be used in this study. This is your choice and you may say ‘no’ if you do not want this. If you say ‘yes’ your privacy will be kept as your features will be blocked out. Also, individual codes will be used and not your name.

Results:

I can’t talk to you about your results during the study as this may change things. But, once you have finished, results for the group will be given. You can ask for your own results which I will give to you in private.

Questions?

Questions are encouraged. Feel free to come to me (Tegan Crymble) or my supervisor (Dr Candice Christie) at any stage. Contact details are below.

Thank you for wanting to take part!

Principal Researcher:

Tegan Crymble

079 482 2032

tegan.777@gmail.com

Supervisor:

Dr Candice Christie

046 603 8470

c.christie@ru.ac.za

APPENDIX 4

INFORMATION LETTER – EXERCISE GROUP

Introduction:

Thank you for wanting to be part of this. You are at the introductory meeting where I (Tegan) will talk about the study and allow you to ask questions. Please ask if you do not understand. At the end you must sign a consent form if you want to take part.

Screening:

To do this study you must go through 6 stages to see that you fit the criteria.

1st level: fill in a 'Participant Screening' form which will ask you about age, race, health etc. You will have your height, weight, fat, muscle, waist, heart rate, and blood pressure measured. This is to see your 'starting' measures.

2nd level: fill in three questionnaires. The questionnaires are about alcohol use, exercise, and medication use.

3rd level: have a physical exam by a doctor to make sure you are safe to exercise.

4th level: you will be given a day and time to go to 'Du Buisson & Partners' in Peppergrove Mall to have a blood test. You must not eat anything 12 hours before the blood test. This test will measure your blood fats and blood sugar.

5th level: do a small fitness test. You must walk up and down a 50 metre field as many times as possible in 6 minutes.

6th level: you must wear a device which counts your steps (called a 'pedometer') for 1 week so I can see your usual activity level. You must not change your usual activity - go about your day as normally as possible.

Control:

You are asked to keep the way you normally eat for the 12 week duration. You must try your best to not lose any weight or put on any weight during this time. To check this you will fill in a 3 day (Tuesday, Thursday, and Saturday) dietary recall each month. This will not take you much time.

Acceptance into study:

After this, if you fit the criteria, you will be asked to take part. If so, you will be in the exercise (EXC) group which is based on the res/hall you work in.

What does the EXC group do?

The EXC group will take part in the walking. You will start a walking programme which will go for 12 weeks. You will wear a 'pedometer' which will count how many steps you take each day - this number must be written into a diary at the end of every day. Every 4 weeks, the step 'goal' will get bigger making the walking programme a little bit harder. To make sure that you are safe during the walking you must tell me about your health as well as any bad things you may feel during walking (such as lack of breath, pain). All information will be secret and will not be shared with employers or with Rhodes University.

Measurements:

Before you start the study, and every 4 weeks after, all of the listed measures will be taken as seen in the table below. These will be taken at meetings where you will have the chance to talk about the study and ask questions.

Risks:

You will be doing the walking on your own which can be a risk to your safety. But, walking is low risk and most people are able to do it easily. The walking will slowly get harder which lets you get used to it so you are at less risk of hurting yourself. During the walking programme, a doctor and I will be on call in case there is an emergency.

During the walking you may feel:

- Embarrassed: you will be walking by yourself and during your own time and place to make you feel better. Walking is a normal exercise in all communities and has positive effects.
- Trip or fall accidents: you must try wearing proper shoes (such as sports trainers or closed shoes with a rubber soles.

MEASURE	RESULT	HOW TO MEASURE
Anthropometry	Height	Stadiometer
	Weight	Scale
Body Composition	Body fat and muscle mass	Body composition machine
	Waist circumference	Tape measure
Cardiovascular	Heart rate	Feel and count
	Blood pressure	Blood pressure meter
	Fitness	Walking fitness test
Physical Activity	Step count	Pedometer
Control	Normal diet	3 day dietary recall
	Energy used	Questionnaire
	Blood sugar and blood fats	Fasting blood test

- Injury: risk of injury to the feet, ankles, and knees.
- Light-headedness, shortness of breath, and dizziness when you get tired: if this happens you must stop walking and take deep breaths until the feeling has gone (can have sips of cold water). If you keep feeling these things you must let me know.
- Muscle cramping: I will show you some stretching you can do before you start walking to avoid this. But, if you do get muscle cramps during walking you must stop and stretch the area until the cramp has gone.
- Delayed onset muscle soreness (DOMS): this is normal and lasts about 4 days with the first 2 days being the sorest. If the muscle soreness does not go away then you must let me know.
- Walking area: women walking alone can be unsafe and so you must always walk in areas where people are and during the day (not night).

There may be risks during the measurements. All risks are small and will not last long. When looking at fat and muscle, the machine sends a very small electrical

current through your body - this will not be felt. Alcohol swabs clean your skin and this may cause allergic reactions and rashes - I will ask you about any allergies before. Sticky pads will be put on your skin and may cause pain when I remove them.

For blood pressure a cuff goes around your left arm and I pump it up with air. This will be tight and may be sore for about 1 minute. This is a short pain and it will go away as soon as I let the air out.

For the fitness test you will need to walk up and down a field as many times as possible in 6 minutes - this might make you feel sick and tired but you can stop and rest if you need to.

Blood tests will be taken to see your blood fats and blood sugar. Blood will be taken by a nurse and this may be sore.

Benefits:

By doing exercise you will get healthy and also have more energy. This may help your heart and give you healthy bones, muscles, and joints. You will have lots of measures done (free of charge) including a check-up with a doctor and blood tests. These tests will tell you about your health and own body. Also, you will be involved in a science environment which may help you learn about health in general.

More information:

To help with the measures there will be female assistants. They are HKE students and will make sure results are kept private. You may be asked to have your photograph taken to be used in this study. This is your choice and you may say ‘no’ if you do not want this. If you say ‘yes’ your privacy will be kept as your features will be blocked out. Also, individual codes will be used and not your name.

Results:

I can’t talk to you about your results during the study as this may change things. But, once you have finished, results for the group will be given. You can ask for your own results which I will give to you in private.

Questions?

Questions are encouraged. Feel free to come to me (Tegan Crymble) or my supervisor (Dr Candice Christie) at any stage. Contact details are below.

Thank you for wanting to take part!

Principal Researcher:
Tegan Crymble
079 482 2032
tegan.777@gmail.com

Supervisor:
Dr Candice Christie
046 603 8470
c.christie@ru.ac.za

APPENDIX 5

CONSENT FORM

RHODES UNIVERSITY
DEPARTMENT OF HUMAN KINETICS AND ERGONOMICS
Phone: 079 482 2032 • E-mail: g08c0994@campus.ru.ac.za

I,_____, willingly consent to take part in this study called: **"EFFICACY OF A 12 WEEK WALKING PROGRAMME ON BLOOD PRESSURE IN PREVIOUSLY SEDENTARY BLACK AFRICAN FEMALES".** I have been told about the study and the possible risks and benefits that might happen. This has been given to me by the primary researcher both talking and in writing.

By agreeing to take part in this study, I give up any legal action against the researcher, the Department of Human Kinetics and Ergonomics, and Rhodes University if any injury happens during testing. I understand that I take full responsibility of any costs involved with any injury. I know that the Department of Human Kinetics and Ergonomics will take no responsibility if the injury is self-inflicted or as a result of lack of care by the participant. I will tell the researcher straight away if I feel any problems during the walking or testing. I know that I am not forced to take part and that if I do take part, I can leave the study at any time and for any reason.

I know that my secrecy will be protected at all times and agree to allow the information collected to be used for scientific purposes. I know that I may be asked to have photographs taken during the testing phase, but I must first say 'yes' to this and that recognisable features will be blocked out. I am happy to have the different variables recorded during the testing phases by the principal researcher and female research assistants. I have read and understood the information above as well as the other information about the study and I am happy that all of my questions have been answered.

Signed at _____, Rhodes University

PARTICIPANT

_____	_____	_____
(Print name)	(Signed)	(Date)

RESEARCHER

_____	_____	_____
(Print name)	(Signed)	(Date)

WITNESS 1

_____	_____	_____
(Print name)	(Signed)	(Date)

WITNESS 2

_____	_____	_____
(Print name)	(Signed)	(Date)

APPENDIX 6

PARTICIPANT SCREENING FORM

Please indicate your SEX (male or female):	
Are you a BLACK AFRICAN (race)?	
Please give your AGE in years and months:	
Are you able to read, write, and understand basic ENGLISH?	
Have you had any MAJOR SURGERY in the last 6 months?	
Have you ever been told that you have DIABETES (high blood sugar) or HIGH CHOLESTEROL (high blood fats)?	
Do you have any HEART problems?	
Do you have any LUNG (or related breathing) problems or difficulties?	
Do you have any LIVER problems?	
Do you have any KIDNEY problems?	
Do you SMOKE? (Currently or during the past 12 months)	
Do you drink ALCOHOLIC drinks often (3 or more drinks per day)?	
Do you do any “formal” EXERCISE?	
Do you have any MUSCULOSKELETAL injuries? i.e. damage of muscles or bones	
Have you taken, or are you taking, any form of ANTIHYPERTENSIVE MEDICATION?	

Do you still have your MENSTRUAL CYCLE (period)?	
Are you currently PREGNANT and/or BREAST FEEDING?	
Are you currently on any form of CONTRACEPTIVE MEDICATION?	
Are you currently on any other MEDICATION that the researcher should know about? (e.g. ARV's)	

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

_____ _____

Signed by participant Date

APPENDIX 7

MONTHLY DATA SHEET

HEIGHT (mm)	
WEIGHT (kg)	
BMI ($kg.m^{-2}$)	

WAIST CIRCUMFERENCE (mm)	
BODY FAT (kg)	
BODY FAT (%)	
LEAN BODY MASS (kg)	
LEAN BODY MASS (%)	

HR 1 ($bt.min^{-1}$)	
HR 2 ($bt.min^{-1}$)	
HR 3 ($bt.min^{-1}$)	
RESTING HR ($bt.min^{-1}$)	

SBP 1 (mmHg)	
DBP 1 (mmHg)	
SBP 2 (mmHg)	
DBP 2 (mmHg)	
SBP 3 (mmHg)	
DBP 3 (mmHg)	
RESTING BP (mmHg)	

LAST MEAL EATEN?	Less than 2 hours ago	More than 2 hours ago

APPENDIX 8

CAGE QUESTIONNAIRE

Alcohol Dependence: CAGE Questionnaire			
		Yes (1)	No (2)
1.	Have you ever thought that you should **C**ut down on your drinking?		
2.	Have people **A**nnoyed you by criticizing your drinking?		
3.	Have you ever felt **G**uilty about your drinking?		
4.	Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (**E**ye opener)?		

APPENDIX 9

GLOBAL PHYSICAL ACTIVITY QUESTIONNAIRE

I am going to ask you about the time you spend doing different types of PA in a typical week. Please answer these questions even if you do not consider yourself to be a physically 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, study/training, household chores, harvesting food/crops, fishing or hunting food, seeking employment. In answering the following questions 'vigorous-intensity activities' are activities that require hard physical effort and cause large increase in breathing or HR, 'moderate-intensity activities' are those that require moderate physical effort and cause small increase in breathing or HR.

	QUESTIONS	RESPONSE	CODE
	Activity at work		
1	Does your work involve vigorous-intensity activity that causes large increases in breathing or HR (such as carrying or lifting heavy loads, digging or construction work) for at least 10 mins continuously?	Yes 1 No 2 *If No, go to P4*	P1
2	In a typical week, on how many days do you do vigorous-intensity activities as part of your work?	Number of days ____	P2
3	How much time do you spend doing vigorous-intensity activities at work on a typical day?	Hours ____ Minutes ____	P3 (a-b)
4	Does your work involve moderate-intensity activity that causes small increases in breathing or HR such as brisk walking (or carrying light loads) for at least 10 mins continuously?	Yes 1 No 2 *If No, go to P7*	P4
5	In a typical week, on how many days do you do moderate-intensity activities as part of your work?	Number of days ____	P5
6	How much time do you spend doing moderate-intensity activities at work on a typical day?	Hours ____ Minutes ____	P6 (a-b)

The next questions exclude the physical activities at work that you have already mentioned.

Now I would like to ask you about the usual way you travel to and from places. For example to work, for shopping, to market, to place of worship.

	QUESTIONS	RESPONSE	CODE
Travel to and from places			
7	Do you walk or use a bicycle for at least 10 mins continuously to get to and from places?	Yes 1 No 2 *If No, go to P10*	P7
8	In a typical week, on how many days do you walk or bicycle for at least 10 mins continuously to get to and from places?	Number of days ____	P8
9	How much time do you spend walking or bicycling for travel on a typical day?	Hours ____ Minutes ____	P9 (a-b)

The next questions exclude the work and transport activities that you have already mentioned.

Now I would like to ask you about sports, fitness and recreational activities (leisure).

	QUESTIONS	RESPONSE	CODE
Recreational activities			
10	Do you do any vigorous-intensity sports, fitness or leisure activities that cause large increases in breathing or HR, like running, for at least 10 mins continuously?	Yes 1 No 2 *If No, go to P13*	P10
11	In a typical week, on how many days do you do vigorous-intensity sports, fitness, or leisure activities?	Number of days ____	P11
12	How much time do you spend doing vigorous-intensity sports, fitness or leisure activities on a typical day?	Hours ____ Minutes ____	P12 (a-b)
13	Do you do any moderate-intensity sports, fitness or leisure activities that cause small increases in breathing or HR such as brisk walking (cycling, swimming, volleyball) for at least 10 mins continuously?	Yes 1 No 2 *If No, go to P16*	P13

14	In a typical week, on how many days do you do moderate-intensity sports, fitness or leisure activities?	Number of days ____	P14
15	How much time do you spend doing moderate-intensity sports, fitness or leisure activities on a typical day?	Hours ____ Minutes ____	P15 (a-b)

The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent sitting at a desk, sitting with friends, travelling in car, bus, train, reading, playing cards or watching TV, but DO NOT include time spent sleeping.			
QUESTIONS		**RESPONSE**	**CODE**
Sedentary behaviour			
16	How much time do you usually spend sitting or reclining on a typical day?	Hours ____ Minutes ____	P16 (a-b)

APPENDIX 10

MEDICATION QUESTIONNAIRE

I want to ask you about any medication you take.		
Do you use any medicine regularly or daily that a doctor or nurse has prescribed?	YES..... NO..... DON'T KNOW.....	} Skip
How many different medicines do you use regularly (more than once a month)?	Number of medicines: _____	

Record all the drugs mentioned and what they are taken for:

NAME OF DRUG	WHAT IS IT FOR?													
	High blood pressure	Heart attack or angina	Stroke	High cholesterol or other blood fats	Diabetes, blood sugar	Emphysema, Bronchitis	Asthma	Sore joints (arthritis)	Osteoporosis	TB	Epilepsy	Cancer	Don't know	Other

APPENDIX 11

SIX-MINUTE WALK TEST

Equation for predicted 6MWT value in healthy women (Enright and Sherrill, 1998):
6MWT = (2.11 x height(cm)) – (2.29 x weight (kg)) – (5.78 x age) + 667m
*Subtract 139m to obtain the lower limit of the normal range for that participant

Date: _____

Distance covered in 6 minutes: _____

Date: _____

Distance covered in 6 minutes: _____

Date: _____

Distance covered in 6 minutes: _____

Date: _____

Distance covered in 6 minutes: _____

APPENDIX 12

STEP COUNT DIARY – CONTROL GROUP

CODE: _____

Week 0 = Habituation Week

*You must not alter your usual physical activity patterns for the entire 12 week duration of this study as I am trying to determine your 'normal' activity patterns. Go about your day as normally as possible please ☺

MVPA = moderate-vigorous physical activity

DATE	WEEK 0	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

Week 1-4

DATE	WEEK 1	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 2	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 3	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 4	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

Week 5-8

DATE	WEEK 5	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 6	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 7	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 8	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

Week 9-12

DATE	WEEK 9	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 10	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 11	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 12	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

FINISHED!! ☺

Thank you very much for your participation in this Masters research.

Tegan Crymble

079 482 2032

MSc Scholar

Human Kinetics and Ergonomics Department, Rhodes University

APPENDIX 12

STEP COUNT DIARY – EXERCISE GROUP

CODE: _____

Week 0 = Habituation Week

*You must not alter your usual physical activity patterns for this first habituation week as I am trying to determine your baseline step count. Go about your day as normally as possible please ☺

MVPA = moderate-vigorous physical activity

DATE	WEEK 0	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

Week 1-4

For the next 4 weeks your step goal = _____ per day for at least 5 days of the week.

DATE	WEEK 1	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 2	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 3	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 4	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

Week 5-8

For the next 4 weeks your step goal = _____ per day for at least 5 days of the week.

DATE	WEEK 5	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 6	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 7	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 8	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

Week 9-12

For the next 4 weeks your step goal = _____ per day for at least 5 days of the week.

DATE	WEEK 9	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 10	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 11	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

DATE	WEEK 12	TOTAL STEP COUNT	TOTAL MVPA TIME
	MONDAY		
	TUESDAY		
	WEDNESDAY		
	THURSDAY		
	FRIDAY		
	SATURDAY		
	SUNDAY		

FINISHED!! ☺

Thank you very much for your participation in this Masters research.

Tegan Crymble

079 482 2032

MSc Scholar

Human Kinetics and Ergonomics Department, Rhodes University

APPENDIX 13

LABORATORY ACCREDITATION

sanas
South African National Accreditation System

CERTIFICATE OF ACCREDITATION

In terms of section 22(2) (b) of the Accreditation for Conformity Assessment, Calibration and Good Laboratory Practice Act 2006 (Act 19 of 2006), read with sections 23(1), (2) and (3) of the said Act, I hereby certify that

AMPATH NATIONAL LABORATORIES
DRS DU BUISSON, KRAMER, SWART, BOUWER INC
GRAHAMSTOWN LABORATORY
Co. Reg. No.: 2007/018337/21
Practice No.: 052 000 5200431

Facility Accreditation Number: **M0380**

is a South African National Accreditation System accredited laboratory provided that all SANAS conditions and requirements are complied with

This certificate is valid as per the scope as stated in the accompanying schedule of accreditation, Annexure "A", bearing the above accreditation number for

MEDICAL TESTING LABORATORY
CHEMISTRY, HAEMATOLOGY AND MICROBIOLOGY

The facility is accredited in accordance with the recognised International Standard

ISO 15189:2007

The accreditation demonstrates technical competency for a defined scope and the operation of a laboratory quality management system

While this certificate remains valid, the Accredited Facility named above is authorised to use the relevant SANAS accreditation symbol to issue facility reports and/or certificates

Mr R Josias
Chief Executive Officer
Effective Date: 01 March 2013
Certificate Expires: 28 February 2017

APPENDIX 14

THREE DAY DIETARY RECALL

You need to remember what have eaten or drunk over the 24 hour period. You need to be as correct as possible about how much i.e. sizes and how you prepared the food or drink.

Important:

1. Write down the time you ate or drank, what you ate or drank (including how it was made) and how much you ate or drank (the portion size).
2. Tell me everything you have eaten or drunk from when you woke up until you went to sleep that evening.
3. Tell me when you ate the food (i.e. lunch, tea, after dinner) and give me the time.
4. Tell me how much you ate or drank (portion size list attached) – such as an apple which was the size of my fist, or a piece of cheese which was the size of my thumb, or a piece of meat the size of the palm of my hand or the size of my hand.
5. Tell me how you made the food (i.e. the skinless chicken was cooked in oil – then you need to tell me how much oil). Everything that has gone into the making of your food must be put down on the list and you must give me an idea of how much.
6. If you eat a sandwich you must remember how much margarine/butter you used and what was on the sandwich.
7. If you drink any liquid you must let me know how much; e.g. coffee mug or a tea cup.
8. If you put milk in coffee or tea you must indicate how much and was the milk full cream or low fat?
9. Sugar added to food or drink must also be included.

Example: Breakfast (07h15): 1x small cup filter coffee (plunger) with 50ml milk (2%)
1x large whole meal rusk (Ouma Brand) with raisins

Example: Lunch (13h00): Two thick-cut slices brown bread, butter spread (Flora light), peanut butter (two dessert spoons)
One small yoghurt (plain, low fat, 250ml)

Today's Date:_____(Day/Month/Year) **Day of the week: Tuesday**

Is this a typical day? ☐ *(If not, give an example of a typical day after this record)*

Time	Type of food and/or drink and how you made it	Quantity
BEFORE BREAKFAST		
BREAKFAST		
MID-MORNING: Between breakfast and lunch		
LUNCH		
MID-AFTERNOON: Between lunch and dinner		
DINNER		
AFTER DINNER		

Today's Date:_____(Day/Month/Year) **Day of the week: Thursday**

Is this a typical day? ☐ *(If not, give an example of a typical day after this record)*

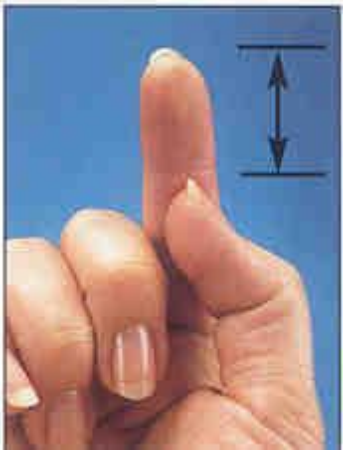
Time	Type of food and/or drink and how you made it	Quantity
BEFORE BREAKFAST		
BREAKFAST		
MID-MORNING: Between breakfast and lunch		
LUNCH		
MID-AFTERNOON: Between lunch and dinner		
DINNER		
AFTER DINNER		

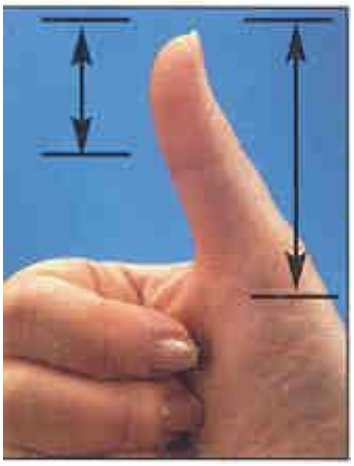
Today's Date:_____(Day/Month/Year) **Day of the week: Saturday**

Is this a typical day? ☐ *(If not, give an example of a typical day after this record)*

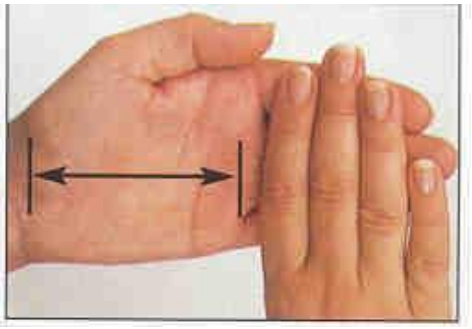
Time	Type of food and/or drink and how you made it	Quantity
BEFORE BREAKFAST		
BREAKFAST		
MID-MORNING: Between breakfast and lunch		
LUNCH		
MID-AFTERNOON: Between lunch and dinner		
DINNER		
AFTER DINNER		

PORTION SIZES

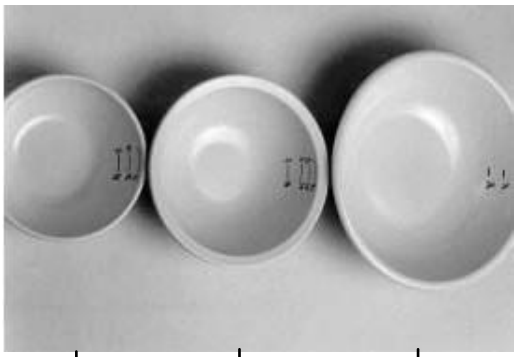


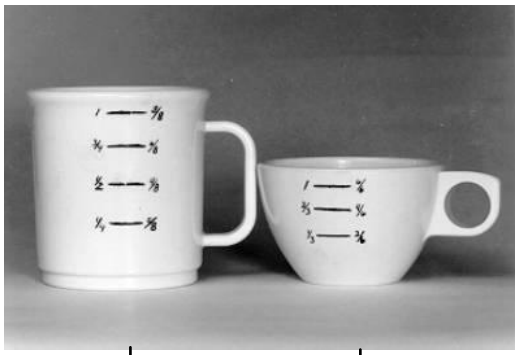


The size of your fist refers to a medium sized fruit or vegetable (i.e. apple, orange, potato) the size of your fingertip refers to one teaspoon (i.e. sugar, butter on bread)
The size of your thumb refers to one tablespoon



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 15

PHYSICAL EXAMINATION CHECK-LIST

ASPECT TO EXAMINE	MEDICAL PRATICITIONER'S COMMENT AND RECOMMENDATIONS
Apical pulse rate and rhythm	
Resting blood pressure: seated, supine and standing	
Auscultation of lungs [note: specific attention to absence of wheezes and other sounds]	
Auscultation of carotid, abdominal and femoral arteries	
Evaluation of abdomen for tenderness	
Palpation and evaluation of lower extremities for oedema and presence of arterial pulses	
Absence or presence of tendon xanthoma or skin xanthelasma	
Follow up examination of orthopaedic/other medical conditions that would limit exercise testing	
Tests of neuralgic function, including reflexes	
Inspection of the skin, especially lower extremities in known diabetics [note: no subject for the purposes of this study may be diabetic]	
SIGNED: Dr CP Jameson	

APPENDIX 16

FULL BLOOD LIPID PROFILE ANALYSIS

Total Cholesterol

Intended Use

The 'CHOL' method used the Dimension® clinical chemistry system which is an *in vitro* diagnostic test intended for the quantitative determination of total cholesterol in human serum and plasma. This method made use of Flex® reagent cartridge. Serum and plasma were collected using recommended procedures for collection and diagnostic blood specimens by venipuncture.

Principles of Procedure

Cholesterol esterase catalysed the hydrolysis of cholesterol esters to produce free cholesterol, which, along with pre-existing free cholesterol, was oxidised in a reaction catalysed by cholesterol oxidase to form cholest-4-ene-3-one and hydrogen peroxide. In the presence of horseradish peroxidise, the resultant hydrogen peroxide was used to oxidise N,N diethylaniline-HCl/4-aminoantipyrine which produced a chormophore that absorbed at 540 nm. This absorbance was directly proportional to the total cholesterol concentration and was measured using a polychromatic (452, 540, 700nm) endpoint technique.

Specimen Collection and Handling

It is important to note that complete clot formation took place prior to centrifugation. Also, storage adhered to recommendations that the specimens were stable for eight hours at room temperature, two days at 2-8ºC, or frozen at -20ºC for longer storage. The specimens were not repeatedly thawed and re-frozen.

Triglycerides

Intended Use

The ‘TGL’ method used on the Dimension® clinical chemistry system is an *in vitro* diagnostic test intended for the quantitative determination of triglycerides in human serum and plasma. Measurements obtained are used in the diagnosis and treatment of patients with diabetes mellitus, nephrosis, liver obstruction, other diseases involving lipid metabolism, or various endocrine disorders. This method made use of Flex® reagent cartridge. Serum and plasma were collected using recommended procedures for collection and diagnostic blood specimens by venipuncture.

Principles of Procedure

The samples were incubated with lipoprotein lipase enzyme reagent that converts triglycerides into free glycerol and fatty acids. Glycerol kinase catalysed the phosphorylation of glycerol by adenosine-5-triphosphate (ATP) to glycerol-3-phosphate. Glycerol-3-phosphate-oxidase oxidised glycerol-3-phosphate to dihydroxyacetone phosphate and hydrogen peroxide (H_2O_2). The catalytic action of peroxidise formed quinoneimine from H_2O_2 aminoantipyrine and 4-chlorophenol. The change in absorbance due to the formation of quinoneimine was directly proportional to the total amount of glycerol in the sample and was measured using a bichromatic (510, 700 nm) endpoint technique.

Specimen Collection and Handling

It is important to note that blood collection tubes did not have glycerol lubricated stoppers to prevent an erroneous result. Also, storage adhered to recommendations that the separated specimens were stable for eight hours at room temperature, two days at 2-8ºC, or frozen at -20ºC or colder for longer storage.

High-Density Lipoprotein Cholesterol

Intended Use

The ‘AHDL’ method is an *in vitro* diagnostic test for the quantitative measurement of high-density lipoprotein cholesterol (HDL-C) in human serum and plasma on the Dimension® clinical chemistry system. Measurements of HDL-C are used as an aid in the diagnosis of lipid disorders (such as diabetes mellitus), various liver and renal diseases and in the assessment of risk for atherosclerosis and cardiovascular disease. This method made use of Flex® reagent cartridge. Serum and plasma were collected using recommended procedures for collection and diagnostic blood specimens by venipuncture.

Principles of Procedure

The ADHL assay measures HDL cholesterol levels directly without the need for sample pre-treatment or specialised centrifugation steps, using a two reagent format. In the first reaction, chylomicrons, VLDL and LDL form water soluble complexes with dextran sulfate in the presence of magnesium sulfate. These complexes are resistant to the polyethylene glycol-modified cholesterol esterase and cholesterol oxidase that react with HDL cholesterol. In the presence of oxygen, the HDL cholesterol is oxidized to ∆4-cholestenone and hydrogen peroxide. The generated hydrogen peroxide then reacts with 4-aminoantipyrine and sodium N-(2-hydroxy-3-sulfopropyl)-3,5-dimethoxyaniline in the presence of peroxidise to form a coloured dye that is measured using a bichromatic (600/700 nm) endpoint technique. The colour intensity of the dye is directly proportional to the serum HDL-C concentration.

Specimen Collection and Handling

It is important to note that serum and plasma should be collected after a 12-hour period of fasting using recommended procedures for collection of diagnostic blood specimens by venipuncture. Complete clot formation should take place before centrifugation. Serum and plasma should be physically separated from cells as soon as possible with a maximum limit of two hours from time of collection. Specimens

should be free of particulate matter. Serum or plasma samples may be refrigerated at 2-8ºC for up to seven days if not tested within eight hours. For longer storage, samples may be frozen at -70ºC for up to three months. Samples may be frozen only once. When using samples stored at 2-8ºC or -70ºC, increases or decreases of up to 10% may be observed in HDL-C concentrations.

Low-Density Lipoprotein Cholesterol

Intended Use

LDLD reagent, when used in conjunction with SYNCHRON LX® Systems, UniCel® DxC 600/800 Systems and SYNCHRON® Systems LDLD Calibrator, is intended for the direct quantitative determination of low-density lipoprotein cholesterol in human serum of plasma.

Principles of Procedure

This direct LDL Cholesterol method is a homogeneous assay without the need for any off-line pre-treatment or centrifugation steps. The method depends on a unique detergent which solubilises only the non-LDL lipoprotein particles and releases cholesterol to react with cholesterol esterase and cholesterol oxidase to produce a non-colour forming reaction. A second detergent solubilises the remaining LDL particles, and a chromogenic coupler allows for colour formation. LDLD reagent is used to measure the cholesterol concentration by a timed-endpoint method. The SYNCHRON® Systems automatically proportions the appropriate LDL cholesterol sample and reagent volumes into a cuvette. The ratio used is one part sample to 93 parts reagent. The System monitors the change in absorbance at 560 nm. This change in absorbance is directly proportional to the concentration of LDL cholesterol in the sample and is used by the System to calculate and express the LDL cholesterol concentration.

Specimen Collection and Handling

It is important to note that tubes are to be kept closed at all times and in a vertical position. It is recommended that the serum of plasma be physically separated from contact with cells within two hours from the time of collection. Specimens should not remain at room temperature longer than eight hours. If assays are not completed within eight hours then the specimens should be stored at 2-8ºC. LDL cholesterol is stable for up to five days at 2-8ºC. Samples may be frozen at -80ºC. Frozen samples should be thawed only once as analyte deterioration may occur in samples that are repeatedly frozen and thawed.

APPENDIX 17

GLYCATED HAEMOGLOBIN ANALYSIS

HbA_{1c}

Intended Use

The Bio-Rad D-10™ Haemoglobin A_{1c} Programme is an *in vitro* diagnostic test intended for the percent determination of haemoglobin A_{1c} in human whole blood using ion-exchange high-performance liquid chromatography (HPLC).

Principle of Procedure

The samples are automatically diluted on the D-10 and injected into the analytical cartridge. The D-10 delivers a programmed buffer gradient of increasing ionic strength to the cartridge, where the haemoglobins are separated based on their ionic interactions with the cartridge material. The separated haemoglobins then pass through the flow cell of the filter photometer, where changes in the absorbance at 415 nm are measured. The D-10 software performs reduction of raw data collected from each analysis. Two-level calibration is used for quantitation of the values HbA_{1c} values. A sample report and a chromatogram are generated for each sample. The peak is shaded. This area is calculated using an exponentially modified Gaussian (EMG) algorithm that excludes the labile A_{1c} and carbamylated peak areas form the A_{1c} peak area.

Specimen Collection and Handling

The whole blood specimens should be collected in a vacuum collection tube containing EDTA. These specimens may be stored up to seven days at 2-8ºC or three days at room temperature (15-30ºC). For specimen preparation, allow sample tubes to reach room temperature before performing the assay.

APPENDIX 18

STATISTICAL ANALYSES

Comparative Study

Body Mass Index

Variable	Test of means against reference constant (SADHS)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
BMI	37.603	8.6425	40	1.3665	27.200	7.6130	39	0.0000

Variable	Test of means against reference constant (SANHANES-1)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
BMI	37.603	8.6425	40	1.3665	29.000	6.2958	39	0.0000

Waist Circumference

Variable	Test of means against reference constant (SADHS)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
WC	1130.6	176.29	40	27.874	830.00	10.783	39	0.0000

Variable	Test of means against reference constant (SANHANES-1)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
WC	1130.6	176.29	40	27.874	889.00	8.6667	39	0.0000

Fat Mass

Variable	Test of means against reference constant (Normal Upper Limit)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
Fat %	45.233	9.4638	40	1.4964	31.000	9.5114	39	0.0000

Resting Heart Rate

Variable	Test of means against reference constant (SADHS)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
HR	77.150	8.0561	40	1.2738	77.000	0.1178	39	0.9069

Variable	Test of means against reference constant (SANHANES-1 females)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
HR	77.150	8.0561	40	1.2738	77.200	-0.0393	39	0.9689

Variable	Test of means against reference constant (SANHANES-1 black)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
HR	77.150	8.0561	40	1.2738	74.500	2.0804	39	0.0441

Systolic Blood Pressure

Variable	Test of means against reference constant (SADHS)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
SBP	130.33	13.564	40	2.1446	121.00	4.3482	39	0.0001

Variable	Test of means against reference constant (SANHANES-1 females)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
SBP	130.33	13.564	40	2.1446	127.55	1.2940	39	0.2033

Variable	Test of means against reference constant (SANHANES-1 black)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
SBP	130.33	13.564	40	2.1446	128.07	1.0515	39	0.2995

Diastolic Blood Pressure

Variable	Test of means against reference constant (SADHS)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
DBP	87.900	10.205	40	1.6136	75.000	7.9947	39	0.0000

Variable	Test of means against reference constant (SANHANES-1 female)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
DBP	87.900	10.205	40	1.6136	74.540	8.2798	39	0.0000

Variable	Test of means against reference constant (SANHANES-1 black)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
DBP	87.900	10.205	40	1.6136	73.820	8.7260	39	0.0000

Energy Expenditure

Variable	Test of means against reference constant (SADHS)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
PA	1362.3	1455.7	40	230.16	1336.4	0.1126	39	0.9109

Full Blood Lipid Profile

Variable	Test of means against reference constant (SANHANES-1)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
TC	4.6375	1.0751	40	0.1700	4.4300	1.2207	39	0.2295

Variable	Test of means against reference constant (SANHANES-1)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
TG	0.9575	0.5178	40	0.0819	1.2100	-3.084	39	0.0037

Variable	Test of means against reference constant (SANHANES-1)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
HDL	1.3050	0.3479	40	0.0550	1.2600	0.8182	39	0.4182

Variable	Test of means against reference constant (SANHANES-1)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
LDL	2.8775	0.9292	40	0.1469	2.6400	1.6166	39	0.1140

Glycated Haemoglobin

Variable	Test of means against reference constant (SANHANES-1 female)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
HbA1c %	5.8675	1.4215	40	0.2248	5.9000	-0.1446	39	0.8858

Variable	Test of means against reference constant (SANHANES-1 black)							
	Mean	Std.Dv.	N	Std.Err.	Reference Constant	t-value	df	p
HbA1c %	5.8675	1.4215	40	0.2248	5.8000	0.3003	39	0.7655

Baseline Correlations

Body Mass Index and Fat Mass

Variable	Correlations (BMI and Fat) Marked correlations are significant at p < .05000 N=40 (Casewise deletion of missing data)
	Fat
BMI	.8967
	p=.000

Body Mass Index and Waist Circumference

Variable	Correlations (BMI and WC) Marked correlations are significant at p < .05000 N=40 (Casewise deletion of missing data)
	BMI
WC	.9522
	p=0.00

Waist Circumference and Fat Mass

Variable	Correlations (WC and Fat) Marked correlations are significant at p < .05000 N=40 (Casewise deletion of missing data)
	Fat %
WC	.8701
	p=.000

Waist Circumference and Fitness

Variable	Correlations (WC and Fitness) Marked correlations are significant at p < .05000 N=40 (Casewise deletion of missing data)
	Fitness
WC	-.3289
	p=.038

Step Count and Fat Mass

Variable	Correlations (Steps and Fat) Marked correlations are significant at p < .05000 N=39 (Casewise deletion of missing data)
	Fat %
Steps	-.3366
	p=.036

Heart Rate and Triglycerides

Variable	Correlations (HR and Triglycerides) Marked correlations are significant at p < .05000 N=40 (Casewise deletion of missing data)
	TG
HR	.4693
	p=.002

Intervention Study: Group Results

Step Count

Effect	Repeated Measures Analysis of Variance (steps) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	1.2655E+10	1	1.2655E+10	532.05	0.0000
CODE	2.6736E+08	1	2.6736E+08	11.24	0.0028
Error	5.4706E+08	23	2.3785E+07		
MONTH	6.0879E+07	3	2.0293E+07	5.08	0.0031
MONTH*CODE	4.4762E+07	3	1.4921E+07	3.74	0.0150
Error	2.7536E+08	69	3.9908E+06		

Cell No.	Tukey HSD test; variable DV_1 (steps - group) Approximate Probabilities for Post Hoc Tests Error: Between MS = 2379E4, df = 23.000 CODE	{1} 9622.0	{2} 12895.
1	1		0.0029
2	2	0.0029	

Cell No.	Tukey HSD test; variable DV_1 (steps - time) Approximate Probabilities for Post Hoc Tests Error: Within MS = 3991E3, df = 69.000 MONTH	{1} 10149.	{2} 11093.	{3} 11740.	{4} 12313.
1	0		0.3467	0.0314	0.0017
2	1	0.3467		0.6626	0.1450
3	2	0.0314	0.6626		0.7420
4	3	0.0017	0.1450	0.7420	

Cell No.	Tukey HSD test; variable DV_1 (steps - interaction) Approximate Probabilities for Post Hoc Tests Error: Between; Within; Pooled MS = 8939E3, df = 47.933 CODE	MONTH	{1} 9084.3	{2} 9828.4	{3} 10119.	{4} 9455.8	{5} 11131.	{6} 12260.	{7} 13237.	{8} 14951.
1	1	0		0.9839	0.9070	0.9998	0.6808	0.1627	0.0229	0.0004
2	1	1	0.9839		1.0000	0.9998	0.9563	0.4724	0.1072	0.0022
3	1	2	0.9070	1.0000		0.9918	0.9894	0.6305	0.1796	0.0045
4	1	3	0.9998	0.9998	0.9918		0.8529	0.2927	0.0513	0.0009
5	2	0	0.6808	0.9563	0.9894	0.8529		0.8349	0.1442	0.0003
6	2	1	0.1627	0.4724	0.6305	0.2927	0.8349		0.9148	0.0215
7	2	2	0.0229	0.1072	0.1796	0.0513	0.1442	0.9148		0.3720
8	2	3	0.0004	0.0022	0.0045	0.0009	0.0003	0.0215	0.3720	

Energy Expenditure

Effect	Repeated Measures Analysis of Variance (EE) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	383555379	1	383555379	81.733	0.0000
CODE	9539548	1	9539548	2.033	0.1674
Error	107934256	23	4692794		
MONTH	23381113	3	7793704	5.194	0.0027
MONTH*CODE	23283262	3	7761087	5.172	0.0028
Error	103531227	69	1500453		

Cell No.	Tukey HSD test; variable DV_1 (EE - time) Approximate Probabilities for Post Hoc Tests Error: Within MS = 1500E3, df = 69.000 MONTH	{1} 1505.3	{2} 1548.0	{3} 2673.8	{4} 2162.4
1	0		0.9994	0.0067	0.2391
2	1	0.9994		0.0096	0.2949
3	2	0.0067	0.0096		0.4573
4	3	0.2391	0.2949	0.4573	

Cell No.	Tukey HSD test; variable DV_1 (EE - interaction) Approximate Probabilities for Post Hoc Tests Error: Between; Within; Pooled MS = 2299E3, df = 67.564 CODE	MONTH	{1} 1813.7	{2} 655.00	{3} 2700.0	{4} 1435.0	{5} 1220.7	{6} 2372.3	{7} 2649.7	{8} 2833.8
1	1	0		0.2997	0.6404	0.9948	0.9763	0.9831	0.8642	0.6993
2	1	1	0.2997		0.0029	0.7720	0.9818	0.1049	0.0327	0.0137
3	1	2	0.6404	0.0029		0.2008	0.2408	0.9994	1.0000	1.0000
4	1	3	0.9948	0.7720	0.2008		1.0000	0.7807	0.4887	0.3064
5	2	0	0.9763	0.9818	0.2408	1.0000		0.2595	0.0737	0.0267
6	2	1	0.9831	0.1049	0.9994	0.7807	0.2595		0.9991	0.9784
7	2	2	0.8642	0.0327	1.0000	0.4887	0.0737	0.9991		0.9999
8	2	3	0.6993	0.0137	1.0000	0.3064	0.0267	0.9784	0.9999	

Diet

Effect	Repeated Measures Analysis of Variance (kcal) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	304413452	1	304413452	997.94	0.0000
CODE	293038	1	293038	0.96	0.3377
Error	6710934	22	305042		
WEEKS	785378	3	261793	2.65	0.0558
WEEKS*CODE	104499	3	34833	0.35	0.7871
Error	6513261	66	98686		

Effect	Repeated Measures Analysis of Variance (carbs) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	4084408	1	4084408	757.57	0.0000
CODE	123	1	123	0.02	0.8813
Error	118612	22	5391		
WEEKS	7497	3	2499	1.56	0.2082
WEEKS*CODE	4045	3	1348	0.84	0.4768
Error	105934	66	1605		

Effect	Repeated Measures Analysis of Variance (protein) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	384926	1	384926	568.75	0.0000
CODE	1476	1	1476	2.18	0.1539
Error	14889	22	677		
WEEKS	872	3	291	1.40	0.2495
WEEKS*CODE	517	3	172	0.83	0.4806
Error	13665	66	207		

Effect	Repeated Measures Analysis of Variance (fats) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	447788	1	447788	397.50	0.0000
CODE	3052	1	3052	2.71	0.1140
Error	24783	22	1127		
WEEKS	4158	3	1386	3.18	0.0295
WEEKS*CODE	1229	3	410	0.94	0.4262
Error	28745	66	436		

Cell No.	Tukey HSD test; variable DV_1 (fats - time) Approximate Probabilities for Post Hoc Tests Error: Within MS = 435.54, df = 66.000 WEEKS	{1} 58.468	{2} 77.132	{3} 71.356	{4} 69.072
1	0		0.0149	0.1516	0.3018
2	4	0.0149		0.7732	0.5425
3	8	0.1516	0.7732		0.9814
4	12	0.3018	0.5425	0.9814	

Full Blood Lipid Profile

Effect	Repeated Measures Analysis of Variance (TC) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	1841.1	1	1841.1	1124.5	0.0000
CODE	3.3	1	3.3	2.0	0.1715
Error	37.7	23	1.6		
MONTH	0.0	3	0.0	0.2	0.9065
MONTH*CODE	0.2	3	0.1	0.9	0.4567
Error	5.8	69	0.1		

Effect	Repeated Measures Analysis of Variance (TG) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	75.561	1	75.561	290.38	0.0000
CODE	0.544	1	0.544	2.09	0.1618
Error	5.985	23	0.260		
MONTH	0.037	3	0.012	0.31	0.8177
MONTH*CODE	0.368	3	0.123	3.06	0.0338
Error	2.763	69	0.040		

Cell No.	Tukey HSD test; variable DV_1 (TG - interaction) Approximate Probabilities for Post Hoc Tests Error: Between; Within; Pooled MS = .09508, df = 45.878 CODE	MONTH	{1} .85000	{2} .95833	{3} .97500	{4} .99167	{5} .90000	{6} .80000	{7} .70000	{8} .78462
1	1	0		0.8858	0.7886	0.6653	0.9999	0.9999	0.9233	0.9995
2	1	1	0.8858		1.0000	0.9999	0.9997	0.9006	0.4347	0.8493
3	1	2	0.7886	1.0000		1.0000	0.9987	0.8445	0.3551	0.7807
4	1	3	0.6653	0.9999	1.0000		0.9951	0.7749	0.2838	0.7013
5	2	0	0.9999	0.9997	0.9987	0.9951		0.9053	0.1934	0.8204
6	2	1	0.9999	0.9006	0.8445	0.7749	0.9053		0.9053	1.0000
7	2	2	0.9233	0.4347	0.3551	0.2838	0.1934	0.9053		0.9593
8	2	3	0.9995	0.8493	0.7807	0.7013	0.8204	1.0000	0.9593	

Effect	Repeated Measures Analysis of Variance (HDL) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	165.48	1	165.48	490.81	0.0000
CODE	0.37	1	0.37	1.08	0.3087
Error	7.75	23	0.34		
MONTH	0.02	3	0.01	0.29	0.8305
MONTH*CODE	0.05	3	0.02	0.94	0.4257
Error	1.22	69	0.02		

Effect	Repeated Measures Analysis of Variance (LDL) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	681.97	1	681.97	514.65	0.0000
CODE	0.78	1	0.78	0.59	0.4500
Error	30.48	23	1.33		
MONTH	0.02	3	0.01	0.09	0.9667
MONTH*CODE	0.14	3	0.05	0.61	0.6091
Error	5.27	69	0.08		

Glycated Haemoglobin

Effect	Repeated Measures Analysis of Variance (HbA1c) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	1579.0	1	1579.0	2341.3	0.0000
CODE	0.0	1	0.0	0.0	0.9935
Error	15.5	23	0.7		
MONTH	1.3	1	1.3	33.7	0.0000
MONTH*CODE	0.3	1	0.3	9.0	0.0064
Error	0.9	23	0.0		

Cell No.	Tukey HSD test; variable DV_1 (HbA1c - time) Approximate Probabilities for Post Hoc Tests Error: Within MS = .03759, df = 23.000 MONTH	{1} 5.4680	{2} 5.7800
1	0		0.0002
2	3	0.0002	

Cell No.	Tukey HSD test; variable DV_1 (HbA1c - interaction) Approximate Probabilities for Post Hoc Tests Error: Between; Within; Pooled MS = .35599, df = 25.556 CODE	MONTH	{1} 5.3833	{2} 5.8667	{3} 5.5462	{4} 5.7000
1	1	0		0.0002	0.9032	0.5557
2	1	3	0.0002		0.5459	0.8971
3	2	0	0.9032	0.5459		0.2089
4	2	3	0.5557	0.8971	0.2089	

Body Mass Index

Effect	Repeated Measures Analysis of Variance (BMI) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	147138	1	147138	555.88	0.0000
CODE	34	1	34	0.13	0.7244
Error	6088	23	265		
MONTH	1	3	0	1.14	0.3386
MONTH*CODE	4	3	1	4.56	0.0057
Error	22	69	0		

Cell No.	CODE	MONTH	{1} 37.647	{2} 37.531	{3} 37.913	{4} 38.143	{5} 39.283	{6} 39.056	{7} 38.645	{8} 38.898
			Tukey HSD test; variable DV_1 (BMI - interaction) Approximate Probabilities for Post Hoc Tests Error: Between; Within; Pooled MS = 66.416, df = 23.168							
1	1	0		0.9997	0.9439	0.4018	0.9996	0.9998	1.0000	0.9999
2	1	1	0.9997		0.7211	0.1605	0.9993	0.9997	1.0000	0.9999
3	1	2	0.9439	0.7211		0.9742	0.9999	1.0000	1.0000	1.0000
4	1	3	0.4018	0.1605	0.9742		1.0000	1.0000	1.0000	1.0000
5	2	0	0.9996	0.9993	0.9999	1.0000		0.9701	0.0960	0.6682
6	2	1	0.9998	0.9997	1.0000	1.0000	0.9701		0.5910	0.9964
7	2	2	1.0000	1.0000	1.0000	1.0000	0.0960	0.5910		0.9465
8	2	3	0.9999	0.9999	1.0000	1.0000	0.6682	0.9964	0.9465	

Waist Circumference

Effect	Repeated Measures Analysis of Variance (WC) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	131755376	1	131755376	1061.8	0.0000
CODE	11	1	11	0.0	0.9926
Error	2854035	23	124088		
MONTH	10981	3	3660	1.7	0.1838
MONTH*CODE	8415	3	2805	1.3	0.2910
Error	152198	69	2206		

Fat Mass

Effect	Repeated Measures Analysis of Variance (fat) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	225261	1	225261	705.90	0.0000
CODE	51	1	51	0.16	0.6921
Error	7340	23	319		
MONTH	60	3	20	7.50	0.0002
MONTH*CODE	10	3	3	1.29	0.2852
Error	184	69	3		

Cell No.	Tukey HSD test; variable DV_1 (fat - time) Approximate Probabilities for Post Hoc Tests Error: Within MS = 2.6607, df = 69.000 MONTH	{1} 46.248	{2} 47.568	{3} 48.428	{4} 47.640
1	0		0.0280	0.0002	0.0184
2	1	0.0280		0.2531	0.9987
3	2	0.0002	0.2531		0.3275
4	3	0.0184	0.9987	0.3275	

Lean Mass

Effect	Repeated Measures Analysis of Variance (lean) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	274263	1	274263	859.61	0.0000
CODE	52	1	52	0.16	0.6898
Error	7338	23	319		
MONTH	61	3	20	7.54	0.0002
MONTH*CODE	11	3	4	1.37	0.2603
Error	185	69	3		

Cell No.	Tukey HSD test; variable DV_1 (lean - time) Approximate Probabilities for Post Hoc Tests Error: Within MS = 2.6880, df = 69.000 MONTH	{1} 53.676	{2} 52.336	{3} 51.480	{4} 52.272
1	0		0.0260	0.0002	0.0179
2	1	0.0260		0.2611	0.9991
3	2	0.0002	0.2611		0.3276
4	3	0.0179	0.9991	0.3276	

Resting Heart Rate

Effect	Repeated Measures Analysis of Variance (HR) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	591102	1	591102	5186.6	0.0000
CODE	78	1	78	0.7	0.4174
Error	2621	23	114		
MONTH	131	3	44	1.1	0.3370
MONTH*CODE	249	3	83	2.2	0.0976
Error	2624	69	38		

Systolic Blood Pressure

Effect	Repeated Measures Analysis of Variance (SBP) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	1745742	1	1745742	3645.5	0.0000
CODE	4	1	4	0.0	0.9314
Error	11014	23	479		
MONTH	200	3	67	0.9	0.4222
MONTH*CODE	314	3	105	1.5	0.2246
Error	4840	69	70		

Diastolic Blood Pressure

Effect	Repeated Measures Analysis of Variance (DBP) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	780500	1	780500	4618.1	0.0000
CODE	117	1	117	0.7	0.4136
Error	3887	23	169		
MONTH	137	3	46	1.4	0.2381
MONTH*CODE	147	3	49	1.5	0.2095
Error	2189	69	32		

Fitness Level

Effect	Repeated Measures Analysis of Variance (fitness) Sigma-restricted parameterization Effective hypothesis decomposition SS	Degr. of Freedom	MS	F	p
Intercept	22821786	1	22821786	5470.7	0.0000
CODE	45705	1	45705	11.0	0.0031
Error	95948	23	4172		
MONTH	30006	3	10002	10.3	0.0000
MONTH*CODE	10153	3	3384	3.5	0.0204
Error	67107	69	973		

Cell No.	Tukey HSD test; variable DV_1 (fitness - group) Approximate Probabilities for Post Hoc Tests Error: Between MS = 4171.6, df = 23.000 CODE	{1} 456.71	{2} 499.50
1	1		0.0032
2	2	0.0032	

Cell No.	Tukey HSD test; variable DV_1 (fitness - group) Approximate Probabilities for Post Hoc Tests Error: Within MS = 972.56, df = 69.000 MONTH	{1} 449.40	{2} 484.40	{3} 488.72	{4} 493.32
1	0		0.0011	0.0003	0.0002
2	1	0.0011		0.9612	0.7435
3	2	0.0003	0.9612		0.9537
4	3	0.0002	0.7435	0.9537	

Cell No.	Tukey HSD test; variable DV_1 (fitness - interaction) Approximate Probabilities for Post Hoc Tests Error: Between; Within; Pooled MS = 1772.3, df = 57.111 CODE	MONTH	{1} 431.25	{2} 444.25	{3} 471.42	{4} 479.92	{5} 466.15	{6} 521.46	{7} 504.69	{8} 505.69
1	1	0		0.9697	0.0464	0.0067	0.4455	0.0002	0.0015	0.0012
2	1	1	0.9697		0.4044	0.1115	0.8953	0.0007	0.0151	0.0127
3	1	2	0.0464	0.4044		0.9976	1.0000	0.0777	0.5076	0.4692
4	1	3	0.0067	0.1115	0.9976		0.9915	0.2315	0.8198	0.7885
5	2	0	0.4455	0.8953	1.0000	0.9915		0.0007	0.0469	0.0377
6	2	1	0.0002	0.0007	0.0777	0.2315	0.0007		0.8672	0.8999
7	2	2	0.0015	0.0151	0.5076	0.8198	0.0469	0.8672		1.0000
8	2	3	0.0012	0.0127	0.4692	0.7885	0.0377	0.8999	1.0000	