THE EFFECT OF A 12–WEEK HOME–BASED EXERCISE INTERVENTION PROGRAMME ON CARDIOMETABOLIC DISEASE RISK FACTORS

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ABSTRACT

Aims: This study evaluated the use, safety, and short-term benefits of a home-based physical activity programme, on cardiometabolic disease risk factors.

Methods: Sedentary individuals (n=67) were recruited for the study and those that qualified obtained medical clearance to participate. Participants received an individual log book to record their full day activities and meals, and were instructed to complete the home–based programme three days/week for 12 weeks. The home-based programme consisted of three exercise routines (aerobic, resistance and stretching). Outcomes included changes from baseline to 12 weeks in: weight, body mass index (BMI), waist to hip ratio, fat percentage, systolic and diastolic blood pressure, resting heart rate, fasting total cholesterol, and fasting blood glucose.

Results: Forty-six participants completed the study. A significant improvement between baseline and 12 weeks post intervention for diastolic blood pressure (77 mmHg – 68 mmHg; ↓11.7%, $p < 0.05$) was identified. From baseline to 12 weeks a decrease of 9% was noted in the number of participants in the moderate risk category while the number of participants in the low risk category increased by 19%. No other statistically significant differences were detected between the baseline and 12 weeks.

Conclusions: Despite the observation that minimal statistically significant changes occurred as a result of the 12–week intervention, scores evidently show that the physical activity programme was beneficial in eliciting some positive changes (%) that may reduce cardiovascular risk. More prominent effectsmay have been observed with the inclusion of a calorie restriction programme.

DECLARATION

I hereby declare that this study represents the original work by the author and has not been submitted in any form at another University. Where use is made of the work of others, it has been duly acknowledged in the text and included in the list of references cited.

Signature: Date:

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LIST OF ABBREVIATIONS

ABBREVIATION	TERM
ACSM	American College of Sports Medicine
BC	Body composition
BM	Body Mass
BMI	Body mass index
CAD	Coronary artery disease
CHD	Coronary heart disease
CMD	Cardiometabolic disease
CVD	Cardiovascular disease
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoproteincholesterol
Min	Minutes
Par–Q	Physical Activity Readiness Questionnaire
RPE	Rating of Perceived Exertion
SD	Standard deviation
VLDL-C	Very low-density lipoprotein cholesterol
WC	Waist circumference
WHR	Waist to hip ratio

CONTENTS PAGE

INTRODUCTION

In 2007 it was estimated that 47 million United States residents; including one out of ten children aged between 12 and 19 years, were affected by cardiometabolic disease (CMD). This syndrome predominantly affects 43.5% of adults aged between 60-69 years. A strong correlation is believed to exist between CMD and the current increase in obesity, which has doubled over the past 25 years. Although obesity is considered one of the many risk factors, there is a combination of risk factors contributing to an increased risk of developing type II diabetes and cardiovascular diseases, these include: abnormally high blood glucose, abdominal obesity, high blood pressure, elevated triglyceride levels, low HDL-C levels, physical inactivity, and smoking (Ford, 2002).

Individuals living with multiple risk factors increase their likelihood of suffering from a heart attack or stroke with each additional risk factor. This principle can be illustrated by considering a person with three risk factors (see Figure 1). The possibility of suffering from a heart attack increases by 3x3x3 equaling 27, not by 3+3+3 equaling 9, with multiple risk factors (Steyn, 2007). It is therefore not surprising that these chronic diseases have been labeled a worldwide epidemic and most challenging health aspect in global health today.

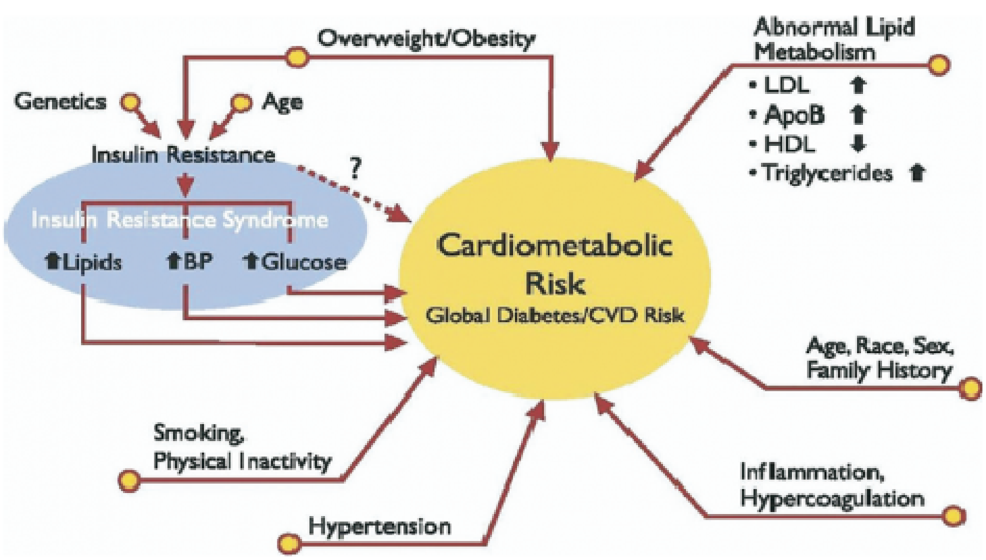

Figure 1: Factors contributing to cardiometabolic risk (John et al., 2008).

More than 1.1 billion adults are overweight worldwide, and of these 312 million are obese (Magnusson, 2009). According to Magnusson, (2009) diabetes mellitus is expected to double; from 171 million in 2000 to 366 million in 2030. This epidemic has been one of the main burdens on the United States society. However, the leading cause of death in the population group over the age of 45 years in Africa is cardiovascular disease. This is equally devastating on the economy, due to the decrease in productivity, increase in health care costs and fatal outcomes related to diabetes, hypertension, stroke, valvular heart disease and heart failure (Gaziano, 2007).

From the data released by Statistics South Africa, there has been a steady increase in the effects of chronic diseases in adults (15-64 years) from 1999 to 2006. These statistics showed that death rates rose to 20% for hypertensive heart disease, 23% for ill-defined heart diseases, 38% for diabetes and 67% for kidney disease (MRC, 2010). In addition to this, there has been an increase in deaths from 565 deaths per day in 2000 to 666 deaths per day in 2010, due to chronic diseases (Steyn, 2007). The estimated total cost of cardiovascular disease in South Africa in 1991 was between R4.135 billion and R5.035 billion. This does not include the cost of rehabilitation and follow-up of cardiovascular disease patients since the necessary data were not available to estimate it. The private sector subsides approximately three quarters of the health care costs, which were estimated to be 42% of the total cost. The rest reflects the indirect cost of earnings foregone as a result of premature morbidity and mortality (Pestana, 1996).

The cost burden associated with this premature morbidity and mortality has resulted, in companies investing in health promotion programmes. There are corporations that are going to great efforts to create a healthy corporate climate and cultural programme to support their employees. Health promotion programmes in the corporate settings are focusing more on helping employees to stay healthy, satisfied, and productive (Aldanaet al., 2005). Over the past 25 years, the offering of health promotion programmes to employees from America’s corporations have increased with most of these companies offering some type of health promotion programme. In companies or organisations with at least 50 employees, the health promotion programme within the company has increased from 81% in 1990 to 90% in 2000. This is due to the fact that adults in the working environment are spending a larger portion of their day at work; with this an increase in poor employee health comes at a cost to employers. Furthermore, the top five health conditions, including: diseases of the heart, cerebrovascular disease, cancers, unintentional injuries and chronic lower respiratory disease are potentially responsive to health intervention. A number of diseases, associated with these conditions and nearly 55% of deaths, are affected by four modifiable behavioral factors. These four factors namely tobacco use, poor diet, sedentary lifestyle and alcohol abuse, are related to five of the 20 most costly physical health conditions for employers in the U.S. These factors can be addressed in the worksite environment. These health conditions included angina pectoris, diabetes mellitus, acute myocardial infarction, chronic obstructive pulmonary disease and back pain (Robin et al., 2010).

In South Africa, the literature on health promotion programmes in the workplace is limited. Ferreira, (2006) reported that of the companies placed among the top 50 profit takers in South Africa, 25% indicated that they did not run a corporate wellness programme at their main worksite or any of their worksites. It is concerning when one considers the tangible benefits (reduced workers' compensation, health benefit cost savings, sick leave reductions, fewer workplace injuries and reduced presenteeism-related losses) and intangible benefits (improvement in employee morale, a more productive workforce, enhanced decision-making capabilities, less organizational conflict or the desire to do something good for employees) to be gained with the implementation of health promotion programmes (Chapman, 2005).

Considering the abovementioned literature as well as the fact that inactivity has been identified as one of the leading preventable causes of premature death, it would seem reasonable to ascertain the effects of a home–based exercise intervention programme on reducing the prevalence of cardiometabolic disease risk factors. A home–based exercise programme may assist individuals who for a number of reasons may not be able to participate in organised activity, such as personal (time, temperament, pain when exercising and after exercise, afraid of injury, and not understanding the importance of exercise) and environmental (lack of professional guidance relating to exercise, not having the opportunity to access a health clubs due to cost, transportation problems and climate) barriers (Wanko et al., 2004; Rimmer et al., 2004; Schutzer et al., 2004;Wilbur et al., 2005; Krousel-Wood et al., 2007). This highlights the need and importance of home-based exercise programmes, but few studies have compared this approach to traditional group/organizational based interventions. The effectiveness of center versus home-based exercises were compared by Perri et al. (1997) in a sample of sedentary, middle-aged obese women undergoing behaviour weight loss treatment. His results indicated that there was better adherence to exercise in a home-based group after 12 months, compared to the center group. Findings by Krousel-Wood et al. (2008) indicated that there was a BMI decrease in diabetes patients which resulted from a three months home-based exercise intervention programme, with an adherence to the programme of 80%.

Clearly there is a need for an intervention programme such as the home-based exercise programme, which will ultimately counter–act the personal and environmental barriers to exercise, and therefore reducing the prevalence of cardiometabolic disease.

CHAPTER 1: LITERATURE REVIEW

The following chapter introduces the reader to 'traditional'cardiometabolic disease risk factors, highlights the prevalence thereof and provides an overview of the impact that physical activity may have on these selected risk factors.

Cardiometabolic Disease Risk Factors

Physical Inactivity

It's been known for a long time that regular physical activity has been regarded as an important component of a healthy lifestyle. As early as 370 BC Hypocrates expressed himself clearly on this point: "All parts of the body which have a function, if used in moderation and exercised in labours in which each is accustomed, become thereby healthy, well–developed and age more slowly, but if unused and left to idle they become liable to disease defective in growth, and age more quickly" (Wallace, 1986). As evidenced by this quote, the value of physical activity has been inscribed since antiquity as a means of preserving or enhancing health. Therefore, the current recommendations for physical activity amongst adults aged 18 to 65 years are: moderate–intensity aerobic physical activity for a minimum of 30 minutes, five days per week and strengthening exercises at least 2 to 3 days/week (ACSM, 2010).

According to the ACSM (2010) a large body of laboratory and population–based studies has been documented, indicating the numerous health benefits associated with physical activity and endurance exercises. These benefits include: improved physiologic, metabolic, and psychological parameters, as well as decreased risk of many chronic diseases and premature mortality. Physical activity and exercise clearly reduce the occurrence of cardiac events, incidence of stroke, hypertension, type 2 diabetes mellitus, colon and breast cancers, osteoporotic fractures, gallbladder disease, obesity, depression, anxiety and delays mortality (ACSM, 2010). Regular physical activity can favourably alter components of cholesterol (Halbert et al., 1999.; Wood et al., 1998; in Coghill et al., 2008), reduce the chance of developing high blood pressure (Whelton et al., 2001. inCoghill et al., 2008) and of course it can contribute to weight loss (Roberts et al., 2005). Additionally, studies have been developed to examine the impact of change in physical activity or fitness in relation to developing coronary heart disease or dying prematurely. Individuals who change from a sedentary lifestyle to being physically active or change from physically unfit to physically fit, have less chance of disease and premature mortality, compared to those individuals that continue to remain sedentary or unfit (ACSM, 2010). This holds true from middle age to older age, indicating that it is never too late to become physically active in order to derive health benefits. From the above it becomes clear that regular physical activity, healthy eating habits, and other healthy behavioral habits all reduce the likelihood of risk factors and may improve quality of life.

Physical inactivity can be defined as engaging in ≤ 30 min of physical activity at least three days/week (ACSM, 2010). With inactivity comes the increase in metabolic and cardiovascular conditions, namely obesity, metabolic syndrome, type 2 diabetes, hypertension, coronary heart disease, stroke and peripheral arterial disease (Vuori, 2007, ACSM 2010), and is thus a well-established, independent risk factor for cardiovascular disease. In concurrence with the above, Ashlee et al. (2009) found that people with a sedentary lifestyle or that were physically unfit were more prone to present with a deleterious cardiometabolic risk factor profile. After controlling for the basic confounding variables, inactive participants were 52% more likely than active participants to have CVD ($p < 0.05$).

The World Health Report in 2002 estimated that global physical inactivity amongst adults was at least 17%, and this was the indirect cause of 1.9 million deaths and 19 million disabilities worldwide (Vuori, 2007). According to Steyn (2007), in 2003 sedentary lifestyle in South Africa was approximately 62% for men and 48% for women older than fifteen years. Furthermore, it is estimated that 3% of men and 4% of women aged 30 years and older who die, die as an indirect result of a lack of physical activity (Steyn, 2007).

In order to overcome the detrimental consequences of a sedentary lifestyle, Coghill et al. (2008) suggested individuals follow a home–based physical activity programme which can be incorporated into everyday life. The home–based physical activity programme is more likely to produce sustainable change in behaviour, as well as being accessible to a larger number of people than structured, facility-based activities. Some employees do not get the opportunity to engage in physical activity at work. The majority of people will go home after a day's work and rest, rather than exercise. For employees that are working long hours and do not get the opportunity to do some kind of physical activity, it may be beneficial to incorporate a home–based programme into their daily routine.

Body Composition (Obesity)

Obesity is defined by Durstine et al. (2003) as an excess amount of body fat resulting in a significant impairment of health. It is well known that excess body fat is associated with hypertension, stroke, type 2 diabetes, coronary heart disease, and hyperlipidemia (ACSM, 2010; Centers for Disease Control, 2011). Approximately 65% of the United States population of today are classified as overweight (BMI ≥ 25 kg/m^2) and almost 34.5% as obese (BMI ≥ 30 kg/m^2). With an increase in obesity from 15% in 1980 to 30.9% in 2000 for adults, there was an alarming increased trend in the incidence of overweight children in the United States. It has been established that there was an approximate increase from 4% in 1970 to 15% in 2000 (Durstine et al., 2003; Centers for Disease Control, 2011). Obesity is a major independent, modifiable risk factor for the development of cardiovascular diseases. It is a complex multi–factorial chronic condition. BMI in the American population has increased in such a way, that the proportion of the population with morbid obesity has increased by a greater extent than overweight and mild obesity (Lavie et al., 2009). According to recent research done by Lavie et al. (2009) obesity is associated with morbidity more so than smoking, alcoholism, and poverty. It has also been indicated that if these trends continue, obesity may soon overtake cigarette abuse, becoming the leading cause of preventable death in the American population.

In 2003, it was found that 29% of men and 56% of women in South Africa, aged 15 years or older were overweight or obese. It was estimated that of all deaths in 2000 in people 30 years and older, a high BMI contributed to 4% of the total male and 10% of the total female deaths (Steyn, 2007). According to Steyn (2007), there were 32 men and 68 women dying in South Africa per day due to the impact of a high BMI.

Obesity is a worldwide epidemic in children and adolescents in most of the developed countries. With this increase in obesity, cardiovascular disease risk factors such as high blood pressure, dyslipidaemia and insulin resistance have frequently been noted in a large number of overweight adolescents (Lavie et al., 2009). Waist circumference (WC) has been considered an acceptable substitute marker of abdominal fat mass in adolescents (ACSM, 2010). An increase in abdominal fat has also been associated with increased levels of cardiovascular risk factors (Lavie et al., 2009). Other guides that have been used less commonly but possibly with more predictive power include body fatness, waist-to-hip ratio (WHR), and weight-to-height ratio (Lavie et al., 2009). It is well established that an unbalanced diet and lack of physical activity promotes the development of excess fat storage in adipose tissue, which is an endocrine organ producing a variety of factors that can regulate energy metabolism and insulin sensitivity (Ounis, 2008). Most of the patients that enroll into a cardiac rehabilitation programme are obese, with a high incidence of upper body obesity (Santa-Clara et al., 2003).The pattern of body fat distribution is a critical factor in the relationship between obesity and metabolic abnormalities. Increased fat mass, particularly abdominal fat mass is associated with poor metabolic profiles and an increase in cardiovascular risk factors, independent of the level of obesity. For example, abdominal fat has been associated with alterations in lipoproteins such as higher low-density lipoprotein (LDL)cholesterol and lower high-density lipoprotein (HDL) cholesterol, hyperinsulinemia, hypertension, and lower peak aerobic capacity (Santa-Clara et al., 2003) According to Santa-Clara et al. (2003) abdominal fat could be classified as an independent risk factor for coronary artery disease (CAD).

Premature mortality and an individual's risk for cardiovascular disease is significantly increased by carrying excess weight, as well as weight gain during adolescence and adulthood (Lavie et al., 2009). Studies assessing mortality based on body fat and lean mass rather than BMI or weight alone have suggested that lower mortality rates were seen in subjects that lost body fat rather than lean body mass (Lavie et al., 2009). In conjunction with increased weight, a decrease in physical activity is noted amongst adolescents and young adults. Cardiometabolic disease risk factors have been independently associated with sedentary behavior and minimal physical activity. The rising incidence of overweight, obesity and obesity-related disorders among young adults stresses the need for effective 'tools' to enhance cardiovascular health of this age group. Excess weight and obesity, type 2 diabetes and cardiovascular disease can be effectively treated and prevented, with a lifestyle intervention that includes dietary modifications and exercise. In regard to this, Cederberg et al. (2011) states that individuals that are less receptive to nutritional advice, such as young men, may be more responsive to exercise interventions.

In other studies, physical activity resulted in a decreased BMI and was considered the best predictor of weight maintenance since the energy expenditure generated was able to compensate for small positive energy imbalances. Furthermore, an optimal body weight is ideal, but does not need to be achieved for health benefits to be obtained. For instance, patients with type 2 diabetes benefit considerably from a 5 to 10% reduction in body weight (Santa-Clara et al., 2003).

Blood Pressure (Hypertension)

Blood pressure refers to the pressure of blood against the arterial walls. In adults, a normal resting pressure is commonly defined as below 120/80 mmHg. Systolic pressure, the higher number, is the pressure exerted by the blood when ejected from the left ventricle. Diastolic pressure, the lower value, is the pressure that is sustained when the ventricles are relaxed. Blood pressure is dependant on cardiac output and peripheral resistance. Peripheral resistance is the force opposing blood flow, or the amount of friction the blood encounters with the vessel walls.

Decreasing the diameter of the blood vessel increases the resistance to blood flow (Gould et al., 2011). Similarly, increasing the length of the vasculature also increases resistance to flow and as a result increases pressure.

This increased pressure also known as high blood pressure is medically diagnosed as hypertension. This is defined as an arterial systolic blood pressure of 140 mmHg or greater and/or a diastolic pressure greater than 90mmHg. High blood pressure is sometimes called the "silent killer" because it usually has no apparent symptoms. In the United States, hypertension accounts for over 1.5 million deaths a year and affects more than 24% of Americans (Brooks et al., 2005). It has been stated by Brooks et al. (2005) that people between the ages of 40 and 60 years, with a blood pressure above 150/90 mmHg have an increased risk of heart disease. Hypertensive men are three times and women are six times more likely to develop heart disease. Approximately 29% of the Australian population has hypertension, and it is the most frequently treated condition by general practitioners (Sharman et al., 2008).Currently in South Africa, there are approximately 6 million people 15 years and older suffering from hypertension. From this population, only 26% of men and 51% of women knew that they had hypertension (Steyn, 2007). Although the etiology is unknown, genetic factors are thought to play a role (Seedat et al., 2006). Hypertension is more likely to develop in people who are physically inactive (≤ 30 min of physical activity at least 3 d/week), overweight (BMI ≥30 kg/m^2; waist circumference >102 cm [men] or >88 cm [women]) or who consume excess dietary sodium (>100 mmol/d or >2.4 g/d) or consume excess alcohol (>2 standard drinks/day [men]; >1 standard drink/day [women]) (Seedat et al., 2006 and Sharman et al., 2008).

According to Sharman et al. (2008) hypertension is one of the major potentially modifiable risk factors for cardiovascular diseases and death. It increases myocardial oxygen consumption by increasing the after load of the heart, causing the heart to pump harder to deliver the same quantity of blood. This process usually results in cardiac hypertrophy, which, by itself, increases myocardial oxygen consumption. Myocardial hypertrophy with atherosclerosis increases the likelihood of the development of coronary ischemia (Brooks et al., 2005).

Research has shown that exercise could be used as a therapeutic measure for individuals with high blood pressure. Exercise recommendations for patients with high blood pressure are: as frequently as possible, (preferably all days of the week), of moderate intensity (60–70% of heart rate maximum), at least 30 minutes in duration and primarily focused upon endurance with some resistance training. It has been shown that exercise geared toward weight loss has the potential to be extremely effective in lowering blood pressure. The combination of exercise and weight control helps to target all of the areas that generally trigger hypertension (ACSM, 2010). The American College of Sports Medicine advise that exercise that moderately raises the heart rate should be the foundation of any therapy for hypertension. In order to subjectively 'personify' exercise we turn to Galen and his insights on healthy living: "To me it does not seem that all movement is exercise, but only when it is vigorous. But since vigor is relative, the same movement might be exercise for one and not for another. The criterion of vigorousness is change of respiration; those movements that do not alter the respiration are not called exercise. But if anyone is compelled by any movement to breathe more or less or faster, that movement becomes exercise for him. This therefore is what is commonly called exercise…" (Galen. *in McArdle et al., 2007*).

Progressive resistance exercise may result in a slight (3 mm Hg Systolic blood pressure and 3 mmHgDiastolic blood pressure) but significant decrease in blood pressure (Sharman et al., 2008). These guidelines recommend that dynamic resistance exercises be performed in a rhythmical fashion, throughout the full range of motion, and at a moderate-to-slow controlled speed with importance placed on eccentric contractions. It is also recommended, while doing the resistance exercises to maintain a normal breathing pattern and not to do any breath holding.

Total Cholesterol

Cholesterol is a waxy fat-like substance that is utilised to aid in cell membrane anabolism, hormone and vitamin D synthesis and the formation of bile secretions that help digestion. Given that fat cannot mix with water, the role of cholesterol is to help transport fat through blood vessels. Cholesterol is coated with a protein substance before it can enter the bloodstream. The term lipoproteins are given to these cholesterol-protein packages. Lipoproteins are composed of various lipids such as cholesterol, phospholipids, triglycerides and proteins known as apoproteins and are involved in the transportation of these lipids in the plasma. Lipoproteins can be characterized as the following: chylomicrons, very low-density lipoprotein cholesterol (VLDL-C), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein (HDL-C). Chylomicrons are the largest lipoproteins, containing approximately 85% triglycerides (UNM, 2011).

An individual's cholesterol profile may be influenced by a range of environmental and personal factors such as age, gender, level of body fat, dietary intake of fat, cholesterol and carbohydrates, alcohol consumption, cigarette smoking, medication, menopausal status, and exercise. It is difficult to establish how each factor autonomously affects an individual's cholesterol levels because of the intricate interactions among these variables (UNM, 2011).

In the bloodstream, LDL-C is the primary transport carrier of cholesterol. Approximately 50 to 60% of cholesterol is transported to the cells by LDL-C. Evidence suggests that LDL-C may be a causative factor in the development of atherosclerotic plaque as it contributes to the cellular alterations of the inner walls of arteries. Thus, LDL-C is proposed to have a stronger association with CHD than total cholesterol (Manson et al., 1992; UNM, 2011). Conversely, high density lipoprotein cholesterol (HDL-C) has an inverse relationship with coronary heart disease, and acts as a counter measure to the development of coronary heart disease (CHD) (Sharrett et al., 2001; Dean et al., 2003; Curb et al., 2004). HDL-C is believed to be the strongest lipid parameter for prediction and detection of CHD in individuals of all ages (Dean et al., 2003). HDL-C plays an essential role in transportation of cholesterol from the tissues and blood to the liver where it can be removed from the body or synthesised into bile acids. HDL-C also inhibits the absorption of LDL-C at body's receptor sites and contributes to the breakdown of other lipoproteins. Based on particle density and size of the phospholipids, HDL-C is categorized into several subclasses. The major subclasses are referred to as HDL2 and HDL3. Women have more protection against CHD as they have a higher percentage of HDL2 than males, which helps to protect women from developing CHD (UNM, 2011).

The main class of lipids found in an individual's diet and adipose tissue are triglycerides. As soon as the triglycerides are removed from the chylomicron at receptor sites in the body, the chylomicron remnant is taken back to the liver for additional breakdown. Triglycerides are the main constituent of VLDL-C, making up 60 to 70% of the lipoprotein (UNM, 2011). Epidemiologic and experimental evidence has indicated that coronary heart disease in adults is significantly increased in children and adolescents with high cholesterol levels (Kelley et al., 2006). In this regard, a longitudinal investigation by Brown et al. (1984) of human populations and a vast number of animal experiments showed sufficient defects in the circulating plasma lipoproteins that can progress to arteriosclerotic cardiovascular disease. High quantities of LDL-C and low quantities of HDL-C are exclusive risk factors for coronary heart disease, peripheral vascular disease and stroke. Similarly with other primate groups, a reduction in high LDL-C levels is followed by a decline in lesions in the coronary arteries and in the larger blood vessels that are caused by atherosclerosis (McArdle et al., 2007). Evidence further suggests that an increased risk for coronary artery disease strongly correlates with high levels of total serum cholesterol and the cholesterol rich LDL-C molecule. The risk of CHD is greatly increased when these risk factors exist with other risk factors such as cigarette smoking, sedentary lifestyle, excess body fat, and uncontrolled hypertension (Andrews, 1997). According to Andrews (1997), patients with existing heart disease elicited a significantly lower total blood cholesterol and LDL-C when using an aggressive drug and diet therapy which resulted in an enhanced coronary blood flow within 6 months. In individuals who are at risk, a cholesterol-rich diet will ultimately lead to atherosclerosis, a degenerative process that results in cholesterol–rich deposits between the 1st and 2nd layers of the medium and larger arteries, causing them to narrow and eventually close. Therefore, to lower serum cholesterol, foods that contain a lower amount of cholesterol and a reduced saturated fatty acids content should rather be consumed (Weggemans, 2001).

Epidemiologic studies have found a direct relationship between plasma LDL-C levels and the prevalence of coronary heart disease. According to Couillard et al. (2001) an effective technique to improve plasma HDL-C is through frequent endurance exercise; one of the metabolic changes that play a role in the reduced risk of CHD observed among physically active and fit individuals. The reduction in total cholesterol, LDL-C, and TG, while simultaneously elevating HDL-C can be achieved through activities such as walking, jogging, and aerobics (Watts et al., 2004; UNM, 2011). Furthermore, the age-related increase in triglycerides usually observed in men can be prevented through a physically active lifestyle (Watts et al., 2004). In addition to aerobic training, there are a few studies suggesting that lipid and lipoprotein profiles can be enhanced through resistance training (Goldberg et al., 1985; Thomas et al., 1993). Decreases in total cholesterol and LDL-C have been reported for both genders using resistance training programmes, with women also eliciting a significant decrease in serum total cholesterol and LDL-C concentrations (Goldberg et al., 1984; Thomas et al., 1993). The modification of lifestyle behaviours in conjunction with a reduction in body fat and an increase in fat-free mass may contribute to these positive changes (UNM, 2011).

Blood Glucose (pre-diabetes / diabetes)

There are approximately 1.5 million people in South Africa living with diabetes. Diabetes caused 6% of deaths in women and 3% of deaths in men in the year 2000, in the population 30 years and older. Furthermore, diabetes is the most common cause of leg amputations and blindness in South Africans and contributes to kidney failure that requires dialyses and transplants (Steyn, 2007).

Diabetes mellitus is a chronic metabolic disease characterised by an absolute or relative deficiency of insulin that results in hyperglycemia. Individuals with diabetes are at risk for developing microvascular complications, including retinopathy and nephropathy; macrovascular disease and various neuropathies. Silent ischemia is common in persons with diabetes, particularly if the person has had the disease for a long period of time (Durstine et al., 2003).

There are varied and complex interactions amongst insulin and insulin action, counter regulatory hormone release, blood glucose regulation and physical activity. The levels of blood glucose are controlled by the balanced released of insulin and other endocrine hormones; the proper functioning of hormone receptors found particularly in insulin sensitive muscle cells, adipose tissues, and the liver, as well as other metabolic factors such as fuel usage and availability (Colberg, 2005).

Typically, insulin resistance is related to the development of cardiovascular disease, with obese children remaining the population most affected by this condition. Accordingly, individuals suffering from obesity and its metabolic malfunction are in urgent need for effective early interventions and treatment. It is assumed that a reduction in body fat and an increase in physical activity will improve insulin sensitivity because insulin resistance is associated with excess body fat, especially in the abdominal area, as well as with low levels of physical activity. Adults who adhere to exercise interventions have shown that weight loss and exercise training may improve insulin's effectiveness on target tissues. Likewise, individuals with diabetes mellitus (DM) are usually instructed by their health care providers to perform regular exercise (Colberg, 2005). Interestingly, George et al. (2005) noted that aerobic exercise training may improve insulin sensitivity even without a reduction in body weight. A significant risk reduction for the development of diabetes is seen with increases in physical activity. It has been well documented that regular physical activity benefits patients with diabetes by improving glyceamic control and insulin sensitivity, reducing abdominal and total fat, and may be protecting individuals against the development of type 2 diabetes aiding in the maintenance of a suitable lean-to-fat balance with respect to body mass (Krousel-Wood et al., 2007).

There are two types of diabetes mellitus, type 1 and type 2. According to Gould et al. (2011), type 1 diabetes is more severe than type 2, and is more frequently found in children and adolescents, but it could develop at any age. Although there is a genetic factor in the development of the disease, the insulin shortfall results from destruction of the pancreatic beta cells in an auto-immune reaction, resulting in an absolute deficit of insulin in the body and therefore requiring replacement therapy (Gould et al., 2011). The insulin that is required is equivalent to the metabolic needs of the body based on dietary intake and metabolic activity. Acute complications such as hypoglycemia or ketoacidosis are more likely to occur in this group. Approximately 1 in 400 to 500 children have type 1diabetes and it occurs in approximately 10% of all individuals diagnosed with diabetes. It is a major factor predisposing an individual to stroke, heart attacks, peripheral vascular disease, amputation, kidney failure, and blindness (Kovar et al., 1987; Gould et al., 2011).

Non-insulin dependent diabetes mellitus, now known as type 2 diabetes is characterized by a decreased effectiveness of insulin or a relative deficit of insulin. This abnormality may involve increased resistance by body cells to insulin, decreased pancreatic beta cell production of insulin, increased production of glucose by the liver, or it could be a combination of these factors. Type 2 diabetes is a milder form of diabetes and often develops gradually in older adults, the majority of whom are overweight. A major concern at the moment is the rapid increase in the incidence of type 2 diabetes, with the prevalence now estimated at about 9% of the population older than 20 years of age. With increasing obesity seen in this population, it is anticipated that the future incidence will increase significantly. Type 2 diabetes increases with age, with approximately half the cases found in persons greater than 55 years of age (Gould et al., 2011). The strongest predisposing factors for type 2 diabetes are obesity and a family history of diabetes (Marble et al., 1985; Barrett-Connor, 1989). Since physical activity is associated with weight loss or the prevention of weight gain, it has the ability to reduce the high morbidity, mortality, and costs associated with diabetes (Blair et al.,1985; Krousel-Wood et al., 2007).

Smoking

Smoking and tobacco use most commonly leads to diseases affecting the heart and lungs. The first signs of smoking–related health issues are often numbness in areas such as the hands and feet. Smoking is without a doubt a major risk factor for heart attacks, chronic obstructive pulmonary disease, emphysema, and cancer, particularly lung cancer, cancers of the larynx and mouth as well as pancreatic cancer (Ferrucci et al., 1999; Doll et al., 2004). A smoker's life expectancy is also reduced, with estimates ranging from 10 to 17.9 years fewer than non-smokers (Doll et al., 1994; Ferrucci et al., 1999; Doll et al., 2004). According to Doll et al. (1994) approximately two thirds of male smokers will die of smoking–related illness.

There are several reasons why tobacco use is a major risk factor for cardiovascular disease; firstly it narrows the blood vessels, increasing the likelihood of a blockage and thus, a heart attack or stroke. Nicotine is the most addictive substance to humans and is the major addictive substance in tobacco products (Steyn, 2007). The use of smokeless tobacco or secondary smoke exposes a person to similar risk of diseases as those who smoke tobacco products, and there is no safe level of exposure to tobacco products (Peto et al., 1992). Smokers and tobacco users may die prematurely because of a range of diseases. Most smokers and tobacco users die as a result of cardiovascular diseases (stroke and heart attack), followed by chronic lung diseases such as chronic bronchitis and emphysema, as well as lung cancer (Peto, 1994).

In the United States it is estimated that 60% of men and 40% of women who have a heart attack are smokers. Furthermore, the risk of developing cardiovascular diseases in the United States is 70% greater in smokers than nonsmokers (Brooks et al., 2005). It has been estimated by the

American Heart Association that almost 450,000 Americans die each year of smoking–related illnesses. Nearly a fifth of all deaths from cardiovascular diseases are due to smoking. Research by Brooks et al. (2005) has estimated that between 37 and 40 thousand non-smokers exposed to environmental tobacco smoke die from cardiovascular disease each year. Currently, there are 25.9 million men and 23.5 million women (equal to 27.8% and 23.3% of the United States population) that are smokers, putting them and the non-smokers at an increased risk of heart attacks. In addition, it is estimated that 4.4 million teenagers aged 13 to 17 years in the United States are smokers (Brooks et al., 2005). In 2000, it was estimated that 12% of men and 4% of women in South Africa, aged 30 years or older died as a result of tobacco related diseases (Groenewald et al., 2007).

Research by Hughes et al. (1992) and West (1999) indicated that stopping smoking prolongs life and reduces morbidity. Smokers attempting to stop without medical assistance have a success rate of approximately 2 to 4%. Attempts to quit with the help of an aid, particularly through a combination of behavioral counseling and nicotine replacement therapy have better success rates, but still remain low at around 18% (Ussher et al., 2000). Exercise has been proposed as an aid for smoking cessation (Hill, 1981; Ussher et al., 2000). Hughes et al. (1994) argued for the use of exercise in smoking cessation, and showed that exercise has a moderating effect on many of the variables negatively affected by nicotine withdrawal. Furthermore, it has been noted that exercise reduces nicotine cravings, mood disturbance, sleep disturbance, feelings of anxiety, subjective stress and post-smoking cessation weight-gain (O’Connor et al., 1995; Kawachi et al., 1996; Ussher et al., 2000). Exercise may be particularly relevant for female smokers in terms of the weight control benefits, (Sorenson et al., 1992). Exercise also showed a positive effect on other factors that may protect against smoking relapse, including perceived coping ability and self-esteem (Mcaulev et al., 1997).

Evidence from surveys indicates that levels of physical activity are inversely related to smoking rates. Smokers on the path of quitting are more likely to be open to an active lifestyle than smokers in general (King et al., 1996). It is evident from the above literature that becoming more active has been positively associated with both stopping smoking and the maintenance of smoking abstinence.

The effect of physical activity on CMD risk factors become more clear, and there is evidence to suggest that physical activity will have a positive effect on CMD risk factors and decrease the risk of morbidity and premature mortality.

CHAPTER 2: SCIENTIFIC PUBLICATION

A B S T R A C T

Aims: This study evaluated the use, safety, and short-term benefits of a home-based physical activity programme, on cardiometabolic disease risk factors.

Methods: Sedentary individuals (n=67) were recruited for the study and those that qualified obtained medical clearance to participate. Participants received an individual log book to record their full day activities and meals, and were instructed to complete the home–based programme three days/week for 12 weeks. The home-based programme consisted of three exercise routines (aerobic, resistance and stretching). Outcomes included changes from baseline to 12 weeks in: weight, body mass index (BMI), waist to hip ratio, fat percentage, systolic and diastolic blood pressure, resting heart rate, fasting total cholesterol, and fasting blood glucose.

Results: Forty-six participants completed the study. A significant improvement between baseline and 12 weeks post intervention for diastolic blood pressure (77 mmHg – 68 mmHg; ↓11.7%, $p < 0.05$) was identified. From baseline to 12 weeks a decrease of 9% was noted in the number of participants in the moderate risk category while the number of participants in the low risk category increased by 19%. No other statistically significant differences were detected between the baseline and 12 weeks.

Conclusions: Despite the observation that minimal statistically significant changes occurred as a result of the 12–week intervention, scores evidently show that the physical activity programme was beneficial in eliciting some positive changes (%) that may reduce cardiovascular risk. More prominent effectsmay have been observed with the inclusion of a calorie restriction programme.

KEY WORDS: cardiovascular disease risk, body composition, home-based exercise

1. Introduction

Engaging in physical activity reduces the risk of developing cardiovascular diseases (Steyn, 2007). A substantial body of literature indicates that regular physical activity can favourably alter components of cholesterol, reduce blood pressure and contribute to weight loss, reduce visceral and total fat and improve glycemic control and insulin sensitivity (Boule et al., 2001; Pigman et al., 2002; Krousel-Wood et al., 2007; Halbertet al., 1999; Coghill et al., 2008; Irwin et al., 2003). Conversely physical inactivity is well established as an independent risk factor for developing cardiovascular disease (Paffenbargeret al., 1993; Coghillet al., 2008). In 2002, it was estimated that the global prevalence of physical inactivity among adults was at least 17%, and that physical inactivity was the cause of 1.9 million deaths and 19 million disabilities (Vuori, 2007). Literature has also shown that the reasons for physical inactivity can be broken down into personal and environmental barriers. Personal barriers include temperament, time, physical ailments, pain or discomfort when exercising, fear of injury, and misconceptions about exercise (Wanko et al. 2004; Schutzeret al., 2004; Rimmer et al., 2004; Wilbur et al., 2005; Krousel-Wood et al., 2007). Environmental barriers include lack of physician guidance concerning exercise (Krousel-Wood et al., 2007), lack of access to health clubs due to cost (Rimmer et al., 2004; Kwon et al., 2004;Krousel-Wood et al., 2007), transportation problems (Jones et al., 1996;Krousel-Wood et al., 2007) and climate (Wilbur et al., 2005;Krousel-Wood et al. 2007). It is arguable that a home-based programme incorporated into everyday life is more likely to produce sustainable change in behaviour, as well as being accessible to a larger number of people compared with structured, facility-based activities (Coghill et al., 2008). Currently, there exists limited research on the efficacy of home-based exercise interventions in reducing cardiovascular and metabolic disease risk. It is also unclear if home-based programmes alone, without calorie restriction can impact favourably on cardiovascular and metabolic disease risk factors. To this end, the purpose of this study was to determine the effects of a home-based exercise programme on cardiometabolic disease risk factors in previously sedentary individuals.

2.Materials and Methods

2.1.Participants

The study was approved by the institutions Faculty of Science and Agriculture Ethics Committee. People who responded to an advertisement were recruited for the study. Inclusion criteria were age 20–65 years old, primary physician clearance for participation in the study, not partaking in regular physical activity (≤ 3 times per week for 30 min) and, not on any diet or calorie restriction programme. All participants had to complete an informed consent form, physical activity questionnaire and a Par–Q. The participants were provided with an individual log book that had to be completed daily. The information that was recorded included the home–based programme performed on the day, any other structured/organized sport/physical activity (if yes: type, frequency, intensity and duration), diet (breakfast, lunch and dinner), any other meals or snacks, rate of perceived exertion for the exercise session, and other e.g.: illness. Participants were instructed to maintain their normal pre-intervention daily calorie intake pattern, for the duration of the 12 weeks, as closely as possible. Participants were also instructed not to ingest supplements/meal replacements during the course of the study.

Of the 80 participants that were recruited and that participated in the first assessment, 46 completed the 12–week programme. Of the 34 participants who discontinued, nineteen withdrew because they had no time or interest, five withdrew because of work–related commitments, two withdrew due to a wedding, two had to withdraw (one was involved in a car accident and the other sprained an ankle).Two had to withdraw before commencement of the exercise programme due to medical problems, and four participants were excluded from the study as that they followed the programme at a gym and not at home. Of the forty-six participants that finished the study, the mean age of the group was 43 ± 10 years; 63 % (n=29) were female with a mean age of 43 ± 10 years and, 37 % (n=17) were male with a mean age of 42 ± 11years.

2.2. Outcome Measures

Weight was measured in kilograms using a calibrated Robusta Seca 813 scale (Teraoka–South Africa, 2010). The participants were weighed without shoes and with as little clothes as possible. Measurements were taken to the nearest 0.1kg. Height was measured in centimetres using a mobile stadiometer rod with a sliding ruler. Height and weight was used to calculate BMI (weight/height^2-kg/m^2). Resting blood pressure was measured using an ALPK2 aneroid sphygmomanometer and a BioCare professional stethoscope (Medical Center Trading Corporation - Japan, 2009; BioCare Medical Systems - China, 2009). Participants were seated quietly for at least 5 minutes in a chair with back support with their feet on the floor and their arms supported at heart level. If the blood pressure exceeded 140/90 mmHg (increased risk for cardiovascular disease), a second measurement were taken after 5 minutes and the average of the 2 measurements were calculated. Skinfold measurements were taken to calculate fat percentage using a Lange skinfold caliper (Beta Technology - United States, 2000). A sum of six skinfold measurements were calculated. The following skinfolds were used: triceps, sub–scapular, suprailiac, abdominal, frontal thigh and calf [Males: Sum of 6 folds x 0.1051 + 2.585; Females: Sum of 6 folds x 0.1548 + 3.58] (YUHASZ, 1974). Resting heart rate was taken using a BioCare professional stethoscope by placing the stethoscope on the participant's chest, over the position of the heart (BioCare Medical Systems - China, 2009). Waist-to-Hip ratio was measured with a non–elastic measuring tape. The participants were standing with their feet together and arms at the sides, elevated just high enough for the measurements to be recorded. The waist measurement was taken at the natural waist (smallest waist circumference). If no natural waist was observable, the measurement was taken just below the last rib (ACSM, 2010). The hip measurement was taken at the maximum circumference of the buttocks (ACSM, 2010). The scores were recorded to the nearest millimetres. Fasting (9–11 hours) glucose and cholesterol were measured using Accutrend GCT kit (Roche Diagnostics - Germany, 2000). The machine was calibrated by using the batch-related calibration strip before the assessments. A Softclix lancet was used to obtain the capillary blood sample. The first drop of blood was not utilised; the finger was wiped clean, and the second drop of blood was placed on the designated pad of the strip.

2.3. Intervention Programme

The participants were required to complete the home–based programme three days per week. Each participant received an individual log book to record their daily activities and meals. The participants were taught to use the Rating of Perceived Exertion (RPE) scale and trained at an intensity equivalent to an RPE of 11 to 13, for each of the 36 sessions. The ACSM's guidelines for improvement of fitness and general health were used as the basis for their exercise programme prescription (Durstine & Moore, 2003:1996). The duration of each exercise session was approximately 50-60 minutes. The participants had to walk or jog 3 times per week for their cardiovascular exercises, each session lasting 20 minutes. The resistance exercises (Appendix E) were prescribed for the conditioning and strengthening of the upper and lower body muscle groups 3 times per week, using their own body weight, or to substitute for weights, any water bottles, or home items. Participants performed the same amount of sets and repetitions. Sets and repetitions depended on the type of exercise and the difficulty level of an exercise. Each exercise session, included a warm–up and cool–down phase where basic stretching exercises were performed (shoulder, neck, quadriceps and hamstring). Each stretch was performed 3 times for 20 sec. per muscle group. The home–based physical activity programme was demonstrated to all participants prior to commencement of the programme. For the duration of the 12 weeks they had to exercise unsupervised. Re-assessments were conducted every 4 weeks. Participants were contacted weekly via phone, sms and email in order to provide motivation and to get feedback on how they were finding the programme, and to determine if they were completing their log books and adhering to the weekly exercise.

The participants' cardiovascular disease risk classification (%) was calculated using a Par–Q. Participants presenting with a known cardiovascular, pulmonary or metabolic disease were classified as high risk, moderate risk candidates were classified if their base-line testing indicated 2 risk factors and low risk if they had 1 cardiovascular risk factor.The American College of Sports Medicine has identified 'thresholds' above which individuals will be at increased risk for cardiovascular disease (ACSM, 2010). These thresholds were utilized to describe risk and included the following:

- Sedentary activity – not participating in at least 30 minutes of moderate intensity exercise at least 3 days of the week for at least 3 months;
- Smoking – current smoker or those who quit within the previous 6 months;
- Obesity – BMI ≥ $30 kg/m^2$ or waist girth > 102 cm for males and > 88cm for females;
- Blood pressure – systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg;
- Dyslipidemia - fasting total cholesterol ≥ $5.18\ mmol.L^{-1}$
- Impaired fasting glucose – fasting plasma glucose ≥ $5.50\ mmol.L^{-1}$

2.4. Statistical Analysis

Results are expressed as means and SD, percentage change and 95% confidence intervals. Data was analyzed using Graph Pad Prism 5 (Graft Pad Software Inc., USA). A repeated measures analysis of variance together with a Tukey post-hoc test was computed to determine if differences occurred across multiple time points. Significance was set at $p \leq 0.05$.

3. Results

Table 1 - Cardiovascular disease risk factors at pre, 4, 8, and 12 weeks [mean ± SD (95 % Confidence Interval)]

	Pre	4 Weeks	8 Weeks	12 Weeks
Weight (kg)	86.50 ± 19.00 (80.47 – 91.76)	86.34 ± 19.30 (80.54 – 92.13)	85.86 ± 18.86 (80.12 – 91.59)	85.80 ± 18.84 (80.56 – 91.75)
BMI (kg/m^2)	30.48 ± 6.18 (28.37 – 32.04)	30.43 ± 6.28 (28.39 – 32.16)	30.26 ± 6.29 (28.34 – 32.16)	30.24 ± 6.12 (28.37 – 32.01)
WHR	0.83 ± 0.99 (0.80 – 0.86)	0.82 ± 0.09 (0.79 – 0.85)	0.81 ± 0.16 (0.76 – 0.86)	0.83 ± 0.09 (0.80 – 0.85)
Fat %	26.71 ± 8.90 (24.07 – 29.36)	26.35 ± 7.69 (24.04 – 28.66)	26.11 ± 7.81 (23.74 – 28.49)	25.41 ± 8.02 (23.02 – 27.79)
Systolic blood pressure (mmHg)	116.50 ± 14.55 (112.20 – 120.80)	113.20 ± 12.10 (109.60 – 116.80)	112.20 ± 13.44 (108.10 – 116.30)	110.20± 12.92 (106.00 – 114.30)
Diastolic blood pressure (mmHg)	77.07 ± 14.68 (72.71 – 81.42)	70.84 ± 13.04 (66.93 – 74.76)	72.09 ± 11.29 (68.66 – 75.52)	68.22 ± 14.00* (64.06 – 72.37)
Resting heart rate (bpm)	70.35 ± 11.94 (66.80 – 73.89)	69.78 ± 13.06 (65.85 – 73.70)	69.27 ± 10.97 (65.94 – 72.61)	69.48 ± 11.32 (66.12 – 72.84)
Fasting blood glucose (mmol.L^{-1})	5.72 ± 2.25 (5.05 – 6.38)	5.46 ± 1.02 (5.15 – 5.77)	5.57 ± 1.19 (5.21 – 5.94)	5.62 ± 1.44 (5.20 – 6.05)
Fasting total cholesterol (mmol.L^{-1})	5.26 ± 0.94 (4.98 – 5.54)	5.15 ± 0.99 (4.85 – 5.45)	5.19 ± 0.95 (4.90 – 5.47)	5.31 ±0.99 (5.01 – 5.61)

BMI: body mass index; WHR: waist-to-hip ratio; bpm: beats per minute.

*$p < 0.05$ (Pre vs. 12 weeks)

Table 2 - Prevalence (%) of individuals above the threshold for increased risk of cardiovascular disease using selected risk factors

	Females (n = 29)	Females (n = 29)	Males (n = 17)	Males (n = 17)	Combined (N = 46)	Combined (N = 46)
	Pre	**12 weeks**	**Pre**	**12 weeks**	**Pre**	**12 weeks**
Age (Male ≥ 45yr; Female ≥ 55yr)	17%	17%	53%	53%	28%	28%
Waist (Male > 102 cm; Female > 88 cm)	52%	38%	35%	35%	46%	37%
WHR (> 1.00)	0%	0%	12%	12%	4%	4%
Systolic blood pressure (≥ 140 mm Hg)	3%	0%	6%	3%	4%	2%
Diastolic blood pressure (≥ 90 mm Hg)	21%	3%	18%	10%	20%	9%
Fasting total cholesterol (≥ 5.18 mmol.L^{-1})	41%	59%	47%	47%	43%	54%
Fasting blood glucose (≥ 5.50 mmol.L^{-1})	17%	38%	71%	71%	37%	50%
Sedentary lifestyle (< 30 minute moderate intensity physical activity p/week)	59%	0%	29%	0%	48%	0%
Smoking (current or quit in last 6 months)	7%	7%	18%	18%	11%	11%
BMI (≥ 30 kg.m^{-2})	45%	41%	71%	71%	54%	52%

Table 3 - Cardiovascular disease risk classification (%)

	Females (n = 29)	Females (n = 29)	Male (n = 17)	Male (n = 17)	Combined (N = 46)	Combined (N = 46)
	Pre	**12 weeks**	**Pre**	**12 weeks**	**Pre**	**12 weeks**
High risk	27.6 %	27.6 %	47.1 %	47.1 %	34.8 %	34.8 %
Moderate risk	51.7 %	48.3 %	41.2 %	35.3 %	47.8 %	43.5 %
Low risk	20.7 %	24.1 %	11.8 %	17.6 %	17.4 %	21.7 %

Table 4 – Percentage change in dependent variable from baseline to post–programme (12 weeks)

	% Change
Weight	↓0.8%
BMI	↓0.8%
Waist to Hip Ratio	0%
Fat%	↓4.9%
Resting Systolic BP	↓4.3%
Resting Diastolic BP	↓11.7% *
Resting HR	↓1.4%
Fasting Blood Glucose	↓1.7%
Fasting Total Cholesterol	↑ 0.95%

*** p = < 0.05**

4. Discussion

Despite an extensive body of evidence demonstrating the health benefits of regular physical activity, the majority of healthy individuals and patients with cardiovascular and metabolic disease do not participate in routine physical activity. The reasons for the lack of engagement are numerous and as such it is incumbent upon researchers to investigate how barriers can be overcome and adherence to physical activity programmes can be improved. To this end, the aims of this study were to determine if a home-based physical activity programme will have an effect on cardiovascular and metabolic disease risk factors. This home-based intervention programme was designed to combine all the ACSM guidelines for improvement of fitness and general health. Optimal body weight does not need to be achieved for health benefits to be realized. Research suggests that a 5 to 10% reduction in body weight significantly improves health in patients with cardiovascular and metabolic diseases, resulting in a 5 mmHg decrease in diastolic blood pressure, a 5 mmHg decrease in systolic blood pressure, a 0.13mmol.L^{-1}increase in HDL cholesterol and a 1mmol.L^{-1}decrease in triglycerides (Look AHEAD, 2007; Krousel-Wood et al., 2007). Clinically significant improvements in most risk factors occur with losses of 10to 15% of body weight (Diabetes Care, 2011).

Although non–significant this moderate intensity, home-based exercise intervention programme resulted in slight changes in body composition (86.5 kg – 85.8 kg; ↓ 0.8%, $p > 0.05$), BMI (30.5kg/m^2– 30.25kg/m^2; ↓0.8%, $p > 0.05$) and fat % (26.7% – 25.4%; ↓4.9%, $p > 0.05$). We believe the reasons for the minimal (0.8%) decrease in body weight and BMI, was due to the fact that the participants were not allowed to change their diet at any time, and final testing was conducted just after a major holiday week. Body fat decreased with 4.9%, indicating that the 0.8% decrease in body weight is attributed to an increase in lean body mass. Each 0.45 kg gain in fat free mass increases the resting metabolic rate by 7 to 10 kCal per day (McArdle et al., 2007). Similar trends were observed for fasting blood glucose (5.7 mmol.L^{-1}–5.6 mmol.L^{-1}**;** ↓1.7%, $p > 0.05$) and total cholesterol (5.26mmol.L^{-1}–5.31mmol.L^{-1}**;**↑ 0.95 %, $p > 0.05$). Resting systolic and diastolic blood pressures decreased with 6 and 9 mmHg respectfully; resting systolic blood pressure (116 mmHg – 110 mmHg;↓ 4.3%, $p > 0.05$), and resting diastolic blood pressure(77 mmHg – 68 mmHg; ↓11.7%, $p < 0.05$). A reduction in blood pressure by 5 mmHg can decrease the risk of stroke by 34%, ischaemic heart disease by 21%, and reduce the likelihood of dementia, heart failure, and mortality from cardiovascular disease (Law et al., 2003).

The percentage changes recorded in Table 4 indicate that despite the lack of statistical significance (Table 1), there were a number of variables that showed percentage improvements.

It could be argued that percentage improvements whilst not statistically significant are clinically significant. According to Knox (1996) the minimal clinically important reduction represents 23% from the baseline measurement. In Table 2, improvements were observed in the number of individuals whose risk factors fell below the threshold for increase disease risk: systolic blood pressure (↓ 50%) and sedentary lifestyle (↓ 100%).It is believed that people who maintain an active lifestyle have a 45% lower risk of developing a heart disease than people living sedentary life styles (UMMC, 2011). According to Steyn (2007), the chances of suffering a heart attack increases exponentially with multiple risk factors. From baseline to final testing there was a decrease of 47.8% to 43.5% in the participants that fell in the moderate risk category. This showed a9% decrease of the participants that fell into the moderate risk category, thus an increase in the amount of participants in the low risk category. With this, there were an increase in participants in the low risk category from 17.4% to 21.7%, which translate to a 19.8% increase in participants in the low risk category.This is noteworthy when considering that 2% to 4% of participants are likely to migrate from low-risk status to higher risk status within one year in the absence of preventative programmes (Musich et al. 2003). Furthermore, individuals who improve just one risk factor can improve work presenteeism by 9% and reduce absenteeism by 2% (Rowe, 2005).

The observed changes were minimal, and can be attributed to the fact that the intensity and/or frequency of the programme were too low. According to the Institute of Medicine (2002), sedentary individuals would need to perform 200 to 300 min.wk^{-1} of moderate-intensity physical activity for long-term weight loss. A negative energy balance generated by physical activity will result in weight loss and the larger the negative energy balance, the greater the weight loss (Nindl et al., 2007). Several studies that targeted <150 min.wk^{-1} of physical activity resulted in no significant change in body weight (Dengelet al., 1998; Boudouet al., 2003;Donnelly et al., 2009). Donnelly et al. (2009) targeted 90 min of continuous moderate-intensity physical activity (30 min, 3 d.wk^{-1}) compared to 150 min of moderate-intensity intermittent physical activity (30 min, 5 d.wk^{-1}) in women for 18 months. Stevens et al. (2006) recently proposed a definition of weight maintenance as < 3% change in body weight with >5% change in body weight considered as clinically significant. Ross et al. (2000) showed that men and women who experienced a 500- to 700-kcal.d^{-1} deficit for 12 weeks had weight loss of 7.5 kg (8%) and 5.9 kg (6.5%), respectively. It is likely that any increase in physical activity has the potential for weight loss, however it seems that physical activity <150 min.wk^{-1} results in minimal weight loss compared to controls. Physical activity >150 min.wk^{-1} results in modest weight loss of 2 to 3 kg, and physical activity between 225 and 420 min.wk^{-1} results in 5 to 7.5kg weight loss (Donnelly et al. 2009). In addition calorie restriction was prohibited. On examination of the log books, it was clear that the participants were adhering to the exercise programme, but not adapting their diet or restricting their calorie intake. Almost 81% of the participants fully completed their log books, 2 % did not complete their log books, and 17% of the participants did not return their log books.

Although most improvements achieved were non-significant, the percentage changes (reduction) in parameters assessed tend to suggest that a home–based programme without calorie restriction or adjustment may impact favorably on health. Furthermore, the percentage changes indicate that this basic, unsupervised and easy "accessible" intervention programme prevents people from migrating to higher risk (higher-cost) status. We believe that more pronounced significant differences would have been noted with the home-based programme in almost all the parameters if diet was factored in (decreasing calorie intake). Based on the results of this research, it was revealed that the exercise intervention was feasible to use, practical, convenient, and safe (no adverse events were reported) and tended towards improving cardiovascular and metabolic disease risk factors.

CONCLUSION

The goal of the current study was to investigate the effect of a 12-week home-based physical activity intervention programme on cardiometabolic disease risk factors in sedentary adults aged between 20-65 years.

In 2002 the World Health Report estimated that the global prevalence of physical inactivity among adults was at least 17%, and that physical inactivity was the cause of 1.9 million deaths and 19 million disabilities (Vuori, 2007). It has been estimated that 62% of men and 48% of women older than fifteen years were following a sedentary lifestyle in South Africa in 2003. (Steyn, 2007). With inactivity comes the increase in cardiometabolic disease risk factors, namely obesity, metabolic syndrome, type 2 diabetes, hypertension, coronary heart disease, stroke and peripheral arterial disease (Vuori, 2007, ACSM 2010). The worldwide prevalence of cardiometabolic disease places great emphasis on the need for future research to establish the long term effects of physical activity interventions on CMD disease risk factors.

Currently, there exists limited research on the efficacy of home-based exercise intervention in reducing cardiometabolic disease risk factors. Generally people are reluctant to exercise, due to restrictions on the type and amount of food that they eat. As a result of the aforementioned we sought to determine if an un-supervised home based exercise programme without dietary/calorie restriction could impact favorably on CMD risk factors.

The results of the study indicated that the exercise intervention was feasible to use, practical, convenient, and safe and tended towards improving cardiometabolic disease risk factors. There was a trend towards a decline in body composition, BMI and fat percentage. There was a modest decrease in resting systolic blood pressure, and a significant decrease in resting diastolic blood pressure. Improvements were seen in fasting blood glucose. Moreover, several participants moved from moderate cardiovascular disease risk category into the low risk category. Despite the lack of statistical significance, there were a number of variables that showed percentage improvements that from a clinical perspective would be meaningful.

More pronounced benefits may have been seen if the intensity/duration and frequency of exercise were increased. In addition we believe the reasons for the minimal decrease in body weight and BMI, was due to the fact that the participants were not allowed to change their diet at any time.The inclusion of a healthy eating plan may have contributed positively towards altering the participants CMD risk profile. With future research it will be advisable to at least include a calorie restriction of some sort, and or, to increase the intensity and/or frequency of the programme. This programme is certainly accessible to a larger number of people than structured, facility-based intervention programmes.

REFERENCES:

ALBREHT, A.E., MARCUS, B.H., ROBERTS, M., FORMAN, D.E., PARISI, A.F. (1998).Effect of smoking cessation on exercise performance in female smokers participating in exercise training. *American Journal of Cardiology, 82: 950-955.*

ALDANA, S. G., MERRILL, R. M., PRICE, K., HARDY, A., HAGER, R. (2005). Financial impact of a comprehensive multisite workplace health promotion program. *Preventive Medicine, 40:131 – 137.*

AMERICAN COLLEGE OF SPORTS MEDICINE. (2010). Guidelines for exercise testing and prescription – *8th ed. Philadelphia: Lippincot Williams and Wilkins, 2–82p.*

ANDREWS, T.C. (1997). Effect of cholesterol reduction on myocardial ischemia in patients with coronary disease.*Journal of the American Heart Association:Circulation, 95:324–328.*

ASHLEE, K., JANSSEN, I., ROSS, R. (2009). Ability of physical activity to predict cardiovascular disease beyond commonly evaluated cardiometabolic risk factors. *The American Journal of Cardiology, 104: 1522–1526.*

BARRETT-CONNOR, E. (1989).Epidemiology, obesity and non-insulin-dependent diabetes mellitus. *Epidemiology Reviews, 11:172–181.*

BLAIR, S.N., JACOBS, D.R., POWELL, K.E. (1985). Relationships between exercise or physical activity and other health behaviors. *Public Health Reports, 100:172–80.*

BOUDOU, P., SOBNGWI, E., MAUVAIS-JARVIS, F., VEXIAU, P., GAUTIER, J.F. (2003). Absence of exercise-induced variations in adiponectin levels despite decreased abdominal adiposity and improved insulin sensitivity in type 2 diabetic men. *European Journal of Endocrinology*, *149:42–46.*

BOULE, N.G., HADDAD, E., KENNY, G.P., WELLS, G.A., SIGAL, R.J. (2001). Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. *Journal of the American Medical Association, 286:1218–1227.*

BROOKS, G.A., FAHEY, T.D., BALDWIN, K.M.(2005). Exercise Physiology – Human Bioenergetics and Its Applications, *4th ed.* McGraw Hill – Higher Education.

CAMBRIDGE SCIENTIFIC INDUSTRIES – Cambridge, Maryland United States (2000).Lange Skinfold Caliper.

CEDERBERG, H., MIKKOLAA, I.B., JARIJOKELAINENA, C., LAAKSOA, M. C., HÄRKÖNEND, P. IKÄHEIMOA, T., LAAKSOE, M., KEINÄNEN-KIUKAANNIEMIA, S., (2011). Exercise during military training improves cardiovascular risk factors in young men.*Journal of Atherosclerosis, 216: 489–495.*

CENTERS FOR DISEASE CONTROL AND PREVENTION OVERWEIGHT AND OBESITY INTRODUCTION.[Web:] http://www.cdc.gov/nccdphp/dnpa/obesity/index.htm[Retrieved: 14 Nov. 2011].

CHAPMAN, L.S. (2005). Planning wellness: Getting off to a good start. *4thed*. Seattle*, WA.: Summex. 213p.*

COGHILL, N., COOPER, A.R., (2008). The effect of a home – based walking program on risk factors for coronary heart disease in hypercholerolaemia men: A randomized controlled trail. *Preventive Medicine*, *46:545–551.*

COLBERG, S.R. (2005). The Impact of exercise on Insulin Action in type 2 Diabetes Mellitus: Relationship to prevention and control. *Journal of Insulin, 1:66–98.*

COUILLARD, C., DESPRÉS, J.P., LAMARCHE, B., BERGERON, J., GAGNON, J., LEON, A.S., RAO, D.C., SKINNER, J.S., WILMORE, J.H., BOUCHARD, C. (2001). Effects of Endurance Exercise Training on Plasma HDL Cholesterol Levels Depend on Levels of Triglycerides. *Arteriosclerosis, Thrombosis, and Vascular Biology, 21:1226–1232.*

CURB, J.D., ABBOTT, R.D., RODRIGUEZ, B.L. (2004). A prospective study of HDL-C and cholesterol ester transfer protein gene mutations and the risk of coronary heart disease in the elderly. *The Journal of Lipid Research, 45:948–953.*

DEAN, B.B., BORENSTEIN, J.E., HENNING, J.M., KNIGHT, K., MERZ, N.B. (2003). Can change in high-density lipoprotein cholesterol levels reduce cardiovascular risk? *American Heart Journal, 147: 966–976.*

DENGEL, D.R., GALECKI, A.T., HAGBERG, J.M., PRATLEY, R.E. (1998). The independent and combined effects of weight loss and aerobic exercise on blood pressure and oral glucose tolerance in older men. *American Journal of Hypertension*, *11:1405–12*.

DIABETES CARE. (2011). Benefits of Modest Weight Loss in Improving Cardiovascular Risk Factors in Type 2 Diabetes. *Complications and Co-morbidities, Obesity, Type 2 Diabetes, Issue 575.*

DOLL, R., PETO, R., BOREHAM, J., SUTHERLAND, I. (2004). Mortality in relation to smoking: 50 years' observations on male British doctors. *British Medical Journal, 328: 1519–1533.*

DOLL, R., PETO, R., WHEATLEY, K., GRAY, R., SUTHERLAND, I. (1994). Mortality in relation to smoking: 40 years' observations on male British doctors.*British Medical Journal, 309: 901–911.*

DONNELLY, J.E., BLAIR, S.N., JAKICIC, J.M., MANORE, M.M., RANKIN, J.W., SMITH, B.K. (2009). Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults.*Journal of the American College of Sports Medicine, 41: 459–471.*

DURSTINE, J.L., MOORE, G.E. (2003). Exercise Management for Persons with Chronic Diseases and Disabilities.*Clinical Exercise Physiology, 76–149p.*

FERREIRA, M. (2006). The wellness programmes of selected companies in South Africa. Port Elizabeth: Nelson Mandela Métropolitain University. *Dissertation,138p.*

FERRUCCI, L., IZMIRLIAN, G., LEVEILLE, S., PHILLIPS, C.L., CORTI, M.C., BROCK, D.B., GURALNIK, J.M. (1999).Smoking, physical activity, and active life expectancy. *American Journal of Epidemiology, 149: 645–53.*

FORD, E.S. (2002). Prevalence of the metabolic syndrome among US adults: findings from the Third National Health and Examination Survey. *Journal of the American Medical Association, 287:356–359.*

GAZIANO, T.A.(2007).Economic burden and the cost-effectiveness of treatment of cardiovascular diseases in Africa. Division of Cardiovascular Medicine, Brigham & Women's Hospital. *Journal of Heart and Education in Heart, 94: 140–144.*

GEORGE, P. N., PAPANTAKOU, K., SKENDERI, K., TRIANDAFILLOPOULOU, M., KAVOURAS, S. A., YANNAKOULIA, M., CHROUSOS, G. P., SIDOSSIS L. S. (2005). Aerobic exercise training improves insulin sensitivity without changes in body weight, body fat, adiponectin, and inflammatory markers in overweight and obese girls. *Journal ofMetabolism, 54: 1472–1479.*

GOLDBERG, L., ELLIOT, D. L., SCHUTZ, R. W., KLOSTER, F. E. (1984). Changes in lipid and lipoprotein levels after weight training.*Journal of the American Medical Association, 252: 504–507.*

GOULD, B.E., DYER, R.M. (2011).Pathophysiology for the Health Professions.4th ed. Saunders, Elsevier.

GRACE, J.M. (2006). The effect of a physical and combined health promotion intervention programme on some selected health indicators of South Africa colliery executives. *1:2–72.*

GROENEWALD, P., VOS, T., NORMAN, R., LAUBSCHER, R., VAN WALBEEK, C., SALOOJEE, Y., SITAS, F., BRADSHAW, D. (2007).Estimating the burden of disease due to smoking in South Africa, 2000.*South African Medical Journal, 97: 674–681.*

HALBERT, J.A., SILAGY, C.A., FINUCANE, P., WITHERS, R.T., HAMDORF, P.A. (1999). Exercise training and blood lipids in hyperlipidaemic and normolipidaemic adults: a meta-analysis of randomised, controlled trials. *European Journal Clinical Nutrition, 7: 514–522.*

HEDBLAD, B., OGREN, M., ISACSSON, S. O., JANZON, L. (1997).Reduced cardiovascular morality risk in male smokers who are physically active. *Archives of Internal Medicine, 157: 893–899.*

HILL, J. S. (1981). Health behaviour: the role of exercise in smoking cessation. *Chapter Journal, 28: 15–18.*

HUGHES, J.R., HIGGINS, S., BICKEL, W. (1994). Nicotine versus other withdrawal syndromes: similarities and dissimilarities.*Journal ofAddiction, 89: 1461–1470.*

HUGHES, J. R., GULUVER, S. B., FENWICK, J. W. (1992). Smoking cessation among self-quitters. *Health Psychology, 11: 331–334*.

INSTITUTE OF MEDICINE. (2002). Dietary Reference Intake for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids. Washington (DC): Washington National Academic Press.

JONES, M., NIES, M.A. (1996). The relationship of perceived benefits of and barriers to reported exercise in older African American women. *Public Health Nursing*, *13:15–18.*

JOHN D. BRUNZELL, J.D., DAVIDSON, M., FURBERG, C.D., GOLDBERG, R.B., HOWARD, B.V., STEIN, J. H.,WITZTUM, J. L. (2008). Lipoprotein management in patients with cardiometabolic risk. *Diabetes Care, 31: 4 811–822.*

KAWACHI, I., TROISI, R. J., ROTNRRZKY, A. G.,COAKLEY, E. H., COLDRRZ, M. S., COLDRRZ, M.D. (1996). Can exercise minimise weight gain in women after smoking cessation. *American Journal of Public Health, 86: 999–1004.*

KELLEY, G.A., KELLEY, K.S. (2006). Aerobic exercise and lipids and lipoproteins in children and adolescents: A meta-analysis of randomized controlled trials.*Journal of Atherosclerosis, 191: 447–453.*

KING, T. K., MARCUS, B. H., PINTO, B. M., EMMONS, K. M., ABRAMS, D. B. (1996). Cognitive behavioral mediators of changing multiple behaviours: smoking and a sedentary lifestyle. *Preventive Medicine, 25: 684–691.*

KNOX. H. (1996). Clinical versus statistical significance in the assessment of pain relief.*Annals of Emergency Medicine. 27: 439–441.*

KOVAR, M.G., HARRIS, M.I., HADDEN, W.C. (1987). The scope of diabetes in the United States population. *American Journal of Public Health, 77:1549–1550.*

KROUSEL-WOOD, M.A., BERGER, L., JIANG, X., BLONDE, L., MYERS, L., WEBBER, L. (2008).Does home-based exercise improve body mass index in patients with type 2 diabetes? *Diabetes Research and Clinical Practice*, *79:230–236.*

KWON, H.S., CHO, J.H., KIM, H.S., LEE, J.H., SONG, B.R., OH, J.A. (2006).Development of web-based diabetes patient management system using short message service. *Diabetes Research and Clinic Practice, 66:133–137.*

LAVIE, C.J., MILANI, R.V., VENTURA, H.O. (2009). Obesity and Cardiovascular Disease: Risk Factor, Paradox, and Impact of Weight Loss. New Orleans, Louisiana.*Journal of the American College of Cardiology, 53: 1925–1932.*

LAW, M., WALD, N., MORRIS, J., (2003).Lowering blood pressure to prevent myocardial infarction and stroke: a new preventive strategy. *Health Technology Assessment 7: 1–94.*

LOOK AHEAD RESEARCH GROUP. Reduction in weight and cardiovascular disease risk factors in individuals with type 2 diabetes: one year results of the Look AHEAD Trial. (2007). *Diabetes Care,30: 1374–1383.*

MAGNUSSON, R.S. (2009). Rethinking global health challenges: Towards a 'global compact' for reducing the burden of chronic disease. *Public Health, 123:265–274.*

MANSON, J. E., TOSTERSON, H., RIDKER, P. M., SATTERFIELD, S., HEBERT, P., G.T., O., BURING, J. E., HENNEKENS, C. H. (1992).The primary prevention of myocardial infarction. *The New England Journal of Medicine, 326: 1406–1416*.

MARBLE, A., KRALL, L.P., BRADLEY, R.F., CHRISTLIEB, A.R., SOELDNER, J.S. (1985). Joslin's diabetes mellitus. 12th ed. Philadelphia: Lea & Febiger.

MCARDLE, W.D., KATCH, F.I., KATCH, V.L. (2001). Exercise physiology: Energy, nutrition and human performance,*5th ed. Philadelphia, PA: Lippincott, Williams and Wilkins. 198p.*

MCARDLE, W.D., KATCH, F.I., KATCH, V.L. (2007). Exercise Physiology: Energy, nutrition and human nutrition, *6th ed*. Philadelphia, PA: *Lippincott, Williams and Wilkins, 17–820p.*

MCAULEV, E., MHALKO, S. L., BANE, S. M. (1997). Exercise and self-esteem in middle-aged adults: multidimensional relationships and physical fitness and self-efficacy influences. *Journal of Behavioral Medicine, 20: 67–83.*

MEDICAL RESEARCH COUNSEL. (2010). Cardiovascular disease.[Web:] http://www.mrc.ac.za/pressreleases/2010/cardiovascular diseasemd_speech_ali.htm [Retrieved: 17 Sep. 2010, author unknown].

MUSICH, S., HIRSCHLAND, D., MCDONALD, T., EDINGTON, D.W. (2003). Examination of risk status transitions among active employees in a comprehensive worksite health promotion program. *Journal of Occupational and Environmental Medicine,45:393–399.*

NINDL, B.C., BARNES, B.R., ALEMANY, J.A., FRYKMAN, P.N., SHIPPEE, R.L., FRIEDL, K.E. (2007).Physiological consequences of U.S. Army Ranger training. *Medicine of Sports Science and Exercise,39:1380–1387.*

O'CONNOR, P. J., YOUNGSTEDT, S.D. (1995). Influence of exercise on human sleep. *Exercise and Sport Science Reviews, 2: 105–134.*

OUNIS, O.B., ELLOUMI, M., CHIEKH, I.B., ZBIDI, A., AMRIC, M., LACB, G., TABKA, Z. (2008).Effects of two-month physical-endurance and diet-restriction programmes on lipid profiles and insulin resistance in obese adolescent boys. *Diabetes and Metabolism, 34: 595–600.*

PAFFENBARGER, R.S., HYDE, R.T., WING, A., LEE, M.I., JUNG, D., KAMPERT, J. (1993). The association of changes in physical-activity and other lifestyle characteristics with mortality among men. *The New England Journal of Medicine, 328:538–545.*

PERRI, M.G., MARTIN, A.D., LEERMAKERS, E.A., SEARS, S.F., NOTELOVITZ, M. (1997). Effects of group- versus home-based exercise in the treatment of obesity. *Journal of Consulting and Clinical Psychology, 65:278–285.*

PESTANA, J.A., STEYN, K., LEIMAN, A., HARTZENBERG, G.M.(1996).The direct and indirect costs of cardiovascular disease in South Africa in 1990. *South African Medical Journal*, *86: 679–684.*

PETO, R. (1994). Smoking and death: the past 40 years and the next 40. *British Medical Journal, 309: 937–939.*

PETO, R., LOPEZ, A.D., BOREHAM, J., THUN, M. (1992). Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *The Lancet Heath Care Journal, 339:1268–1278.*

PIGMAN, H.T., GAN, D.X., KROUSEL-WOOD, M.A. (2002). Role of exercise for type 2 diabetic patient management. *Southern Medical Journal,95:72–77.*

RIMMER, J.H., RILEY, B., WANG, E., RAUWORTH, A., JURKOWSKI, J. (2004).Physical activity participation among persons with disabilities.*American Journal Preventive Medicine*, *26:419–425.*

ROBERTS, C.K., BARNARD, J. (2005).Effects of exercise and diet on chronic disease. *Journal of Applied Physiology,98: 3–30.*

ROBIN, E. S., LEEKS, K.D., RAZI, S., HOPKINS, D. P., GRIFFITH, M. (2010). A systematic review of selected interventions for worksite health promotion. The assessment of health risks with feedback. *American Journal of Preventive Medicine, 38: 237–262.*

Roche Diagnostics GmbH D – 68298 Mannheim Germany, (2000). Accutrend GCT. User's Manual.

ROSS, R., DAGNONE, D., JONES, P.J.H., SMITH, H., PADDAGS, A.,HUDSON, R. (2000). Reduction in obesity and related co morbid conditions after diet-induced weight loss or exercise induced weight loss in men. *Annals of Internal Medicine,133: 92–103.*

ROWE, J.W. (2005). Aetna Inc.: Healthy Lifestyles. Partnership for Prevention. ed. Leading by example: Washington, DC. USA.

SANTA-CLARA, H., FERNHALL, B.O., BAPTISTA, F.A., MENDES, M., BETTENCOURT S. L. (2003). Effect of a One-Year Combined Exercise Training Program on Body. Composition in Men with Coronary Artery Disease. *Journal ofMetabolism, 52: 1413–1417*.

SCHUTZER, K.A., GRAVES, B.S. (2004).Barriers and motivations to exercise in older adults.*Preventive Medicine*. *39:1056–1061.*

SEEDAT, Y.K., CROASDALE, M.A., MILNE, F.J., OPIE, L.H., PINKNEY-ATKINSON, V.J., RAYNER, B.L., VERIAVA, Y. (2006). Joint National Hypertension Guideline Working Group. South African Hypertension Guidelines. *South African Medical Journal, 96: 337–362*.

SHARMAN, J.E., STOWASSER, M. (2009). Australian association for exercise and sports science position statement on exercise and hypertension. *Journal of Science and Medicine in Sport,12: 252–257.*

SHARRETT, A.R., BALLANTYNE, C.M., COADY. S.A. (2001). Coronary heart disease prediction from lipoprotein cholesterol levels, triglycerides, lipoprotein, apolipoproteins A-I and B, and HDL density sub fractions: the Atherosclerosis Risk in Communities Study. *Journal of the American Heart Association:Circulation,104:1108–1113.*

SORENSON, G., GOLDBERG, R., OCKENE, J., KLAR, J., TANNENBAUM, T., LHMESHOW, S. (1992). Heavy smoking among a sample of employed women. *American Journal of Preventive Medicine, 8: 207–214.*

STEVENS, J., TRUESDALE, K.P., MCCLAIN, J.E. & CAI, J. (2006).The definition of weight maintenance. *International Journal of Obesity*, *30:391–399.*

STEYN, K. (2007). The heart and stroke foundation South Africa: Heart disease in South Africa. Department of Medicine, University of Cape Town and Chronic Diseases of Lifestyle Unit.*Medical Research Council.*

THOMAS, W., BOYDEN, T.W., PAMENTER, R.W., GOING, S.B., LOHMAN, T.G., HALL, M. C., HOUTKOOPER, L. B., BUNT, J.C., RITENBAUGH, C., AICKIN, M. (1993). Resistance Exercise Training Is Associated With Decreases in Serum Low-Density Lipoprotein Cholesterol Levels in Premenopausal Women. *Archives of Internal Medicine, 153:97–100.*

UNIVERSITY OF MARYLAND MEDICAL CENTER. (2011). Peripheral artery disease. [Web:]http://www.umm.edu/patiented/articles/medications_used_peripheral_artery_disease_intermittent_claudication_000102_7.htm [Retrieved: 28 June. 2011].

UNIVERSITY OF NEW MEXICO. (UNM)(2011). Cholesterol. [Web:]http://www.unm.edu/~lkravitz/Article%20folder/cholesterol.html [Retrieved: 18April .2011, KRAVITZ, L., HEYWARD, V.].

USSHER, M.H., TAYLOR, A.H., WEST, R., McEWEN, A. (2000). Does exercise aid smoking cessation? *Journal of Addiction, 95: 199–208.*

VUORI, I. (2007). Physical activity and health: Metabolic and cardiovascular issues. *Advances in Physiotherapy. 9:50–64.*

WALLACE, A.G. (1986). Fitness, health and longevity: A question of cause and effect. *Inside tract, 2:3.*

WANKO, N.S., BRAZLER, C.W., YOUNG-ROGERS, D., DUNBAR, V.G., BOYD., B. GEORGE, C.D. (2004). Exercise preferences and barriers in urban African Americans with type 2 diabetes.*Diabetes Education, 30:502–513.*

WATTS, K., BEYE, P., SIAFARIKAS, A. (2004).Effects of exercise training on vascular function in obese children. *Journal of Pediatrics, 144: 620–625.*

WATTS, K., BEYE, P., SIAFARIKAS, A. (2004).Exercise training normalizes vascular dysfunction and improves central adiposity in obese adolescents. *Journal of the American College of Cardiology, 43: 1823–1827.*

WEGGEMANS, R.M. (2001).Dietary cholesterol from eggs increases the ratio of total cholesterol to high-density lipoprotein cholesterol in humans: a meta-analysis. *American Journal of Clinical Nutrition, 73:885–891.*

WEST, R. (1999). Getting Serious About Smoking Cessation: a review of products services and techniques. London, No Smoking Day Report.*Journal of Drug and Therapeutics Bulletin, 37: 52–54.*

WHELTON, P., CHIN, A., XIN, X., HE, J. (2001).Effect of aerobic exercise on blood pressure: a meta-analysis of randomised controlled trials. *Annals of Internal Medicine, 136: 493–503*.

WILBUR, J., VASSALO, A., CHANDLER, P., MCDEVITT, J., MILLER, A.M. (2005). Midlife women's adherence to home-based walking during maintenance.*Nursing Research,54:33–40.*

WOOD, P. O., HASKELL, W. L. (1979).The effect of exercise on plasma high density lipoprotein.*Lipids, 14: 417–427.*

YUHASZ, M.S. (1974). Physical fitness manual. Ontario: University of Western Notario.

APPENDIX

Appendix A

UNIVERSITY OF ZULULAND
DEPARTMENT OF BIOKINETICS AND SPORTS SCIENCE

INFORMED CONSENT

Explanation of the tests:
You will be assessed on a series of tests, such as your height, weight, resting heart rate and blood pressure. Your percentage body fat measurement will be taken, as well as your hip to waist ratio. There will be two blood tests done, fasting glucose and cholesterol; with thisyour finger will be pricked to assess us to obtain a blood drop for each test.

Risks and Discomforts:
The possibility of certain changes during the tests may occur which include abnormal blood pressure, fainting or irregular heart rhythmin rare instances. There may be some discomfort in your finger when the blood tests are done. Every effort will be made to minimize these risks.

Responsibilities of the Patient:
Information you possess about your health status or of previous experiences of unusual feelings related to physical activity that may hinder safety and value of the study/programme must be disclosed. Your prompt report of feelings with effort during the study/programme itself is of great importance. You are responsible of therefore fully disclosing such information when requested by the tester.

Benefits to be expected:
The results obtained from the study/programme may potentially assist in the diagnosis of any illness or help you lose weight and adopt a healthier lifestyle.

Inquiries:
Any questions about the procedures used in the study/programme or the result of the study/programme are encouraged. If there are any concerns or questions, please ask us for further explanations. Please feel free to contact my supervisor Prof. Stuart J. Semple at 079 490 0977 or E-Mail him at (ssemple@pan.uzulu.ac.za).

Freedom of consent:
Your permission to perform this test is voluntary. You are free to stop the testing at any time, if you so desire.

I have read this form and understand the test procedures that I will perform, as well as the related risks and possible discomforts. With full knowledge of this, and having had an opportunity to ask questions that have been answered to my satisfaction, I consent to participate in this testing procedure

________________________	________________________	______________
Client	Signature	Date
________________________	________________________	______________
Tester	Signature	Date

Appendix B

Data Sheet				
Tester:				
Name:				
Surname:				
Occupation:				
Date of Birth:				
Date of Test:				
Test Number:	**1**	**2**	**3**	**4**
Weight:				kg
Height:				cm
BMI:				
Waist:				cm
Hip:				cm
Skin folds:				
Triceps				mm
Suprailiac				mm
Subscapular				mm
Calf				mm
Abdominal				mm
Thigh				mm
Total				mm
Fat%				%
Resting BP:				mmHg
Resting HR:				bpm
Blood Glucose:				mmol/L
Blood Cholesterol:				mmol/L

Appendix C

UNIVERSITY OF ZULULAND **DEPARTMENT OF BIOKINETICS & SPORT SCIENCE**		Website: http**://www.uzulu.ac.za** Private Bag X1001 3886 KwaDlangezwa Tel: 035 9026396 Fax: 035 9026386 E-Mail: gerrit-3@hotmail.com

1. Has anyone in your family (parents, grandparents, brothers, sisters) have had any of the following?

	NO	YES	UNSURE
Heart problems			
Stroke			
High blood pressure			
Diabetes			
High cholesterol			
Obesity			

2. Do you have, or have you been previously diagnosed with any of the following?

	NO	**YES**	**UNSURE**
1.1 Heart problems			
1.2 Palpitations			
1.3 High cholesterol			
1.4 High blood pressure			
1.5 Diabetes			
1.6 Asthma or pulmonary diseases			
1.7 Epilepsy			
1.8 Hernias			

1.9 Arthritis			
1.10 Osteoporosis			
1.11 Cancer			
1.12 Rheumatic fever			
1.13 Lower back pain			
1.14 Pregnancy			
1.15 Depression			
1.16 Stress			
1.17 Ulcer			

3. Do you currently smoke? Y / N
(If yes, how much? If no, when did you stop, if smoked before)

4. Do you take any chronic medication? Y / N

Medication	**Reason**

5. Is there any physical state, including any joint or muculoskeletall problems that I have to consider before you start an exercise programme? Y / N

6. Is there any other reason, not mentioned above, why you cannot follow an exercise programme or undergo an exercise test? Y / N

If you answered yes on any of the abovementioned questions, please specify below.

NR	EXPLANATION

I, ____________________ have completed the questionnaire and understand all the questions. I have had the opportunity to discuss all unclear aspects with the Biokineticist. I hereby give my permission to be evaluated and agree to follow the prescribed exercise programme.
I further agree that I or any of my relatives, executor, administrator or legal representative will not impose any claim against the Biokineticist or practice, except in case of negligence or malpractice by the Biokineticist.

I understand that I am using the facilities and equipment at my own risk.

Signature: ____________________ Date: __________

Witness: _____________________ Date: __________

Appendix D

International Physical Activity Questionnaire

- Below are questions about individual's physical activity levels.
- Please read the descriptions and answer the questions even if you do not consider yourself to be an active person.
- Consider all activities, those you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Hard physical activity:

Think about all the **vigorous activities** which take hard physical effort that you did in the **last 7 days.** Vigorous activities make you breath harder than normal and may include heavy lifting, aerobic, or fast bicycling. Think only about those physical activities that you did for at least 10 minutes at a time.

1. During the last 7 days, on how many days did you do vigorous physical activities? **_____days/week _____don't know/not sure**
2. How much total time did you usually spend doing vigorous physical activities on one of those days? **_____hours/day _____minutes/day _____don't know/not sure**
3. if your pattern of activity varies from day to day, how much total time did you spend over the last 7 days doing vigorous physical activity. **_____hours/week _____minutes/week _____don't know/not sure**

Moderate physical activity:

Think about the activities which take **moderate physical effort** that you did in the last 7 days. Moderate physical activity makes you breath somewhat harder than normal and may include carrying light loads, bicycling at a regular pace, or double tennis. Do not include walking. Again, think about only those physical activities that you did for at least 10 minutes.

4. During the last 7 days, on how many days did you do moderate physical activities?

_____days/week _____don't know/not sure

5. How much total time did you usually spend doing moderate physical activities on one of those days?

_____hours/day _____minutes/day _____don't know/not sure

6. If your pattern of activity varies from day to day or includes multiple tasks, how much total time did you spend over the last 7 days doing moderate physical activity?

_____hours/week _____minutes/week _____don't know/not sure

Walking:

Now think about the time you spend **walking** in the **last 7 days.** This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise or leisure.

7. During the last 7 days, on how many days did you walk for at least 10 minutes at a time?

_____days/week _____don't know/not sure

8. How much total time did you usually spend walking on one of those days?

_____hours/day _____minutes/day _____don't know/not sure

9. if your pattern of activity varies from day to day or include multiple tasks, how much total time did you spend walking over the last 7 days?

_____hours/week _____minutes/week _____don't know/not sure

Sitting:

Finally, think about the time you spent **sitting** on weekdays during the **last 7 days.**
Include time spent at work, at home, while doing course work, and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, sitting or lying down to watch television.

10. During the last 7 days how much total time did you usually spend sitting on a week day?

_____hours/weekday _____minutes/weekday _____don't know

Appendix E

UNIVERSITY OF ZULULAND **DEPARTMENT** **OF** **BIOKINETICS & SPORT SCIENCE**		**Website:** **http://www.uzulu.ac.za** Private Bag X1001 3886 KwaDlangezwa Tel: 035 9026391 Fax: 035 9026386 E-Mail: gerrit-3@hotmail.com

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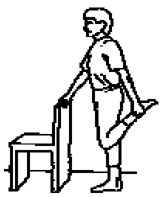
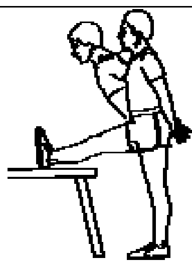
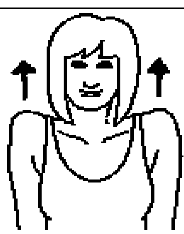
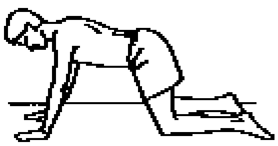
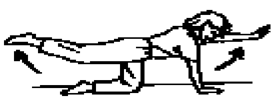
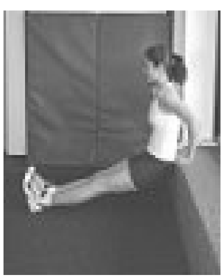
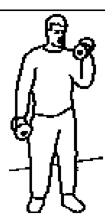
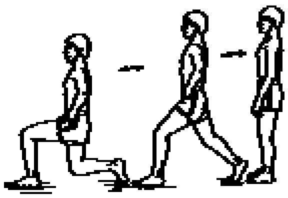
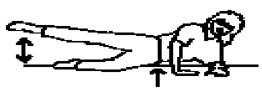
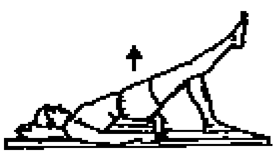
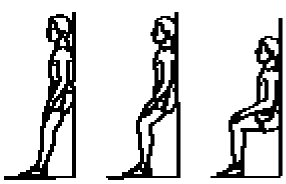
NB>>Cardiovascular exercises: Jogging, Walking, for **20 minutes a**t medium intensity.

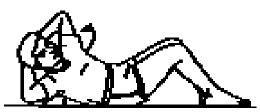
It can also be done in intervals for instance 2 minutes fast then 1 minute slow for the whole **20 minutes**.

Exercise	**Description**	**Sets**	**Repetitions**
Flexibility Exercises: (before & after)			
	**Shoulder or Deltoid stretch** Stand or sit. Stretch one arm over to the opposite shoulder by pushing it at the elbow with your other arm.	**3**	**20 sec**

	**Neck stretch** Pull head wtih hand towards shoulder	**10**	**5 sec**
	**Quadricep stretch** Stand holding on to a support. Bend one knee and take hold of the ankle. Do not lock the knee of the leg you are standing on. Draw your heel towards your buttock. Tilt your hip forwards so that your knee points towards the floor. Feel the stretch in the front of your thigh.	**3**	**20 sec**
	**Hamstring stretch** Stand with one leg on a chair (straight). Bend your upper body forward until you feel the stretch.	**3**	**20sec**
UPPER BODY STENGTHENING EXERCISES			
	**Shoulder shrugs** Hold a weight (2L-5L bottle) at your sides. Pull up with shoulders (shrug).	**3**	**15-12-10**

	**Push-Ups** Lying face down with your hands and knees on the floor at shoulder height. Bend your elbows with your chest fist height off the floor and straighten your elbows again. Do push-ups slowly and remember to straighten your elbows properly.	**2**	**20**
	**Superman's** On all fours. Lift opposite arm and leg to horizontal position. Try to keep your body still. Immediately repeat to other side.	**2**	**12**
	**Triceps dips** Stand with your back to a firm bench or support. Place your hands (shoulder-width apart and fingers facing forward), behind you close to your buttocks. Extend your legs with knees slightly bent so that you're balancing on your heels, toes pointing upwards. Squeeze your shoulder blades down and together to hold your body up. Keep your back straight and your torso vertical. Keeping buttocks as close to the bench as possible, bend elbows until you feel tension in the shoulders. Straighten arms but don't lock elbows.	**3**	**15-12-10**
	**Elastic bicep curl (or with water bottle)** Stand holding elastic tubing with hand and the other tied to foot as shown or use a 1L-2L bottle as a resistance. Keep palm facing upwards as shown. Bend elbow as shown, slowly lower	**3**	**12-10-8**

LOWER BODY STRENGTHENING EXERCISES			
	**Lunges** Stand with your feet shoulder width apart. Step forwards and down with your right leg, allowing your left knee to come towards the level of your right foot. Finish the one side and repeat towards the other side.	**3**	**12-10-8 x weight**
	**Strengthening of the abductors** Lie on side. Lift and lower the upper leg.	**1**	**15 - 20**
	**Pelvic lift with straight leg** Lie on your back with one knee bent, straighten your other leg. Squeeze your buttocks together and lift your bottom off the floor. Return to starting position.	**10**	**10sec each side**
	**½ Squats with weight** Stand with back against wall; feet shoulder width apart and 40cm from the wall. Slowly slide down wall until you are in a "chair position". Push back up but do not “lock out” your knees. Repeat for the number of repetitions.	**3**	**12-10-8 x weight**
	**Standing calf raise** Stand on both feet on a step with both of your heels over the edge. Let your heels drop downwards. Push up on your toes.	**1**	**12-10-8 x weight**
	Sit ups in 3 levels Lie on your back, push your lower back against the floor and keep your eyes on the roof. Do sit-ups as	**3**	**12**

	follow: 1. Hands behind head 2. Hands 90 ° in air 3. Hands to sides **Every level bit higher up**		
	**Crossover Sit-Ups** Lie on back with knees bent. Place hands behind head. Without pulling with your hands, raise head and shoulders, curl trunk upward and to the side as shown. Finish on the one side and repeat in opposite direction.	**3**	**12**

Do your upper bodies exercise 3x per week, your lower body exercises 3x per week and your cardiovascular exercises 3x per week.

NB >>

- **Do your exercises with slow controlled movements!**
- **Remember to breathe out during the hard part (lifting or pushing) and breathe in during the relaxation part.**
- **Rest at least 1 minute in between sets.**
- **Stretch before and after training.**
- **Contact me if you experience any problems or have any questions.**
- **If you experience any pain, stop and contact me!**

Gerrit Breukelman

Biokinetics

Appendix F

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Turin, 23/02/2012

We declare that paper authored by

BREUKELMAN GERRIT JAN
SEMPLE S.J.
GRACE J.M.

entitled

THE EFFECT OF A 12 WEEK HOME-BASED EXERCISE INTERVENTION PROGRAM ON CARDIOMETABOLIC DISEASE RISK FACTORS

Has been accepted and will be published in the journal GAZZETTA MEDICA ITALIANA.

per The Managing Editor
Prof. Alberto Oliaro

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10126 TORINO

Appendix G

1. 14th Biennial South African Sports Medicine Congress (18 - 20 October 2011)
   Sandton Convention Centre, Johannesburg
   SASMA President & Congress Convenor - Dr Jon Patricios
   - Free Communications - Exercise in Chronic Disease & Occupational Health (15h00–17h00, Wednesday 19 October 2011 in the Boardroom)
     - The effect of a 12 week home-based exercise intervention program on cardiometabolic disease risk factors - Mr Gerrit Breukelman

2. 6th Annual Faculty of Science &Agriculture Research Symposium (27 October 2011)
   At the University of Zululand, Department of Biokinetics and Sport Science
   Theory Meets Practice
   - (08h45–10h15, Thursday 27 October 2011 in the Large Lecture Hall)
   - The effect of a 12 week home-based exercise intervention program on cardiometabolic disease risk factors - Mr Gerrit Breukelman