

**Effect of a 12-week aerobic exercise programme on percentage
body fat, fasting blood glucose and dyspnoea in insulin resistant,
obese female university employees in the Western Cape.**

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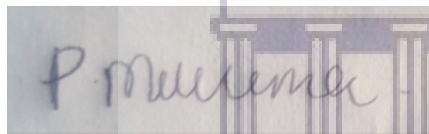
DECLARATION

I, Maphoko Phindile Malema hereby declare that “Effect of a 12-week aerobic exercise programme on percentage body fat, fasting blood glucose and dyspnoea in insulin resistant, obese female university employees in the Western Cape “is my own work and it has not been submitted for any other degree or University.

MAPHOKO PHINDILE MALEMA

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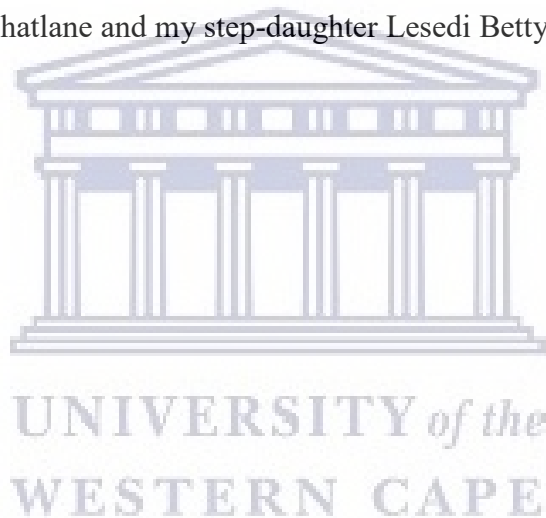
SIGNED:

A rectangular box containing a handwritten signature in blue ink that reads "P. Malema". The signature is written in a cursive style.

DEDICATIONS

I dedicate this thesis to my husband Makhaya Johannes Malema, who helped me in every way possible to understand research principles. Thank you for your support, love and patience, all this means more than I can explain in any mortal language or human understanding. I love you so much.

I also wish to dedicate this thesis to my mother, Reinett Phatlane. Your prayers, love and humility really pushed and inspired me to do the best that I can in life and my academics. My love and respect for you goes beyond eternity. Lastly, I dedicate this thesis to my two sisters Zama and Ntombizodwa Phatlane and my step-daughter Lesedi Betty Malema. I love you all.



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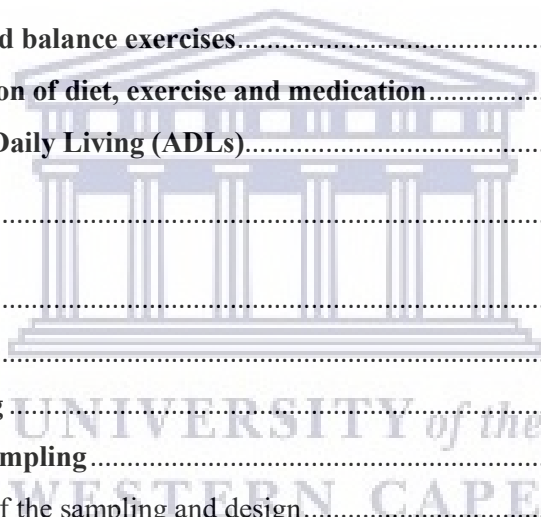
And lastly, my Mother, two sisters and my daughter whom have been my biggest fans from day one. I am very blessed to have you as a family.



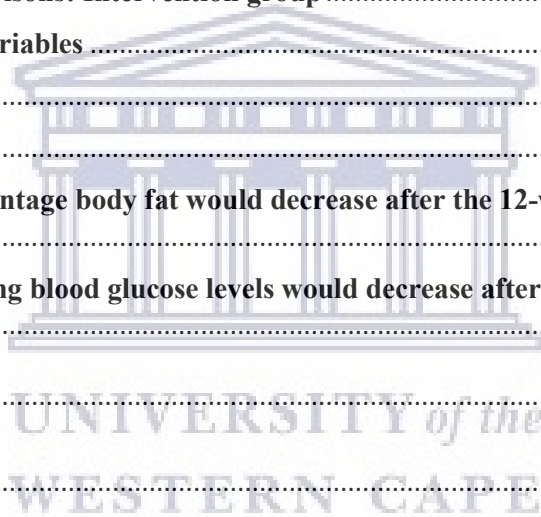
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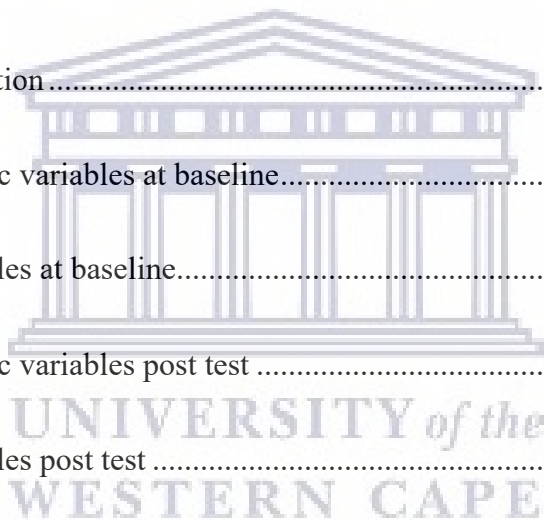
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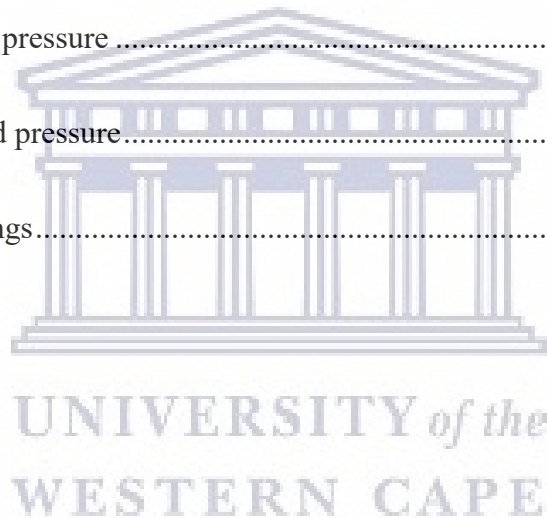
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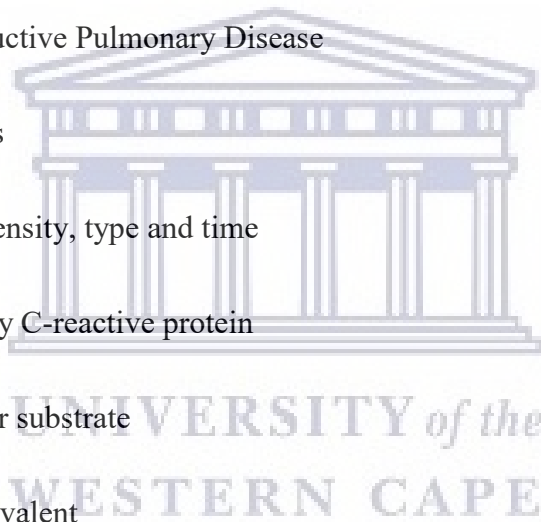
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LIST OF ABBREVIATIONS

ACSM	American College of Sports Medicine
ADA	American Diabetes Association
ADLs	Activities of daily living
ADP	Air displacement plethysmography
BMI	Body mass index
CNS	Central nervous system
COPD	Chronic Obstructive Pulmonary Disease
FFAs	Free fatty acids
FITT	Frequency, intensity, type and time
HS-CRP	High-sensitivity C-reactive protein
IRS	Insulin receptor substrate
MET	Metabolic equivalent
PA	Physical activity
PWS	Prader-Willi syndrome
REM	Rapid eye movement
RPM	Revolutions per minute
SADHS	South African Demographic and Health Survey



T2DM	Type 2 diabetes mellitus
TNF- α	Tumor necrosis factor
WHO	World Health Organization
WHtR	Waist-to-height ratio



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DEFINITION OF TERMS

Obesity in this study is defined as body mass index $\geq 30 \text{ kg} \cdot \text{m}^{-2}$ and central obesity as a waist circumference $> 88 \text{ cm}$ in women and greater than 102 cm in men (WHO, 2000).

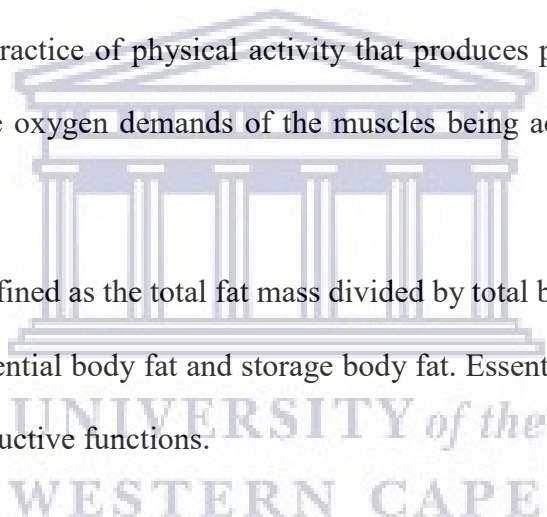
Type 2 diabetes is referred to as a chronic illness marked by decreased insulin sensitivity and overall poor glucose control (Snowling & Hopkins, 2006).

Dyspnoea is defined as an aversive and threatening subjective experience of breathing discomfort that differs from person to person (Sucec, Herzog, Van Diest, Van den Bergh, & von Leupoldt, 2018).

Aerobic training is any practice of physical activity that produces prominent heart rate and breath volume to meet the oxygen demands of the muscles being activated (Patel & Bhise, 2017).

Percentage body fat is defined as the total fat mass divided by total body mass, multiplied by 100; body fat includes essential body fat and storage body fat. Essential body fat is necessary to maintain life and reproductive functions.

Normal fasting blood glucose is defined as concentration kept within a narrow physiological range of $3.5\text{-}5.5 \text{ mmol} \cdot \text{L}^{-1}$ (Rahman & Hussain, 2016).



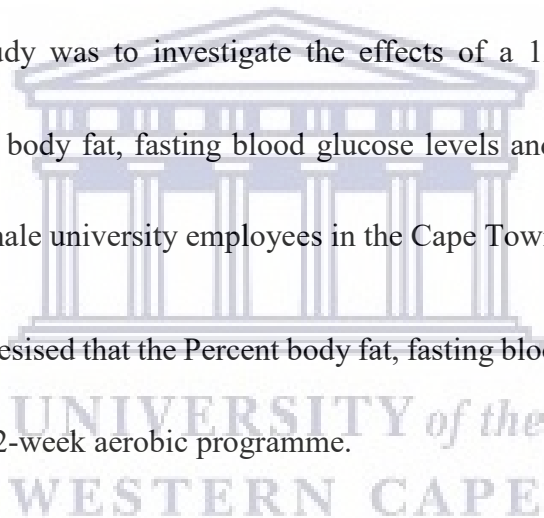
ABSTRACT

Background: Obesity is recognised as a risk factor for non-communicable diseases which has reached epidemic proportions globally. South Africa is one of the developing countries with significant statistical representation reported for these conditions. Obesity is associated with other conditions such as type 2 diabetes, hypertension and dyslipidaemia which are all part of what is called metabolic syndrome. As a strategy to reduce the levels of obesity, physical activity has been introduced to compliment clients who are on medication for diabetes.

Aim: The aim of this study was to investigate the effects of a 12-week aerobic training programme on percentage body fat, fasting blood glucose levels and ratings of dyspnoea in insulin resistant, obese female university employees in the Cape Town metropole.

Hypothesis: It was hypothesised that the Percent body fat, fasting blood glucose and dyspnoea would decrease after the 12-week aerobic programme.

Methods: The study utilised a quantitative approach with a pre-test post-test design and an intervention of an aerobic exercise programme for 12 weeks. Participants were recruited using convenience sampling from the staff at a university. The intervention exercise training programme consisted of moderate-intensity exercise for 45 minutes (which included a 5-minute warm-up and 5-minute cool down), 3 days a week for 12 weeks. Pre-test, all participants underwent a battery of tests, including fasting blood glucose, blood pressure, height, weight, body composition and aerobic capacity, which were repeated again after the 12-week



intervention period was complete. Ethics clearance was obtained from the institution's Research Ethics Committee (Ethics number: BM 17/8/11) and participants were asked to give written consent before taking part in the study. It was hypothesised that a 12-week aerobic exercise programme would reduce the percentage of body fat, blood glucose and dyspnoea in insulin resistant obese female university employees.

Results: In comparing the dependent variables between the control and intervention groups, the findings of the current study indicate that the exercise intervention was a success, given the statistically significant changes which occurred in the intervention group, and those values that were not significant, certainly improved. This can be considered clinically significant, as with diabetics, any positive change in FBG levels, blood pressure and/or body composition is indeed a good outcome. Simultaneously, the control group showed some concerning results, which highlights the unhealthy consequences of T2DM and a sedentary lifestyle.

Conclusion: Based on the findings of this study, aerobic exercise training is an accepted therapeutic strategy in the management of type 2 diabetes mellitus and obesity.

KEYWORDS: Obesity, type 2 diabetes mellitus (T2DM), Insulin resistance, dyspnoea, aerobic training, percentage body fat, fasting blood glucose

CHAPTER 1

Statement of the Problem

1.1 Introduction

This chapter introduces the research study conducted. It includes the background and rationale of the study, the purpose of the research, aim and objectives as well as hypotheses. The abbreviations used within the context of the study are operationally defined, and an outline of each of the chapters in the research is included.

1.2 Background

Obesity (body mass index $>30 \text{ kg}\cdot\text{m}^{-2}$) has been recognized as a major risk factor for chronic disease, specifically cardiovascular disease (CVD) and diabetes (World Health Organization, 2018). Furthermore, obesity is linked with various health problems which include Type 2 diabetes mellitus (T2DM), hypertension, stroke and heart attacks, sleep disordered-breathing, and respiratory disorders (Jagsi *et al.*, 2017). Salazar (2006) suggests that obesity affects every aspect of an individuals' life, which incorporates, physical health, social and psychological well-being, and economic status. Furthermore, obesity is regarded as a key feature for chronic non-communicable diseases such as hypertension, coronary heart disease, T2DM, dyslipidaemia, and some hormone-dependent cancers (García-Álvarez *et al.*, 2007; Mariano *et al.*, 2015; Pieterse, Schutte & Schutte, 2015; Puoane *et al.*, 2002; Weinstein *et al.*, 2014).

The prevalence of obesity has increased steadily in recent decades, due to changes in diet and increasing physical inactivity (World Health Organisation [WHO], 2018). Interestingly, obesity as an epidemic reveals the rise in an obesogenic atmosphere impacts long-term changes in physical activity levels and energy expenditure (Thow, Jan, Leeder & Swinburn, 2010).

In 2012, the number of deaths emanating from cardiovascular diseases stood at 48%, 21% for cancer, 12% for chronic respiratory diseases, and 3.5% for type 2 diabetes (WHO, 2012).

Consequently, behavioural risk factors such as physical inactivity, unhealthy diet, alcohol abuse, and tobacco use make up 80% of coronary heart disease and cerebrovascular disease (WHO, 2012).

In South Africa, Motala, Sobngwi, Assah and Enoru (2008) reported very high rates of obesity (58–65%) in individuals with diabetes. South Africa is a country in a state of transition with rapid urbanization occurring in all parts of the country. South Africa is transitioning from infectious or rather communicable diseases to non-communicable diseases (NCDs) (Shisana, 2014). NCDs, mainly cardiovascular diseases, cancers, chronic respiratory diseases, obesity and diabetes represent a leading threat to human health and development. Recent reports have suggested high prevalence rates of obesity and overweight in almost all segments of the population (Somers, Hassan, Rusford & Erasmus, 2006). Furthermore, obesity is a major public health problem among black African women in South Africa. Puoane *et al.* (2002) reported that obesity is a notable health threat in adult women. A national survey conducted in 1998 found that over a quarter (27%) of black women were overweight (BMI 25-29.9 kg·m⁻²) and nearly a third (32%) were obese (BMI 30 or more) (Puoane *et al.*, 2002). Abdominal obesity was also apparent in 35 percent (WHR >0.85 cm).

Somers *et al.* (2006), conducted research to determine the prevalence of overweight and obesity amongst 10-16-year-old learners in an urban area of Cape Town, South Africa. The findings of their study revealed that 15.7% of the learners were overweight and 6.2% were obese. Their findings also revealed that overweight was significantly higher in African (21.8%) than Coloured learners (13.7%), however obesity rates were similar between the two groups (5.8% vs. 6%). African females had significantly higher rates ($p < 0.05$) of overweight (30.8%) than Coloured females (17.6%) (Erasmus *et al.*, 2006).

Obesity is characterised by alterations in metabolic function that results from an increase in total body fat mass as well as accumulation of visceral adipose tissue (Murray *et al.*, 2016; Zatu, van Rooyen, Du Toit, Greeff & Schutte, 2015). Salazar (2006) stressed that excessive body weight is directly allied with metabolic syndrome, T2DM, coronary artery disease and stroke, gallbladder disease, sleep apnea, respiratory problems, osteoarthritis, and cancers of the breast, prostate and colon. Metabolic changes are strongly associated with the expansion of important comorbid diseases like T2DM, hypertension and dyslipidaemia (Mariano *et al.*, 2015; Pieterse *et al.*, 2015). Furthermore, Hu, Lindstrom, Valle and Eriksson (2004) also reported that obesity and weight gain are leading contributing factors linked with T2DM.

Type 2 diabetes is a result of insulin resistance, in which the cells of the body lose their ability to recognise the hormone insulin and, as a result, do not take up the glucose in the blood (American Diabetes Association [ADA], 2010). Genetic and environmental factors are strongly implicated in the development of T2DM. The exact genetic defects are complex and not clearly defined, however, it is noted that risk increases with age, obesity, and physical inactivity (ADA, 2010). Meisinger *et al.* (2003) suggests that women with diabetes have a 3 to 6-fold compared to 2 to 4-fold increased risk of myocardial infarction.

Insulin sensitivity and glycaemic control can be enhanced by proper diet and exercise, which reduces the constant need for oral medications and insulin (Hu *et al.*, 2004; Nelson, Reiber & Boyko, 2002). These authors stress the need to increase fruits and vegetables, whilst simultaneously reducing the consumptions of saturated fats. Notably, Cai and Zou (2016) reported that obese and overweight people, often rely on anti-obesity drugs, diet and exercises as leading strategies for weight loss. They further add that diet and exercise interventions are gaining popularity for weight loss in obese and overweight people.

1.3 Statement of the problem

Numerous researchers have reported that regular physical activity (PA) can prevent or delay diabetes and its complications (Balducci, Iancobellis & Parisi, 2006; Cohen *et al.*, 2008; Ghosh *et al.*, 2009). Yet most people with T2DM are not active (Morrato, Hill, Wyatt, Ghushchyan & Sullivan, 2006). Diet and PA are central to the management and prevention of T2DM because they help treat the associated glucose, lipid and blood pressure, as well as aid in weight loss and maintenance. When medications are used to control T2DM, they should augment lifestyle improvements, not replace them.

Additional strategies are needed to increase the adoption and maintenance of PA. One of the most consistent predictors of increased levels of PA has been higher levels of self-efficacy (Aljasem, Peyrot, Wissow & Rubin, 2001; Delhanty, Conroy, David & Nathan, 2006; Dutton, Provost, Sorenson, Allen & Smith, 2009) which suggests confidence in the ability to exercise (McAuley & Blissmer, 2000). Social support has also been associated with greater levels of PA (Gleeson-Kreig, 2008; Mier, Medina & Ory, 2007; Penn, Moffatt & White, 2008) supporting the role of social networks in the control of obesity (Christakis & Fowler, 2007). Unfortunately, those same social dynamics may be exploited to increase the effects of interventions beyond the targeted individual (Bahr, Browning, Wyatt & Hill, 2009; Gorin, Wing & Fava, 2008) and potentially can help spread PA behaviour.

Advice delivered by health care professionals may be a meaningful source of support and an effective source of delivery (Armit *et al.*, 2009; Kirk, Mutrie, MacIntyre & Fisher, 2004). On average, advice or referral related to exercise occurred at 18% of doctor's office visits among diabetic patients (Peek, Tang, Alexander & Chin, 2008) and 73% of patients reported receiving, at some point, advice to exercise more (Morrato *et al.*, 2006). Most adults with T2DM, or those who are at risk of developing it, do not engage in regular PA with their rate of participation being significantly lower than national norms (Morrato *et al.*, 2006).

The concern over obesity is global and, as a curable condition, methods and techniques need to be put in place to ensure there is a significant reduction of obesity in South Africa. Therefore, in an effort to reduce obesity in the workplace, this study focused on the effects of supervised aerobic exercise in obese, pre-diabetic and diabetic female university employees in the Western Cape.

1.4 Significance of the study

This study adds to the current body of knowledge on “exercise is medicine” for overweight or obese people with T2DM. It provides information for South African stakeholders and health care practitioners to implement exercise programmes for this population, and to help to get the country moving through PA. Smith, Neidig, Nickel, Mitchell, Para and Fass (2001) reported that supervised aerobic exercise, when executed and implemented correctly, can safely decrease fatigue levels, weight, BMI, subcutaneous and abdominal girth to their participants, however no evidence from the results suggest improvements on dyspnoea.

Furthermore, Bernhardt, Stickford, Bhammar and Babb (2016) argue that a 12-week aerobic training programme could reduce dyspnoea in obese women, hence, the need to conduct this study on obese, insulin resistant population with dyspnoea. In contrast, according to Swift, Johannsen, Lavie, Earnest and Church (2014), there is no evidence that a low to moderate PA programme will elicit weight loss. Although, it is expected to achieve overall health benefits through this study, it is hoped that this will highlight the effect of exercise training on reducing the risks of multiple chronic diseases.

1.5 Aim and objectives

The aim of this study was to investigate the effects of a 12-week aerobic exercise training programme on insulin resistant, obese female university employees in the Western Cape.

Specifically, the objective of this study was to determine the effect of a 12-week aerobic exercise training programme on percentage body fat levels, fasting blood glucose levels and dyspnoea rating during exertion.

1.6 Hypotheses

It was hypothesised that:

1. Percent body fat would decrease after the 12-week aerobic programme.
2. Fasting blood glucose levels would go down after the 12-week aerobic programme.
3. Dyspnoea during exertion would improve after the 12-week aerobic programme.

1.7 Chapter outlines

Chapter 1. provides the introduction of the study, the background of the topic at hand, and the definition of all the abbreviations that were used.

Chapter 2. consists of a review of literature and provides the theoretical frame work of the study. This chapter includes information and literature on the effects of exercise on percentage body fat, obesity, type 2 diabetes mellitus and dyspnoea.

Chapter 3. provides the outline of aims, hypothesis, Objectives, Significance of the study as well as inclusion and exclusion criteria of the participants. The equipment used is also discussed in this chapter, as well as data collection procedures and ethical consideration.

Chapter 4. provides an overview of the results in accordance with the objectives and hypotheses of the study. Data were organised, analysed and interpreted so that conclusions could be drawn regarding whether the 12-week exercise programme yielded significant results or not.

Chapter 5. provides a critical discussion of the findings in relation to current literature.

Chapter 6. provides the summary, conclusions, and recommendations for future research.



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CHAPTER 2

Review of Literature

2.1 Introduction

This chapter provides information regarding the literature reviewed of the prevalence of obesity, Type 2 diabetes, body fat percentage and dyspnoea and the theoretical framework for the study. Also, an attempt to describe the link between obesity and T2DM the risk factors for diabetes and obesity are described.

2.2 Overweight and obesity

Obesity has become a global health problem, affecting billions of people worldwide (Armstrong, Lambert, Sharwood & Lambert, 2006). Overweight and obesity are important risk factors for cardiovascular diseases, type 2 diabetes, certain type of cancer, and musculoskeletal disorders and are hence of great public health concern (WHO, 2011). Globally, some 2.8 million people die annually as a result of being overweight and obese, and about 35.8 million disability-adjusted life years are caused by overweight and obesity. In Africa, some 27% of adults aged 20 years and over are overweight, and 8% are obese (WHO, 2011)

From a public health perspective obesity is a great concern as it is a risk factor for several chronic and life-threatening health conditions (Manson, Stamter, Hennekens & Willett, 1987). Assessment and classification of these conditions are dependent on specific body mass index (BMI in $\text{kg}\cdot\text{m}^{-2}$). Obesity is defined as abnormal or excessive fat accumulation that negatively affects health (WHO, 2000). Obesity is defined as abnormal or excessive fat accumulation that negatively affects health (WHO, 2000) and has been classified as a BMI greater than or equal to $30\text{kg}\cdot\text{m}^{-2}$ or a waist girth greater than 102cm for men and greater than 88cm for women (Bouchard, 2000).

In Germany, the population from ages ≥ 18 years, 36.3 % are classified as overweight, and 12.9% are obese (Lehnert, Streltchenia, Konnopka, Riedel-Heller, & König, 2015). Furthermore, in America, a high percentage of 62% on males and 26% on females are classified as overweight and obesity respectively (Bastien, Poirier, Lemieux & Després, 2014). Bastien *et al.* (2014) further reported that the prevalence of obesity in Canada was lower than the United States, with 27% and 25% of Canadian men and women being obese or overweight respectively. However, in South Africa, a higher percentage of women are obese, notably in black woman (South Africa Demographic and Health Survey, 1998; Foster, Wadden & Vogt, 1997).

The South African Demographic and Health Survey (SADHS) of 1998 reported on a sample of 6,143 women, and results showed that 25.9% were classified as overweight and 31.2% as obese (Voster *et al.*, 1997). Research shows a progressive increase in the prevalence of obesity in South Africa, particularly among women, with an increasing prevalence being observed in female children (Averett, Stacey & Wang, 2014; Fayne, Crush & McLachlan, 2014).

There is a perspective that obesity does not affect South African communities (Mvo, Dick & Steyn, 1999). Evidently, women know and they are aware that they are obese but they have no intention of losing weight (Kruger, Van Aardt, Walker & Bosman, 1994; South Africa Demographic & Health Survey, 1998). A stereotype exists in South Africa that when an individual gains weight society perceives them as rich, happy and living a successful lifestyle (Chesler, 1961; Mvo *et al.*, 1999). However, it is a well-known fact that overweight and obesity are leading causes of co-morbidities which can lead to further morbidity and mortality (Guh *et al.*, 2009).

It can be perceived that the stereotype is likely to influence programmes that would seek to promote health and well-being. A person's attitude will influence his or her behaviour and is a

link between knowledge and practice (Searle & Ready, 1991). Information on people's knowledge and attitude can help health professionals to formulate operative objectives and develop applicable techniques for health education programmes (Foley, Hertzler & Anderson, 1979).

2.3 Factors that contribute to the prevalence of obesity

2.3.1 Urbanisation

Urbanisation is accompanied by the adoption of a Westernised lifestyle, however in South Africa, many cultural beliefs around lifestyle behaviours and body image do not change when people move to the cities and urban areas (Fallahzadeh, Ostovarfar & Lotfi, 2019). This means that individuals moving from the rural areas to towns, cities and urban areas still maintain the belief that being obese shows that one is rich, successful and happy. This is a traditional stereotype that can be detrimental to one's health and well-being, that needs attention. The later prompts the individuals to continue eating fatty/unhealthy foods and being physically inactive just so they can gain weight (Fallahzadeh *et al.*, 2019).

Urbanisation is happening rapidly, which encompasses a lifestyle of unhealthy eating and physical inactivity, resulting in increased rates of chronic diseases (Steyn, Katzenellenbogen, Lombard & Bourne, 1996; Vorster, Venter, Wissing & Margetts, 2005) gallbladder disease, coronary heart disease, hypertension, osteoarthritis (Pi-Sunyer, 2009) sleep apnoea, and respiratory disorders (Bernhardt *et al.*, 2016). According to Burkitt (1982), the traditional African diet is healthier as it contains low levels of fat and high levels of carbohydrates and fibre (Burkitt, 1982; Segal & Walker, 1986).

Individuals living in urban areas, and eating a Western diet are exposed to medical conditions such as obesity and diabetes. Obesity is an environmental issue. Societies that are transitioning to westernized lifestyles are experiencing substantial increases in its prevalence (Poston &

Foreyt, 1999). The primary environmental determinants of obesity are high calorie intake and low levels of activity. This is the reason why most people who stay in the villages and farms eat healthier than people who stay in urban areas (Burkitt, 1982; Segal & Walker, 1986).

Mollentze *et al.* (1995) state that, even though obesity among women has been noted in rural areas, more obese female individuals were found in urban areas. This was subsequently supported by Voster *et al.* (2000) and Wolmarans *et al.* (2000). A lot of people are moving to the cities and towns all over the country, this means that obesity will be increasing as many people will be adopting the Western way of living including diet/food (Mollentze *et al.*, 1995; Voster *et al.*, 2000). These adoptions expose individuals to obesity when they are presented with fatty foods or they are earning a salary that enables them to have access to all sorts of unhealthy foods (Fallahzadeh *et al.*, 2019).

2.3.2 Loss of Sleep

There is a direct relationship between sleep loss obesity and glucose intolerance. Depriving normal people of sleep has been shown to result in insulin responses to hyperglycaemia characteristic of insulin resistance and a pre-diabetic metabolic state (Van Helder, Symons & Radomski., 2003). Spiegel *et al.* (1999) found that healthy men whose sleep was restricted to 4 hours per night for 6 nights experienced a 30% reduction in insulin response to glucose. Furthermore, inadequate sleep and poor-quality sleep have been associated with obesity in children (Locard *et al.*, 1992; Sekine *et al.*, 2002; Von Kries *et al.*, 2002), adolescents (Gupta, Muella, Chan & Meininger, 2002) and adults (Kripke, 2002; Vioque, Torres & Quiles, 2002). Advanced age has associated with changes in sleep characteristics and structure, with increased difficulties in sleep initiation and maintenance (Webb, 1989).

Along with the physical changes that occur as one gets older, changes to sleep patterns are a part of the normal ageing process (Oyahon, 2004; Redline *et al.*, 2004; Moraes *et al.*, 2014).

As people age, they tend to have a harder time falling asleep and more trouble staying asleep than when they were younger. Sleep occurs in multiple stages including dreamless periods of light and deep sleep, and occasional periods of active dreaming (REM sleep) (David, Neubauer & Johns, 1999). The sleep cycle is repeated several times during the night and although total sleep time tends to remain constant, older people spend more time in the lighter stages of sleep than in deep sleep (Oyahon, 2004; Redline *et al.*, 2004, Moraes *et al.*, 2014).

Many older adults, though certainly not all, also report being less satisfied with sleep and more tired during the day (Foley *et al.*, 1995). This results in the elderly wanting to rest during the day instead of having the energy to exercise (David *et al.*, 1999). Obese subjects would be less likely to survive into their later years, since they are at an increased risk for serious and potentially fatal conditions such as diabetes mellitus, hypertension, dyslipidemia, coronary artery disease, and some cancers (Solomon & Manson, 1997). Secondly, advanced age is associated with changes in sleep characteristics and structure, with increased difficulties in sleep initiation and maintenance (Webb, 1989).

2.3.3 Sedentary Lifestyle

Obesity and the number of homes with television sets and remotes and an extended amount of time spent watching television (Nielson Report Media, 1998). High screen time is associated with unhealthy eating behaviour, low levels of physical activity, delayed bed time which results in reduction of the duration of sleep (Hale & Guan, 2015). Insufficient sleep could cause exhaustion during the day and lack of interest in participating in physical activity due to exhaustion (Basta *et al.*, 2008; Hastings *et al.*, 2006; Roehrs, Turner & Roth, 2000).

Compared to other sedentary activities such as sewing, reading or playing board games, watching television results in lower metabolic rate (Ainsworth *et al.*, 1993). Constant food advertisements on the television provoke hunger, which leads to increased eating and unhealthy

eating patterns (Dietz & Gortmaker, 1985; Hu *et al.*, 2001; Lank, Vickery, Cotugna & Shade, 1992).

2.3.4 Unhealthy Diet

An unhealthy diet could also lead to overweight and obesity and contribute directly to different causes of illnesses and eventually, death. According to Ng *et al.* (2014), globally, one in every three adults are either overweight or obese. Food structures or organizations have the potential to nourish human health and to sustain the environment; however, they are currently threatening both (Willet *et al.*, 2019). Unhealthy diets pose a greater risk to morbidity and mortality than does unsafe sex, and alcohol, drug, and tobacco use combined. Many of the environmental systems and processes are pushed beyond safe boundaries due to short supply of proper nourishment (Willet *et al.*, 2019).

The combination of a sedentary lifestyle and unhealthy diet is responsible for 10% of disability-adjusted life years lost globally (Lim *et al.*, 2012). Studies have shown that obesity and unhealthy diets are more common in socioeconomically disadvantaged groups (Darmon & Drewnowski, 2008; Jones-Smith, Gordon-Larsen, Saddiqi & Popkin, 2012).

Evidence suggests that urbanization on the African continent began around 1975 with a massive growth of industries, especially emerging manufacturing industries located in the urban areas (Rakodi, 1997; The United Nations Industrial Development Organization, 2013). This was then followed by a massive growth of fast food franchises, especially those that sell energy-dense food (high in added sugar and high in fat). The increase in fast food franchise is one of the factors related to the increase in obesity globally (Popkin, 2006; Popkin, Adair & Nq, 2012).

Presently, a significant contributor to poor health and an increase in a person's risk of being obese or having non-communicable disease is excessive ingestion of meat (EAT-Lancet Commission., 2019). Unhealthy diets are now the leading risk factor for non-communicable diseases such as diabetes, heart disease and cancers. Currently, food contributes to both poor health outcomes and extreme degradation of the environment. Increasing food demand and the adoption of unhealthy diets including persistent hunger, generalized overconsumption as well as overconsumption of unhealthy foods lead to incredible strains on public health and has grave outcomes on natural resources and the environment.

The EAT-Lancet Commission (2019) suggests that the planetary health diet will optimize human health by setting out scientific targets for healthy diets. According to the EAT-Lancet Commission (2019), adopting the guidelines can result in a reduction in undernutrition, overnutrition and diet-related non-communicable diseases, which are continuously on the rise globally. Ingestion or consumption of the planetary health diet all over the world, can reduce approximately 11 million premature adult deaths yearly, effectively contributing to a 19-23% overall reduction in premature deaths per year.

2.3.5 Maternal and early life factors

Lack of proper nutrition during pregnancy results in foetal programming which causes adaptations in the genes (Ásbjörnsdóttir *et al.*, 2019). The authors' further stress that pregnancies can be complicated by diabetes, which increases the risk of adverse perinatal results that involve infants being born large for their gestational age. The increase in number the of babies being born with a low birth weight of less than 2.5 kg is usually the result of poor nutrition during pregnancy. In South Africa this rate is 15%, which is higher than the overall increase of 13% in Sub-Saharan Africa (Puoane *et al.*, 2002).

The rise in numbers of obesity, especially in children, is a serious concern, due to the negative consequences thereof (Bastien *et al.*, 2014). Five low-to middle-income countries, including SA, have shown that the size of the baby at birth is linked to the main features of metabolic syndrome in adulthood, including obesity (Young *et al.*, 2018). Concurrently, low birth weight has been aligned with increased levels of adult abdominal adiposity, while a high birth weight was associated with overall adult adiposity (WHO, 2018).

The World Health Organization (2018) encourages the idea of breastfeeding new-born babies until they are about six months old. Additionally, under-nutrition during the first six-months of life increases the risk of poor growth due to malnutrition. Ásbjörnsdóttir *et al.* (2019) showed that poor growth due to poor nutrition in children under the age of nine years resulted in a 1.8-fold increased risk of obesity. Moreover, other evidence suggests that individuals who experienced poor nutrition when they were still young had a higher chance of being overweight/obese as adults (Rasmussen *et al.*, 2019). On the other hand, mothers who gained extra weight during pregnancy have been shown to give birth to babies with high birth weight (Rasmussen *et al.*, 2019). This is of concern in SA, given the high prevalence of obesity in adolescents and adult women. The concern is worsened by the disparities in the healthcare inequalities, associated perceptions of the healthcare system, and the periodic lack of adequate resources disturbs required visits for antenatal sessions in clinics (Jeanette & Bonnie, 2010). The impact of obesity and overweight, has an unending impact on society and the country at large, it is therefore, important to report on the risks associated.

2.3.6 Genetics

There is substantial evidence for the heritability of obesity, and research in both rare and common forms of obesity has identified genes with significant roles in its aetiology (Walley, Blakemore & Froguel, 2006). Obesity is commonly classified into subgroups depending on suspected etiology: monogenic obesity (extremely severe obesity in the absence of

developmental delays) (Farooqi & O’Rahilly, 2008), syndromic obesity (clinically obese subjects additionally distinguished by mental retardation, dysmorphic features, and organ-specific developmental abnormalities) (Cormier-Daire *et al.*, 2003), and polygenic or common obesity, which affects the general population (but may have associated health risks, such as the increased risk of CVD) (Thorleifsson *et al.*, 2009).

The first single gene defect causing monogenic obesity was described in 1997, and to date, there are about 20 single-gene disruptions that result in an autosomal form of obesity (O’Rahilly, 2009). Interestingly, all these mutations position the leptin/melanocortin pathway in the central nervous system (CNS) as critical in the regulation of whole-body energy homeostasis and obesity in these cases appears to be the result of increased appetite and diminished satiety (Coll *et al.*, 2004). Syndromic obesity arises from discrete genetic defects or chromosomal abnormalities at several genes, and can be autosomal or X-linked (Cormier-Daire *et al.*, 2003). One of the most well-known forms of syndromic obesity is Prader-Willi syndrome (PWS), which is caused by a chromosomal abnormality of an imprinted region on chromosome 15q11-q12. PWS is characterized by early-onset obesity resulting from hyperphagia caused by CNS dysfunction (Shapira *et al.*, 2005).

2.4 Common health risks associated with obesity

Some of the health complications are caused by overweight and obesity independently or in combination with other diseases, overweight and obesity contribute so much to ill-health (Ezzati *et al.*, 2002; Willett, Dietz & Colditz, 1999). According to Meigs *et al.* (2006) intra-abdominal visceral deposition of adipose tissue, which symbolizes upper body central obesity is a leading factor of hypertension, elevated plasma insulin concentrations and insulin resistance, hyperglycaemia and hyperlipidaemia.

Guh *et al.* (2009) stated that abdominal obesity which is distinctive by waist circumference (WC) measurement as opposed to the traditional obesity defined by BMI measurement, is an interpretative cause of cardiovascular diseases and T2DM. Cardiovascular diseases are imminent consequences of overweight and obesity, in which hypertension and related conditions develop. The occurrence of conditions such as hypertension, elevated plasma insulin concentrations and insulin resistance, hyperglycemia and hyperlipidaemia are understood as metabolic syndrome (Meigs, 2006). Metabolic syndrome reflects a merging fundamental pathophysiology that needs a holistic approach for its management (Meigs, 2006).

2.5 The link between obesity and dyspnoea

Dyspnoea is an aversive and threatening subjective experience of breathing discomfort that differs from person to person (Sucec *et al.*, 2018) and is a common symptom of respiratory, cardiac, neuromuscular, and psychological disorders (Baille *et al.*, 2019). Dyspnoea is a main symptom in various diseases including asthma, chronic obstructive pulmonary disease (COPD), cardiovascular diseases, advanced stages of cancer and psychological disorders such as anxiety (Sucec *et al.*, 2018).

Bernhardt *et al.* (2016) reported that dyspnoea is a well-known and common symptom of obesity in patients and further stress that obesity is limited to low intensity exercises due to the associated shortage of breath with physical activity. The physiological state of breathing is affected by obesity (Luce, 1980). Furthermore, the author states that the pulmonary system is affected by obesity whereby the respiratory systems are compromised due to increased weight and the thoracic cage and abdomen. Dyspnoea is the main symptom in various diseases including asthma, chronic obstructive pulmonary disease (COPD), cardiovascular diseases, advanced stages of cancer and psychological disorders such as anxiety (Sucec *et al.*, 2018)

An excessive increase of fat on the chest wall and abdomen leads to respiratory system defects during respiration and it also decreases lung volume. Luce (1980) stated that one of the most vital irregularities that accompanies obesity is the decrease in respiratory function. Respiratory function is limited to two thirds of the normal significance in obese individuals. This is because of the decrease in lung compliance due to excess fat around the chest wall and the abdominal area as well as the diaphragm.

Complete respiratory compliance is noticeably reduced by recumbence in obese individuals compared to non-obese individuals (Yap, Watson, Gilbey & Pride., 1995). This reduction is due to the declined compliance of the chest wall, although it can also be due to an increase in respiratory resistance (Yap *et al.*, 1995). Although substantial energy may be spent in overcoming the decrease in the chest wall compliance, this accounts for only one-third of the increased work of breathing (Naimark & Chermiack, 1960).

Obese individuals have a low breathing pattern which is evident in patients with neuromuscular disorders and deformities of the chest wall (Riaz, Wolden, Gelblum & Eric, 2016). Obese individual's respiratory muscles work constantly against a less compliant chest wall and higher airway resistance (Sheel, Foster & Romer, 2011). The main respiratory complications of obesity with or without hypoventilation include delicate demand for ventilation, raised work of breathing, respiratory muscle inefficiency and reduced respiratory compliance (Kaeuffer, Ledoux & Herbrecht, 2019).

It is very common for obese individuals to experience shortness of breath during exercise (Gibson, 2000). In another study, Bulpitt, Palmer, Battersby and Fletcher (1998) reported that one-third of obese patients with T2DM reported irregular breathing that became more prevalent with cumulative BMI. The cause of dyspnoea in obese people was due to a variety of factors related to the abnormal physiological effects (Bulpitt *et al.*, 1998). In obese subjects, the

increase in breathing rate is an attempt to overcome decreases in pulmonary compliance and increases in resistance (Pankow *et al.*, 1998; Sahebjami, 1998).

Reduction in airflow at low lung volumes (partly due to respiratory muscle weakness in obese individuals) stimulates chemoreceptor activity, that respond to increasing hydrogen ions and carbon dioxide CO₂ production, which increases breathing rate, resulting in a sense of breathlessness (Pankow *et al.*, 1998; Sahebjami, 1998).

2.6 Diabetes

Diabetes is a growing cause of premature mortality and morbidity worldwide. Globally, diabetes has been estimated to cause 3.96 million excess deaths and 6.8% of all deaths in 2010 (Roglic & Unwin, 2010). McAdam-Marx, Bouchard, Aagren, Conner and Brixner (2011) suggested that approximately 24 million people in the United States have diabetes, and 95% have T2DM. In diabetes, plasma proteins are exposed to high absorptions of glucose for continued periods of time and this leads to protein glycation (Zemlin, Barkhuizen, Kengne, Erasmus & Matsha, 2019).

Notably, T2DM develops when insulin secretion is inadequate to meet the increased insulin measures caused by tissue insulin resistance (Barrett, 2017). T2DM is often accompanied by obesity, dyslipidaemia, hypertension, albuminuria, ovarian hyperandrogenism, non-alcoholic fatty liver disease and obstructive sleep apnoea (Barrett, 2017). Furthermore, blindness, amputation, kidney and nerve disease are also associated with T2DM (Colberg *et al.*, 2010). Bener *et al.* (2017) warns that additional risk factors for T2DM for vulnerable groups are socio-economic status, gender, lifestyle-habits, obesity, family history and the elderly.

World-wide statistics of people with diabetes are anticipated to increase by 48% by 2045 from 425 million in 2017 (Zemlin *et al.*, 2019). Lukács, Kiss-Tóth, Csordás, Sasvári, and Barkai (2018) estimate that by the year 2040 there will be over 642 million people all over the world

living with diabetes. These estimates are concerning, especially for future generations. Prediabetes and diabetes are public health concerns, and the continuation of such behaviour in our society will lead to serious public health and economic challenges (Lukács *et al.*, 2018). Prediabetes, is a condition that includes blood glucose levels that are above the normal range, but less than 7 mmol·L⁻¹ and is a run-up to the development of T2DM (Colberg *et al.*, 2010; Deschênes, Burns, Graham & Schmitz, 2016). Interestingly, the authors note that not everyone who is confronted by prediabetes will develop T2DM, however, there is a 70% possibility.

Prediabetes is an intermediate dysregulation of glucose metabolism that is formed between the normal metabolic state and the development of T2DM (Roncero-Ramos *et al.*, 2019). However, taking note and addressing the predictors of the progression from prediabetes to T2DM can have a significant challenge for reducing the incidence of T2DM (Roncero-Ramos *et al.*, 2019). The likelihood of glucose levels in pre-diabetic individuals deteriorating to normal glucose levels for people living with prediabetes, an increase in healthy lifestyles changes is required (Roncero-Ramos *et al.*, 2019).

2.7 The link between obesity and diabetes

Overweight and obesity are some of the leading risk factors for the development of type I and II diabetes mellitus (Fox *et al.*, 2006; Wild *et al.*, 2004). Fox *et al* (2006) and Wild *et al* (2004) suggest that a clear relationship between central obesity and type II diabetes exists. Obesity is characterized by raised fasting plasma insulin and an insufficient insulin response to an oral load (Mbanya, Motala, Sobngwi, Assah & Enoru, 2010). In obese individuals, adipose tissue releases increased amounts of non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines and other factors that are involved in the development of insulin resistance (Wild *et al.*, 2004). An inverse relationship exists between adiponectin, adiposity and insulin sensitivity, with T2DM characterized by low levels of adiponectin (Bener *et al.*, 2017). According to

Zanuso, Jimenez, Pugliese, Corigliano, and Balducci (2010), adiponectin is similar to tumour necrosis factor-alpha (TNF-a), which paradoxically seemingly increase abdominal adipose tissue.

Escalations in pro-inflammatory cytokines [interleukin 6, resistin, TNF- and CRP] duplicate the overproduction of adipose tissue mass (Frohlich *et al.*, 2000; Timpson, et al 2005). All of these factors contribute to the unusual release of free fatty acids (FFAs) from abdominal adipocytes in to the portal system. These FFAs have a toxic effect on insulin uptake by the liver and increase the hepatic gluconeogenesis and hepatic glucose relief detected in central obesity (Frohlich *et al.*, 2000; Timpson, 2005). Insulin insensitivity is not limited to the adipocytes because the process is emphasized by skeletal muscle insulin resistance (Frohlich *et al.*, 2000; Timpson, 2005).

Studies have shown that the prevalence of T2DM increases with body-mass index (Aspray *et al.*, 2000; Motala *et al.*, 2008), total body obesity (Ahren & Corrigan, 1984), waist-to-hip ratio (Ahren & Corrigan, 1984; Balde *et al.*, 2007; Fisch *et al.*, 1982; Levitt, Katzenellenbogen, Bradshaw, Hoffman, & Bonnici., 1993; Motala *et al.*, 2008) and waist circumference (Balde *et al.*, 2007; Motala *et al.*, 2008), and abdominal obesity (Mollentze *et al.*, 1995; Motala *et al.*, 2008), therefore, a link exists between obesity and T2DM. Additionally, Puone *et al.* (2002) stress that women have the highest prevalence of T2DM compared with their male counterparts, with 56.6% of female participants being obese.

Several lifestyle factors affect the incidence of Type 2 diabetes. Obesity and weight gain dramatically increase the risk and physical inactivity further elevates the risk (Heath *et al.*, 2012). In addition, a low-fibre, high glycaemic index diethas been associated with an increased risk of diabetes and specific dietary fatty acids may differentially affect insulin resistance and the risk of diabetes (Hu, Van Dam, Liu, 2001). Frequent exposure to food and advertising of

food products, promotes food and calories intake, which causes unhealthy eating patterns (Hu, Li, Colditz, Willett, & Manson, 2003). Interestingly, Binkley, Eales, and Jekanowski (2000) assert that weight gain is impacted by a balance between calorie intakes versus energy expenditure.

There is a need for awareness to increase knowledge of the importance of good nutrition and its correlation to specific health problems, to achieve nutritional monitoring in the general population (Binkley *et al.*, 2000). The author reiterates that a clear health concern is nutrition and its immediate impact on obesity. However, despite the reports and information sharing across all platforms of communication, and the availability of low calories and low or no-fat foods, people in America continue gaining weight (Binkley *et al.*, 2000).

2.8 Risk factors of diabetes (prediabetes and type 2 diabetes)

2.8.1 Diet

When caloric intake is not matched by energy expenditure, this leads to obesity (Goran.,2000). According to Chen *et al.* (2020) drinks that contain a lot of sugar contribute a lot to long-term weight-gain, T2DM, cardiovascular disease and early death. Sweet drinks cause weight gain because they contain a lot of sugar and they do not completely take the feeling of hunger away, which leads to increased energy intake (Nelson *et al.*, 2002). A lot of people choose sweetened drinks like soda and energy drinks over healthy food, especially in very busy workplaces, and this leads to more energy intake throughout the day, which result in obesity and eventually T2DM (Thow *et al.*, 2010). Occupational demands versus food intake is not the only concern reported in the literature, this varies to all people, in different life stages respectfully.

Stress-related eating is reported in America to constitute 43% of people using food to cope with stress (Groesz *et al.*, 2012). The prevalence of stress-related eating increases the prevalence of overweight and obesity in the US, whereby 72.3% of adult men and 64.1% of their female

counterparts are overweight or obese (Groesz *et al.*, 2012). Furthermore, lifestyle trends such as fast-food consumption and passive entertainment through TV, are major contributors towards obesity (Jeffery & French, 1998). Stress-related eating is also recognised as emotional eating (Tan & Chow, 2014). It refers to a behavioural tendency of reacting to negative emotions with food consumption, and can lead to an eating disorder such as anorexia or bulimia nervosa, and depression (Tan & Chow, 2014). In combination with a high caloric intake, being physically inactive and living a sedentary lifestyle can have dire consequences.

2.8.2 Physical inactivity

Physical inactivity is perceived as a public health crisis (Lee, Dvorak, Schuett & van Riper, 2017). Physical inactivity is recognised as a significant modifiable health behaviour for chronic diseases (Farrell, Hollingsworth, Propper & Shields, 2014) decreased quality of life, and premature mortality (Prochaska, Buschmann, Jupiter, Mutambudzi & Peek, 2018). The authors expand that being informed of the prevalence of physical inactivity is essential to designing a cost-effective intervention.

Physical inactivity and increased body mass index are established independent risk factors in the development of T2DM (Weinstein *et al.*, 2014). Time spent in sedentary activities, such as TV watching and computer or video-game use, leads to an increase in obesity (Hu *et al.*, 2003), and T2DM development. The terms “Physical Activity” and “Exercise” describe different concepts, but are often used interchangeably. Physical inactivity is an important risk factor for accumulating fat weight (LaMonte & Blair, 2006). Individuals who are obese do not typically engage in physical activity and this leads to decreased muscle strength and fat-free mass (Duvigneaud *et al.*, 2008). The authors further stress that reduced muscle strength leads to a decline in metabolic rate both at rest and during physical activity and may further facilitate the sedentary state.

Sedentary behaviour (watching TV, working at a desk for long hours at a time etc.) is a ubiquitous and significant population-wide influence on cardiometabolic health (Hu *et al.*, 2003). Lower amounts of PA followed by higher amounts of sitting and increased mortality, with an accelerated sitting-related risk becomes essential at PA levels below 35.5 metabolic equivalent of task (MET)-h per week (Andersen, Mota, & Di Pietro, 2016). Higher amounts of sedentary time are associated with increased mortality and morbidity, mostly independent of moderate to vigorous physical activity participation (Hu *et al.*, 2003).

Individual behaviour such as unhealthy diets, physical inactivity, tobacco use, and excessive alcohol consumption are leading modifiable risk factors for Non-Communicable Diseases (Chan *et al.*, 2019; Prochaska *et al.*, 2018). The authors further emphasized that the above factors can be influenced by individuals' lifestyle choices that are often affected by levels of economic development, rapid urbanisation and environmental change.

In people with or at risk for developing T2DM, extended sedentary time is also associated with poorer glycaemic control and clustered metabolic risk (Chan *et al.*, 2019; Hu *et al.*, 2003; Prochaska *et al.*, 2018). Prolonged sitting interrupted by a brief (< 5 min) bouts of standing (Farrell *et al.*, 2014) or light intensity ambulation (Lee *et al.*, 2017) every 2-30 minute improves glycaemic control in sedentary overweight/obese populations and women with impaired glucose regulation. In adults with T2DM, interrupting prolonged sitting with 15 minutes of post-meal walking (Chan *et al.*, 2019) or with 3 minutes of light walking and simple body-weight resistance activities every 30 min (Chan *et al.*, 2019; Farrell *et al.*, 2014; Prochaska *et al.*, 2018) has been shown to improve glycaemic control.

2.9 Physical activity and exercise

2.9.1 Aerobic exercise

Physical Activity is defined in this study as any bodily movement produced by skeletal muscles that results in energy expenditure (Burton, Khan, & Brown, 2012; Colberg *et al.*, 2010). Physical activity can be categorized in to occupational, sports, leisure, conditioning, household chores or other activities. Exercise is a subset of PA that is planned, structured and repetitive and has a final or an intermediate objective (Caspersen, Powell & Christenson, 1985). Physical Activity is a technique that can be used as a coping strategy, which includes stress management, and can yield psychological benefits for anxiety and depression (Edwards, Rhodes, Mann, & Loprinzi, 2018).

Kohl *et al.* (2012) reported that over 31% of the world population does not meet the recommended levels of PA. Being physically inactive has dire consequences which includes but are not limited to being overweight, obese and even cardiovascular disease. PA could be used to manage chronic illnesses and to promote health, well-being and rehabilitation programmes to boost physical endurance (Smith *et al.*, 2001). Significantly, Cai and Zou (2016) suggest that aerobic exercise and not resistance training decrease the intrahepatic lipid content and visceral fat in obese adolescent females.

Professional health care and service providers recommend weight loss as a key strategy for glycaemic control (Franz, Boucher, Rutten-Ramos & VanWormer, 2015). Weight-loss therapies usually comprise of lifestyle interventions, weight-loss medications, and bariatric surgery (Franz *et al.*, 2015). Aerobic exercise is significant in weight loss, in that it helps manage signs and symptoms of other chronic illnesses and it is beneficial for health promotion and rehabilitation programmes to improve physical endurance (Para *et al.*, 2003).

Aerobic exercise, such as brisk walking together with meditation for a minimum of 5 minutes are reported by Edwards *et al.* (2018) to yield health-related benefits, both physical and psychological. Exercise training has been shown to improve exercise performance on the 6-minute walk test and to reduce ventilator efficiency and exertional breathlessness in obese children and adolescents (Chlif, Chaouachi, & Ahmaidi, 2017). Aerobic exercise is therefore important to an individual, especially to obese people and people living with diabetes. Hence, it is important to highlight the benefits of exercise, to raise awareness to all people.

2.9.2 Benefits of physical activity and training for overweight and obese individuals

In this day and age, a lot of people (both educated and uneducated) know that a sedentary lifestyle results obesity (King, Hills, & Blundell, 2008; Lindstrom, Isacson & Merlo, 2003). Being physically active or exercising on a daily basis is one of the wellness cornerstones that are effective when it comes to treating and preventing obesity. Klein *et al.* (2004) highlighted that weight-loss should be a primary therapeutic strategy in all obese individuals who have T2DM or are at risk of developing T2DM. Bernhardt *et al.* (2016) agree that exercise is key to obesity management and the prevention of incident diabetes. They reported that reasonable weight loss using aerobic exercise was also effective in reducing dyspnoea in healthy obese women.

Exercise training contributes to declining ventilator demand and probably decreases the impact of ventilators constraints on exercise capacity and may be a way to increase inspiratory muscle function (Chlif *et al.*, 2017). Exercise has a positive influence on cognition, with increases in selective attention, processing speed and executive function-related tasks (Douris *et al.*, 2018). They reported that a variety of intensities of aerobic exercise provides the most benefit, the optimal effect on cognitive performance being 60-70% of the maximum heart rate, which is defined as moderate intensity. Interestingly, moderate intensity aerobic exercise has been shown to increase arousal levels in the reticular activating system, increasing ascending

stimulation of the cortex, thus the outcome leading to improved cognitive performance (Douris *et al.*, 2018). As a result, PA benefits can be experienced by all people who become active, even after an extended period of physical inactivity (Burton *et al.*, 2012).

Physical activity and/or exercise training should therefore be an essential part of any treatment plan for obese individuals, regardless of their weight loss goals, which is linked with various cardiovascular benefits (Swift *et al.*, 2014). The benefits of PA are diverse and can be achieved by frequent participation according to the required standards. As it is evident from the above regarding the benefits of PA on obese and overweight people, people living with T2DM can also derive benefits that are supportive of their condition, therefore, the following passage reports on benefits of PA on people living with T2DM.

2.9.3 Benefits of physical activity and training for people with type 2 diabetes

People with diabetes should perform aerobic exercises regularly. Aerobic activity bouts should ideally last at least 10 minutes with the goal of 30 minutes/day or more, most days of the week for adults with T2DM (Khan.,2013). Daily exercise or at least not allowing two days to elapse between exercise sessions is recommended to decrease insulin resistance regardless of diabetes type (Dube', Lavoie & Weisnagel, 2013; Jolleyman *et al.*, 2015; Little *et al.*, 2011; Tonoli *et al.*, 2012). Overtime, activities should increase frequency, intensity and duration to at least 150 min/week of moderate-intensity. Adults with diabetes should engage in 2-3 sessions/week of non-consecutive days (American Diabetes Association, 2011; Amatuli, 2020; Colberg *et al.*, 2010; Jolly *et al.*, 2007). Although heavier resistance training with free weights and weight machines may improve glycaemic control and strength more, doing resistance training of any intensity is recommended to improve strength, balance and the ability to engage in activities of daily living throughout the lifespan (Willey & Singh, 2003).

Insulin action in the muscle and liver can be adjusted by acute bouts of exercise and by frequent physical activity (Roberts, Henever & Barnard, 2013). The authors further report that exercise increases muscle glucose uptake up to fivefold through insulin-independent mechanisms. After exercise, glucose uptake is elevated by insulin-independent (>2h) and insulin-dependent (up to 48 h) mechanisms if exercise was prolonged (Magkos *et al.*, 2008), which is associated with muscle glycogen repletion (Wang *et al.*, 2013). Changes in insulin action may last for 24 h following shorter duration of exercise (> 20 min) if the intensity was elevated to near-maximal effort.

The mechanism by which exercise improves insulin sensitivity has been researched by, amongst others Rutter, Da Silver, Xavier and Leclerc, (2003) and Santos *et al.* (2008). Muscle contraction causes the translocation of glucose carrier protein-4 (GLUT-4) to the plasma membrane due to the activation of 5'-adenosine monophosphate-activated protein kinase (Rutter *et al.*, 2003). When the former is triggered it increases cytoplasmic calcium concentration due to membrane depolarization and a higher intracellular ratio of adenosine monophosphate to adenosine triphosphate, which reflects a compromised energy status of the cell (Rutter *et al.*, 2003; Santos *et al.*, 2008).

The enzyme 5'-adenosine monophosphate-activated protein kinase has visible effects on the expression of many glycolytic and lipogenic enzymes in the liver which adds to the valuable effects on fatty acids metabolism (Rutter *et al.*, 2003). C-jun N-terminal kinase and I κ B kinase /I κ B/nuclear factor-K β activation by stimuli such as cytokines, endoplasmic reticulum stress, and fatty acids influences serine phosphorylation of insulin receptor substrate (IRS)-1. The latter leads to reduced phosphorylation of the Akt substrate 160, the main insulin signalling that controls GLUT-4translocation (Mathur & Pedersen, 2008).

Furthermore, exercise can decrease the inflammatory status by the decline of high-sensitivity C-reactive protein (hs-CRP) and tumor necrosis factor (TNF- α) and the enhancement of adiponectin (Mathur & Pedersen, 2008; Monzillo *et al.*, 2003). A single bout of exercise has been shown to increase plasma membrane GLUT-4 protein by nearly 4-fold which may be caused by an increase in blood flow during exercise (Mathur & Pedersen, 2008; Monzillo *et al.*, 2003).

Lucotti *et al.* (2011) support the notion that aerobic training is a cornerstone for people with T2DM, furthermore, aerobic training improves mobilization of visceral adipose tissue, that reduce insulin resistance. During muscle contraction *in situ* the amount of plasma membrane GLUT-4 rises a similar degree as the lack of insulin (Wojtaszewski, Nielsen & Richter, 2002). These results suggest that GLUT-4 translocation is independent of the action of insulin (Ryder, Chibalin & Zierath, 2001).

The reduction in blood glucose reductions relates to the intensity of the exercise, pre-exercise control, and state of physical training (Boulé, Haddad, Kenny, Wells, & Sigal, 2001; Boulé, *et al.* 2005; Colberg *et al.*, 2010). Although previous physical activity of any intensity usually promotes these effects by improving the uptake of blood glucose for glycogen synthesis (Christ-Roberts *et al.*, 2003). Stimulating fat oxidation and glycogen storage in muscle (Boon *et al.*, 2007; Goodpaster, Katsiaras & Kelley, 2003), intense physical activity has been shown to acutely improve insulin action for longer periods (Braun, Zimmerman & Kretchmer, 1995; Houmard *et al.*, 2004; Evans *et al.*, 2005; Muller *et al.*, 2008). Furthermore, Bellia *et al.* (2017) noted that aerobic exercise is significant to promote glycaemic control and risk for cardiovascular diseases in T2DM patients. Aerobic exercises are linked to improvements in glycaemic control (Boulé *et al.*, 2001; Snowling & Hopkins, 2006; Thomas, Elliott & Naughton, 2006).

2.10 Other ways to manage obesity and type 2 diabetes.

2.10.1 Combination of aerobic and resistance exercise.

Aerobic and resistance exercise has been shown to reduce glycated haemoglobin A1C levels by approximately 0.6% (Boulé *et al.*, 2001; Thomas *et al.*, 2006). Further, aerobic exercise is known to encourage frequency, intensity, type and time (FITT) exercise principle to mediate health of patients with T2DM (Borghouts & Keizer, 2000). The American College of Sports Medicine (ACSM) also suggested the use of progressive resistance training as part of a seasoned exercise for T2DM people.

Diabetes is an independent risk factor for an accelerated decline in muscle mass therefore, low muscular strength (Nishitani *et al.*, 2011) and functional status (Anton, Karabetian, Naugle & Buford, 2013). The health benefits of resistance exercise training for all adults include improvements in muscle mass, body composition, strength, physical function, mental health, bone mineral density, insulin sensitivity, blood pressure, lipid profiles and cardiovascular health (Garber *et al.*, 2011). Resistance exercise training benefits for individuals with T2DM include improvements in glycaemic control, insulin resistance, fat mass, blood pressure, strength and lean body mass (Gordon, Benson, Bird & Fraser, 2009).

Aerobic training increases mitochondrial density, insulin sensitivity, oxidative enzymes, compliance and reactivity of blood vessels, lung function, immune function and cardiac output (Garber *et al.*, 2011). Moderate to high volumes of aerobic activity are associated with substantially lower cardiovascular and overall mortality risk in both Type 1 and T2DM (Sluik *et al.*, 2012). In Type 1 diabetes, aerobic training increases cardiorespiratory fitness, increases insulin resistance and improves lipid levels and endothelial function (Chimen *et al.*, 2012). In T2DM, regular exercise has been shown to reduce HbA1c, triglycerides, blood pressure and insulin resistance (Snowling & Hopkins, 2006).

Both aerobic and resistance training promote adaptations in skeletal muscle, adipose tissue, and liver associated with enhanced insulin action, even without weight-loss (Bacchi *et al.*, 2013; Hallsworth *et al.*, 2011). Regular aerobic exercise increases muscle insulin sensitivity which reduces the blood glucose levels associated with pre-diabetes and T2DM in proportion to exercise volume (Dube *et al.*, 2012; Kirwan *et al.*, 2013). Even low-volume training improves insulin action in previously sedentary adults (Dube *et al.*, 2012).

2.10.2 Flexibility and balance exercises

Flexibility and balance exercises are also important for older adults with diabetes. Limited joint mobility is frequently present in diabetics, resulting in part from the formation of advanced glycation end products, which accumulate during normal aging and are accelerated by hyperglycemia (Abate, Schiavone, Pelotti & Salini, 2010). Stretching exercises increase range of motion around joints and flexibility (Herriott *et al.*, 2004) but they do not affect glycaemic control.

The benefits of alternative training such as yoga, may promote improvement in glycaemic control, lipid levels, and body composition in adults with T2DM (Innes & Self, 2016). Tai Chi training may also improve glycaemic control, balance and neuropathic symptom, and some dimensions quality of life in adults with diabetes and neuropathy (Ahn & Song, 2012). Structured lifestyle intervention trials that include physical activity at least 150-175 min/week and dietary energy restriction targeting weight-loss of 5%-7% have demonstrated reductions of 40%-70% in the risk of developing T2DM in people with impaired glucose tolerance (Church *et al.*, 2010).

2.10.3 A Combination of diet, exercise and medication

Praet and Van Loon (2007) have suggested that intervention programmes that include a combination of physical activity, healthy diet and oral blood-glucose-lowering medication are

effective ways to manage and treat T2DM (Praet & Van Loon, 2007; Praet & Van Loon, 2008). Continuous endurance-type exercise training has been shown to lower HbA1c levels, increase insulin sensitivity and reduce adipose-tissue mass in patients with T2DM (Boulé *et al.*, 2001; Snowling & Hopkins, 2006). Exercise is the only programme that has consistently been shown to improve whole body and skeletal muscle oxidative capacity (Toledo *et al.*, 2008). Change in dietary carbohydrate for protein in a low-fat (<30% total energy) diet may increase body composition and cardiovascular disease (CVD) risk factors including glycaemic control, and blood lipids in obese populations with T2DM (Parker, Noakes, Luscombe & Clifton., 2002; Samaha *et al.*, 2003).

2.10.4 Activities of Daily Living (ADLs)

Increasing unstructured physical activity (e.g. running errands, household tasks, dog-walking or gardening) increases daily energy expenditure and assists with weight management (Levine, Eberhart & Jensen, 1999; Levine and Kotz, 2005). Unstructured activity also reduces daily sitting time and increasing activity bouts (3 -15 minutes) are effective in acutely reducing postprandial hyperglycemia and improving glycaemic control in those with pre-diabetes and type 1 and T2DM, most prominently after meals (Colberg *et al.*, 2009; Dempsey *et al.*, 2016; Dunsten *et al.*, 2012; Henson *et al.*, 2016; Larsen *et al.*, 2015; Manohar *et al.*, 2012; Nygaard, Tomten & Hostmark, 2009; Van Dijk *et al.*, 2013). Increasing unstructured physical activity should be encouraged as part of a whole-day approach or at least initially as a stepping stone for individuals who are sedentary and unable/reluctant to participate in more structured exercise.

CHAPTER 3

Methods

3.1 Introduction

This chapter provides an outline of the research methods that the researcher used in this study. Information on the participants is provided, the inclusion and exclusion criteria and how the participants were sampled. The research design that was chosen for this study is also described in this chapter. The researcher explains the instruments that were used for data collection and analysis of the procedures that were followed, plus ethics consideration.

3.2 Design and setting

This research utilized a quantitative research approach using a pre-test post-test design with a 12-week exercise intervention. A pre-test post-test design is an experimental design where measurements are taken before and after a treatment (Dimitrov & Rumrill, 2003). This design means that the researcher is able to see the effects of some type of treatment on a group. The design of this study was true-experimental, meaning that there was a control group and the participants were assigned randomly to the groups (Gribbons & Herman., 1996). Participants were recruited from the staff at a university in the Western Cape, which included lecturers, administrators and lab assistants.

3.3 Population and sampling

The university population at the time of recruitment consisted of approximately 25,000 staff members and students, however, the current study focused only on the full-time staff members. For this study, 40 female participants were initially purposefully recruited, and checked for eligibility. The current ACSM guidelines (10th edition) (Riebe, Ehrman, Liguori & Magal, 2018) classify individuals as physically active or inactive according to the level of exercise or

physical activity engagement over the past 3 months (defined as planned, structured physical activity of at least 30 min of moderate intensity on at least 3 days per week). Screening outcome was determined based on presence or absence of known cardiovascular, metabolic, or renal disease and the presence or absence of signs or symptoms of these conditions with recommendations for medical clearance based on the desired intensity of exercise (Price *et al.*, 2018). Written consent was received from the participants, and they were randomly divided into two groups: An intervention group and a control group. The participants were recruited through emails, word of mouth and pamphlets.

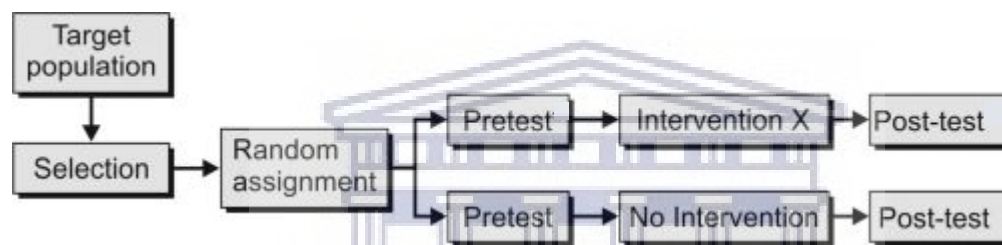


Figure 3.1 Flow diagram of the sampling and design

3.4 Inclusion and exclusion criteria

This study targeted female university employees who were obese, pre-diabetic or diabetic (Table 3.1) illustrates the inclusion and exclusion criteria for participants for the study. Participants who met the inclusion criteria were recruited as participants. Those who did not were excluded in an attempt to avoid participants experiencing hyperglycaemia.

Table 3.1 Inclusion and exclusion criteria for participant selection

Inclusion Criteria	Exclusion Criteria
1. Obese: BMI > 30.0 kg·m ⁻² Waist circumference > 88 cm	1. Uncontrolled blood pressure with no medication.
2. Pre-diabetic or Diabetic: Fasting blood glucose > 5.5 mmol·L ⁻¹	2. Known cardiovascular disease and/or pulmonary disease.
3. Medical clearance was required from all participants.	3. Any orthopaedic condition that will prevent them from exercising.

3.5 Procedures

Participants who volunteered for the study were verbally informed of the research aim and were provided with an information sheet (Appendix A), which explained the study in more detail and allowed them to ask questions. After the participants volunteered to participate in the study, they were asked to sign a consent form (Appendix B). At that point, participants were checked to ensure that they met the inclusion criteria and body composition and fasting blood glucose were measured.

Participants were then randomly allocated to the intervention group, who underwent the aerobic exercise-training programme, or to the control group, who did not participate in any formal exercise training for 12 weeks. After the 12 weeks of aerobic exercise training for the intervention group was complete, participants from the control group were given the opportunity to undergo the same exercise programme.

Assessments of anthropometric variables, clinical variables and ratings of dyspnoea were conducted prior to the commencement of the intervention as well as post-intervention. All testing and exercise sessions took place in the Biokinetics practice at the university.

3.5.1 Validity of the instruments

Construct validity was applied in this study as a measure to validate the effects of an aerobic exercise training programme on type 2 diabetes mellitus, body fat percentage and dyspnoea on obese females. Construct validity is defined as the degree to which a test measures what it claims, or purports, to be measuring (Brown, 1996; Chronbach & Meehl, 1955; Polit & Beck, 2012).

3.5.2 Reliability

Test-retest reliability was applied in this study to assess the consistency of a measure across time. Test-retest reliability is the closeness of the agreement between the results of successive measurements of the same measure carried out under the same conditions of measurement (Polit, 2014). In other words, measures of reliability obtained by administering the same test twice over a period of time to a group of individuals. The same tests were carried out in the same way for each individual to ensure consistency. The same instruments were used both pre and post intervention. The participants in the intervention group did the same exercises during the intervention period. The participants were given the same information about the intervention programme and the aerobic training programme was similar for each individual.

3.5.3 Tests

3.5.3.1 Height

Before and after the 12-week intervention, all participants' height was measured using a portable stadiometer. The stadiometer was positioned against the wall for support, wet wipes were used to clean the stadiometer. The participants were requested to remove their shoes and stand on the stadiometer with their feet together, heels and torso against the Stadiometer. Baharudin *et al.* (2017) valuated the reliability of measurement of portable Stadiometer and the inter-examiner, intra-examiner and inter-instrument correlation coefficients for height measurement were almost perfect.

3.5.3.2 Body Composition

Body composition was assessed using a Tanita-300™ Bioelectrical Impedance Analysis (BIA) scale that provided measures of body mass (kg) and percent body fat. The scale was cleaned using wet wipes. It was then switched on and the participants' height, age and waist circumference were entered on to the scale. The participants were then requested to step on the scale (bare-footed) until the scale reflected their results. Von Hurst *et al.* (2016) assessed the validity and reliability of BIA against Air Displacement Plethysmography (ADP) and Dual-energy X-ray Absorptiometry (DXA) to measure BF%, and to test the reliability of each method. BIA showed excellent relative agreement to the estimated true value ($\rho = 0.97$ (0.96, 0.98)) and to ADP ($R^2 = 0.88$) and DXA ($R^2 = 0.92$), but wide limits of agreement (-4.25 to 8.37%). BIA underestimated BF% by 2%, across all values. DXA showed excellent relative agreement to the estimated true value ($\rho = 0.97$ (0.96, 0.98)) and with ADP ($R^2 = 0.92$), good absolute agreement but wide limits of agreement (-6.13 to 6.91%) and under- and overestimation at high and low BF % levels, respectively. All methods showed excellent reliability with repeat measurements differing by less than 0.2% with very small 95%. BIA may be a valid method in research and population samples. All three methods showed excellent reliability.

Ritchie, Miller and Smiciklas-Wright (2005) used the Tanita BIA scale for body-composition assessment of older adults. The correlation for percent body fat measurements between the Tanita and traditional BIA was $r=0.84$ ($p<.001$). Percent body fat estimates from both BIA measures were significantly correlated with waist circumference, body mass index, and age (all $p<0.01$). Their research shows that Tanita BIA scale provides a valid measure of percent body fat in older adults, and could be a convenient and practical approach for assessment in public health settings.

Body mass index (BMI, ratio of height and weight, expressed as $\text{kg}\cdot\text{m}^{-2}$) is widely used to define overweight and obesity across many countries, populations, races and ethnicities. It is obvious that weight and height are linked (Misra & Dhurandhar., 2019). Hence, to adjust for the proportion between height and weight, the use of BMI assumes that in a given population, weight scales to height squared. This assumption, as well as the thresholds for defining overweight (BMI between 25 to $<30 \text{ kg}\cdot\text{m}^{-2}$) or obesity ($\text{BMI} \geq 30 \text{ kg}\cdot\text{m}^{-2}$) have been derived from studying Caucasian population, hence may or may not apply to various other groups globally (Hood *et al.*, 2018).

Table 3.2 BMI classification

BMI ($\text{kg}\cdot\text{m}^{-2}$)	Classification
< 18	Underweight
18.5 – 24.9	Normal weight
25.0 - 29.9	Overweight
30.0 – 34.9	Class I Obesity
35.0 – 39.9	Class II Obesity
>40	Class III Obesity (Morbid Obesity)

3.5.3.3 Aerobic Capacity

Participants were then taken through the YMCA Sub-Maximal Cycle Ergometer test before and after the intervention programme. This is a test of submaximal aerobic capacity based on the subject's heart rate response to several submaximal workloads. From these data, $\text{VO}_{2\text{max}}$ can be predicted by extrapolating the heart rate they would have reached if they had continued until their maximum heart rate (Beekley *et al.*, 2004). A Monark 847e cycle ergometer was used to conduct this test.

All participants were given detailed instructions for rating of perceived breathlessness (RPB) during exercise as described previously (Babb *et al.*, 2008). Scales (0–10 Borg scale for rating of perceived breathlessness, 6–20 Borg scales for RPE) with verbal expressions of severity anchored to specific numbers were used (Bernhardt *et al.*, 2016; Borg, 1982). Ratings of perceived breathlessness (dyspnoea) were collected every two minutes of the test and the last value recorded was used for analysis.



Figure 3.1 Modified Borg Dyspnoea Scale (RPB)

The cycle ergometer was calibrated prior to all testing (according to the manufacturer’s instructions) and all equipment was checked to make sure that it was fully functional. Screening of health risks was performed and informed consent was obtained prior to the commencement of the intervention programme. Resting heart rate and blood pressure measurements were obtained and the test procedure was explained to the participants. The cycle ergometer seat height and handlebar height were adjusted for each participant and they were allowed a brief

period to orientate themselves with the equipment before the warm up began. Screening of health risks was performed and informed consent was obtained. Information such as age, height, body weight, gender, and test conditions were recorded before the commencement of the intervention programme.

During the test, participants were asked to pedal on the cycle ergometer at 50 revolutions per minute (rpm) for 3 minutes at a resistance of 0 kg, which was the warm-up. From there, the resistance was increased in increments of 0.5kg every 3 minutes until a steady state that included 85% of predicted heart rate max was reached, after which recovery commenced at 0.5 kg resistance. Heart rate was measured at the end of each minute and blood pressure was measured within the last minute of each 3 minutes interval throughout the test (Garatachea *et al.*, 2017).

Garatachea *et al.* (2017) did a study that aimed to investigate the validity of the YMCA Submaximal Cycle Ergometer Test to predict energy expenditure (EE) at submaximal intensities. Twenty-eight participants, both male and female volunteered for the study and performed a maximal oxygen uptake ($\text{VO}_{2\text{max}}$) incremental test, a submaximal exercise test (three 5-min loads at 40%, 55%, and 70% $\text{VO}_{2\text{max}}$) and the YMCA Test that provides an extrapolation based on heart rate (HR) to predict VO_2 (YMCA $\text{VO}_2\text{—HR}$). Results from their study shows that predicted $\text{VO}_{2\text{max}}$ from the YMCA Test was significantly different from the gold standard. However, predicted VO_2 at different relative submaximal intensities did not significantly differ from directly measured VO_2 . In conclusion, the HR method using the YMCA $\text{VO}_2\text{—HR}$ relationship appears to be effective in predicting energy expenditure at submaximal intensities but not at maximum efforts.

These tests were repeated after 12 weeks for all participants and predicted $\text{VO}_2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and anthropometric measures of body composition were then compared with the pre-test data

to determine if the intervention had any effect. The researcher, a qualified Biokineticist, performed the tests with the assistance of trained Biokinetics Honours students and interns. The researcher monitored the exercise programme throughout to make sure that it was executed accurately.

3.6 Aerobic Intervention

Participants in the intervention group underwent an exercise training programme (Appendix C) that consisted of moderate intensity exercise for 45 minutes (which included 5 minutes warm up and 5 minutes cool down), 3 days a week for 12 weeks (Colberg *et al.*, 2010, Garber *et al.*, 2018). At the beginning of each exercise session, blood pressure and fasting blood glucose levels were assessed to ensure that it was safe for the participant to exercise on each day. Furthermore, blood pressure and blood glucose levels were assessed after each exercise session to check for an appropriate response and to monitor the risks of post-exercise hypoglycaemia to ensure participant safety before they departed.

The warm up consisted of brisk walking for 5 minutes, followed by dynamic stretches for 5 minutes. During the aerobic exercise intervention, a circuit of four stations was followed, with participants moving from one station to the next every seven minutes, with a two minutes interval that allowed them to move from one station to the next, to wipe sweat and drink water. This programme was designed to differ from programmes in other intervention research, in that the participants used multiple stations to complete the aerobic exercises, rather than just one.

The intervention group began exercising at an intensity of 40% predicted heart rate reserve (HRR) on the treadmill, and progressed by 5-10% every 4 weeks, to accommodate increased fitness (Appendix C). Heart rate was monitored throughout each session using a Polar™ RS800™ heart monitor. After each session, there was a cool down session involving slow

walking for five minutes followed by active static stretches for a further 5 minutes. The entire exercise training took approximately 45 minutes per session.

3.7 Data analysis

Data analyses were carried out using the Statistical Package for Social Science (SPSS) software Version 25.0. Descriptive statistics, such as mean and standard deviations, were used to describe the baseline characteristics of the participants and dependent variable scores for each group. A One-Way Analysis of Variance (ANOVA) with repeated measures was used to determine whether there were any statistically significant differences between the means of the two groups and within the groups. Cohen's D was computed as $D = \frac{M1 - M2}{Sp}$, where M1 and M2 signify the sample means for group 1 and 2 and symbolises the collective expected population standard deviation. Cohen's D was an appropriate effect size when comparing between two means. A Cohen's D of 0.2 was considered to be a 'small' effect size; 0.5 symbolises a 'medium' effect size and 0.8 signifies a 'large' effect size (Cohen.,1992) (Thalheimer & Cook.,2002). All statistical tests were one-tailed and $p < 0.05$ was considered statistically significant.

3.8 Ethics considerations

Permission to conduct the study was requested from the university's Biomedical Research Ethics Committee and ethics clearance was provided (ethics number BM17/8/11). Permission to recruit university staff was sought from the university Registrar and line managers as necessary. Participants were informed verbally about the study in a form of a written information sheet and any queries arose were addressed. The risks and benefits of participating in the study were explained and it was made clear that their participation was purely voluntary and they had the right to withdraw from the study at any-time without prejudice. Participants

who agreed to be part of the study were asked to sign a consent form and were assigned a participant ID.

The names of the participants were not be revealed at any time and anonymity was ensured through the use of a participant ID code allocated by the research supervisor from the consent forms, so all subsequent forms had only their ID code on them. Furthermore, no names will be used on any publication emanating from this research. All information about the participants was kept confidential with data forms being kept in the supervisor's office in a locked cabinet and/or on a password protected computer. All data will be stored for 5 years, after which it will be destroyed, as per the university requirements.

Blood glucose levels were measured before and after exercise sessions for safety precautions. The participants exercised in an air-conditioned environment to prevent any possibility of heat illnesses during the hot summer temperatures, when data collection took place. No participant experienced any signs nor symptoms of a heat illness. Participants exercised in a safe environment free of loose weights and obstacles that might cause injuries. The risk of injury was minimized by providing an adequate warm up before each exercise session (brisk walking and dynamic stretches) as well as a cool down, which involved slow walking and static stretches.

CHAPTER 4

Results

4.1 Introduction

This chapter reports on the results of this current study in accordance to the objectives and hypothesis of the study. The study aimed to investigate the effects of a 12-week aerobic exercise training programme on insulin resistant, obese female university at employees. Data have been organised, analysed and reported to determine whether the researcher's designed exercise intervention had an effect on the participants' body fat percentage, glucose levels, waist circumference and dyspnoea.

4.2 Characteristics of the sample

The participants were all female. The participants were between the ages of 40 and 63 years. The participants in both the control and the intervention group were diabetic and obese but they were not hypertensive. They did not have any other chronic conditions beside being diabetic. There were 43 participants in total, 22 participants in the control group and 21 participants in the intervention group. The control group was not allowed to engage in any exercise program, whereas the intervention group were engaged in a 12-week, supervised intervention programme.

The participants were engaged in an aerobic training programme without caloric restriction. At the beginning of the intervention, it was observed that the participants were struggling to complete a session of exercise without pausing/resting. After the 8th week, however, most participants could complete their exercise sessions without taking any breaks between the sets. It was also observed that, in the first few weeks of the intervention, most of the participants were unable to hold a conversation during the exercise session, as they would run out of breath while trying to talk.

4.3 Between group comparisons at baseline (pre-test): Control vs intervention group

This study was conducted with 43 participants, 21 in the intervention group and 22 participants in the control group. Between group comparisons of anthropometric variables are illustrated in Table 4.1, which includes mean, standard deviations, the mean difference between the means for the control and intervention group and the *p*-value.

4.3.1 Anthropometric variables

The table 4.1 below shows results comparison for anthropometric variables for the control and intervention group at pre-test (baseline).

Table 4.1 Anthropometric variables at baseline

Variable (units): Group		Mean \pm SD	Mean Difference	<i>p</i> -value	Effect Size (<i>d</i>)
BMI (kg·m ⁻²):	Control	33.12 \pm 2.08	1.94	0.251	0.40
	Intervention	35.07 \pm 6.49			
Waist Circumference (cm):	Control	97.42 \pm 11.28	0.23	0.341	0.34
	Intervention	101.47 \pm 12.27			
Waist-to-height ratio:	Control	0.60 \pm 0.05	0.08	0.003*	0.04
	Intervention	0.62 \pm 0.79			
Percent Body Fat (%):	Control	44.73 \pm 3.45	2.33	0.303	0.29
	Intervention	46.50 \pm 7.86			

* significant at $p < 0.01$

The differences between the two groups at baseline (pre-test) are presented in Table 4.1. Means and standard deviations are shown for each anthropometric variable measured as well as the difference between these means for the intervention group and control group. A positive mean difference indicates a higher mean in the intervention group compared to the control group. Lastly, the *p*-value from the independent t-tests is provided for each comparison. As can be

seen, both groups fall into the obese category for BMI ($> 30 \text{ kg}\cdot\text{m}^{-2}$) and were not significantly different from each other.

Similarly, waist circumference was high and above the risk threshold of 88cm for CV, metabolic or renal disease (ACSM, 2018). Body fat percentage is also high for both groups and well above the 18-22% recommended for females (ACSM, 2018). The only significantly different variable ($p=0.003$) between the groups at baseline was the waist-to-height ratio, with a difference of 0.08, which is not very large, but the variation in the intervention group was broad. Comparisons of the clinical variables between the control and the intervention group at baseline are illustrated in table 4.2. The clinical variables included fasting blood glucose, blood pressure and ratings of dyspnoea (on a scale of 1-10).

The effect size (Cohen's d) for BMI, waist circumference, and percentage body fat between the control and intervention groups at baseline was medium (0.5). The effect size of waist-to-hip ratio between the control and the intervention group was small at baseline. The mean for BMI in the control group was 65.4% below the mean of the intervention group with an effect size of 0.40. The mean for waist circumference was 61.7% lower in the control group compared to the intervention group with the effect size of 0.34. Waist-to-height ratio showed an effect size of 0.04 at baseline (50%) and body fat percentage had an effect size of 0.29, which puts the mean score of the control group 58% and 61.7% lower than the mean score of the intervention group respectively.

4.3.2 Clinical variables

Table 4.2 below shows the comparison for clinical variables between control and intervention group at baseline. As can be seen on the table, the mean difference for fasting blood glucose and systolic blood pressure are positive and the mean difference for diastolic blood pressure and dyspnoea are negative. A positive mean difference indicates a decrease in the mean for the

intervention group post-intervention whilst a negative mean difference indicates an increase in the mean for the intervention group. This means that fasting blood glucose and systolic blood glucose decreased in the intervention group compared to the control group, diastolic blood pressure and dyspnoea increased in the intervention group compared to the control group.

Table 4.2 Clinical variables at baseline

Variable (units): Group	Mean ± SD	Mean Difference	p-value	Effect Size (d)
Fasting blood glucose (mmol·L ⁻¹): Control	7.24 ± 0.74	4.04	0.319	0.25
Intervention	7.56 ± 1.68			
Systolic blood pressure (mmHg): Control	113.66 ± 9.36	0.32	0.458	0.10
Intervention	114.57 ± 8.80			
Diastolic blood pressure (mmHg): Control	71.23 ± 9.03	-0.90	0.741	0.31
Intervention	68.90 ± 5.36			
Dyspnoea rating (/10): Control	7.61 ± 0.74	-1.77	0.411	1.20
Intervention	6.80 ± 0.60			

These results, for the anthropometric and clinical variables, show that there were no significant differences between the two groups at baseline, except for waist-to-height ratio, so the two groups can be viewed overall as homogenous. All the mean differences were positive, except for diastolic blood pressure and ratings of dyspnoea, indicating a lower mean in the control group compared with the intervention group.

The effect size for fasting blood glucose and diastolic blood pressure was medium (less than 0.5), the effect size of systolic blood pressure at baseline between the control and the intervention group is small (less than 0.2) and the effect size for dyspnoea at baseline between control and intervention group is large.

Fasting blood glucose had an effect size of 0.25 at baseline. Systolic blood pressure had an effect size of 0.10. The effect size for diastolic blood pressure was 0.31 at baseline.

4.4 Between group comparisons at post-test: Control group vs intervention group

4.4.1 Anthropometric variables

The mean difference between the groups for anthropometric variables from pre- to post-intervention are presented in Table 4.3. As can be seen, there were no significant differences between the groups for BMI, waist circumference or percent body fat. Although the waist-to-height ratios were almost identical for both groups, they were significantly different ($p=0.000$).

Table 4.3 Anthropometric variables at post-test

Variable (units): Group	Mean \pm SD	Mean Difference	<i>p</i> -value	Effect Size (<i>d</i>)	
BMI (kg·m ⁻²):	Control	33.95 \pm 2.23	0.45	0.790	0.09
	Intervention	34.40 \pm 6.54			
Waist Circumference (cm):	Control	99.62 \pm 11.32	0.09	0.980	0.01
	Intervention	99.71 \pm 12.42			
Waist-to-height ratio:	Control	0.61 \pm 0.06	0.00	0.000*	0.00
	Intervention	0.61 \pm 0.08			
Percent Body Fat (%):	Control	45.43 \pm 3.63	2.00	0.270	0.06
	Intervention	45.84 \pm 7.96			

The effect size for BMI, waist circumference, waist-to-hip ratio and percentage body fat between the control and intervention group after the intervention programme was small. BMI had an effect size of 0.05, waist circumference had an effect size of 0.01, waist-to-height ratio had an effect size of 0.00 whilst body fat percentage had an effect size of 0.06. There was 50% overlap in the above anthropometric variables post-intervention.

4.4.2 Clinical variables

The table 4.4 below presents results for clinical variables between the control and intervention group at post-intervention assessment. After the intervention programme, the mean for fasting blood glucose, diastolic blood pressure and dyspnoea increased in the control group and decreased in the intervention group, hence the negative mean differences. However, systolic blood pressure increased with a mean difference of 0.45 mmHg. As indicated previously, both systolic and diastolic blood pressure were still within the normal ranges (<120mmHg/<80mmHg) despite the fluctuations.

Fasting blood glucose had an effect size of 0.40, which is considered to be medium by Cohen (1988). Systolic blood pressure had a small effect size (0.04), diastolic blood pressure had a medium effect size (0.27) and dyspnoea had a large effect size of 1.49.

Table 4.4 Clinical variables at post-test

Variable (units): Group	Mean ± SD	Mean Difference	p-value	Effect Size (d)
Fasting blood glucose (mmol·L ⁻¹): Control	7.48 ± 0.60	-0.95	9.810	0.40
Intervention	7.02 ± 1.50			
Systolic blood pressure (mmHg): Control	115.57 ± 9.01	0.45	0.246	0.04
Intervention	115.97 ± 7.65			
Diastolic blood pressure (mmHg): Control	69.14 ± 7.37	0.38	0.880	0.27
Intervention	71.14 ± 7.03			
Dyspnoea rating (/10): Control	6.71 ± 1.23	-0.40	0.850	1.49
Intervention	5.19 ± 0.74			

4.5 Within group comparisons: Control group

For the control group, at the beginning of the research project, both anthropometric and clinical variables were high. The comparisons for all dependent variables within the control group from pre-test to post-test are now presented.

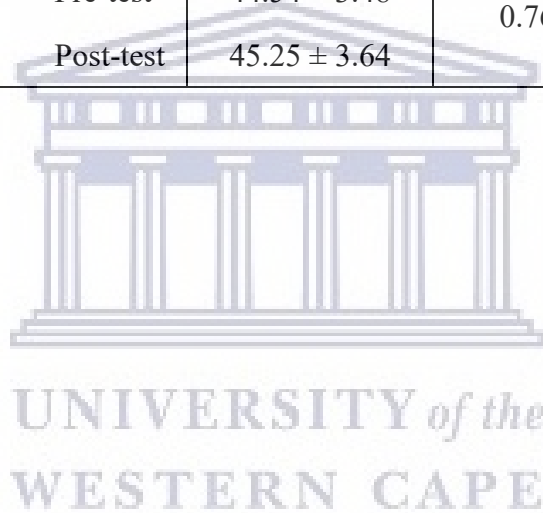
4.5.1 Anthropometric Variables

Table 4.5 presents the mean differences in the control group before and after the intervention programme for all the anthropometric variables. The findings above show a significant increase in BMI, waist circumference, waist-to-height ratio, and percent body fat ($p < 0.01$), which all increased in the control group from pre- to post-intervention. The effect size for BMI was 0.37, waist circumference had an effect size of 0.20, waist-to-height ratio had an effect size of 0.4 and body fat percentage had an effect size of 0.19. The effect size for BMI and waist-to-height ratio was medium and the effect size for waist circumference and percentage body fat was small after the intervention programme.

Table 4.5 Within group comparisons of anthropometric variables – Control group

Variable (units): Assessment time	Mean ± SD	Mean Difference	p-value	Effect Size (d)	
BMI (kg·m ⁻²):	Pre-test	33.01 ± 2.10	0.82	0.000*	0.37
	Post-test	33.83 ± 2.23			
Waist Circumference (cm):	Pre-test	97.45 ± 11.01	2.22	0.000*	0.20
	Post-test	99.68 ± 11.05			
Waist-to-height ratio:	Pre-test	0.59 ± 0.05	0.01	0.000*	0.4
	Post-test	0.61 ± 0.05			
Percent Body Fat (%):	Pre-test	44.54 ± 3.48	0.76	0.001*	0.19
	Post-test	45.25 ± 3.64			

* significant at $p < 0.01$



4.5.2 Clinical Variables

The table 4.6 below shows the clinical variables in the control group before and after the intervention programme.

Table 4.6 Within group comparisons of clinical variables – Control Group

Variable (units): Assessment time	Mean ± SD	Mean Difference	p-value	Effect Size (d)
Fasting Blood Glucose (mmol·L ⁻¹): Pre-test	7.22 ± 0.72	0.23	0.000*	0.35
Post-test	7.45 ± 0.59			
Systolic Blood Pressure (mmHg): Pre-test	113.77 ± 9.15	1.63	0.269	0.18
Post-test	115.40 ± 8.82			
Diastolic Blood Pressure (mmHg): Pre-test	70.82 ± 9.04	-2.09	0.940	0.25
Post-test	68.72 ± 7.45			
Dyspnoea (/10): Pre-test	7.59 ± 0.73	-0.90	0.000*	0.91
Post-test	6.68 ± 1.21			

* Significant at $p < 0.01$

As can be seen, fasting blood glucose, systolic blood pressure increased post-intervention, whereas there was a decrease in diastolic blood pressure and dyspnoea. The only significantly different variables in this table are fasting blood glucose with a mean difference of -0.23 mmol·L⁻¹ ($p=0.000$) and dyspnoea with a mean difference of 0.90 ($p=0.000$).

The effect size for fasting blood glucose and diastolic blood pressure in the control group between pre and post intervention was medium. A small effect size was calculated for systolic blood pressure however the effect size for dyspnoea rating between pre- and post-test was large.

4.6 Within group comparisons: Intervention group

4.6.1 Anthropometric Variables

Table 4.7 below shows the within group results for anthropometric variables after the intervention programme: intervention group.

Table 4.7 Within group comparisons of anthropometric variables - Intervention group

Variable (units): Time of assessment	Mean ± SD	Mean Difference	p-value	Effect Size (d)	
BMI (kg·m ⁻²):	Pre-test	35.07 ± 6.49	-0.66	0.000*	0.10
	Post-test	34.4 ± 6.54			
Waist-to-height ratio:	Pre-test	0.62 ± 0.08	-0.01	0.000*	0.13
	Post-test	0.61 ± 0.07			
Waist Circumference (cm):	Pre-test	101.48 ± 2.28	-1.76	0.000*	0.19
	Post-test	99.71 ± 12.4			
Percent Body Fat (%):	Pre-test	46.51 ± 7.86	-0.66	0.000*	0.08
	Post-test	45.84 ± 7.96			

* significant at $p < 0.01$

The mean differences for the clinical variables between pre-and-post interventions are negative, indicating that all the scores all went down as a result of the intervention. These changes were all significant at $p < 0.01$.

There was an overlap of 54% in BMI, waist-to-height ratio and waist circumference. Body fat percentage had an overlap of 50% with an effect size of 0.08. BMI had an effect size of 0.10, waist-to-hip ratio had an effect size of 0.13 whereas waist circumference had an effect size of 0.19.

The following graphs show the changes between the control and intervention groups from pre- to post-testing for each variable. In figure 4.1, BMI for the control group increased by 2.5% during the 12-week intervention, but the intervention group's BMI decreased significantly by 7.2% ($p < 0.01$).

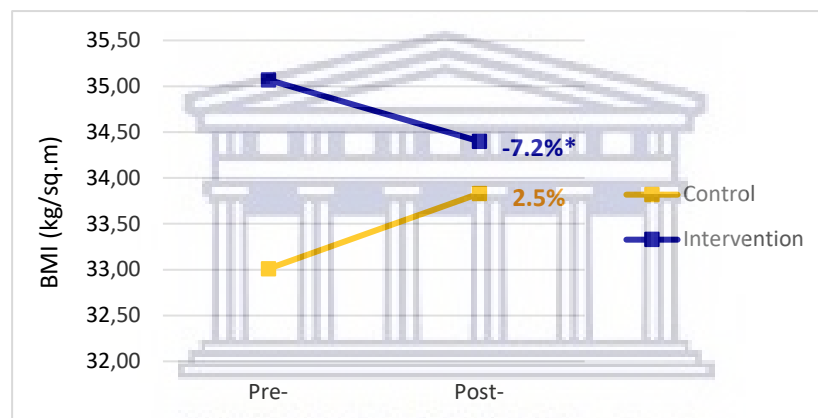


Figure 4.1 Change in BMI of the intervention and control group (post-intervention).

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Figure 4.2 represents the changes from pre-to-post intervention for waist circumference in the control and intervention groups. As can be seen, the waist circumference of the control group increased by 2.3% from pre-to post-test, but it decreased by 1.7% in the intervention group.

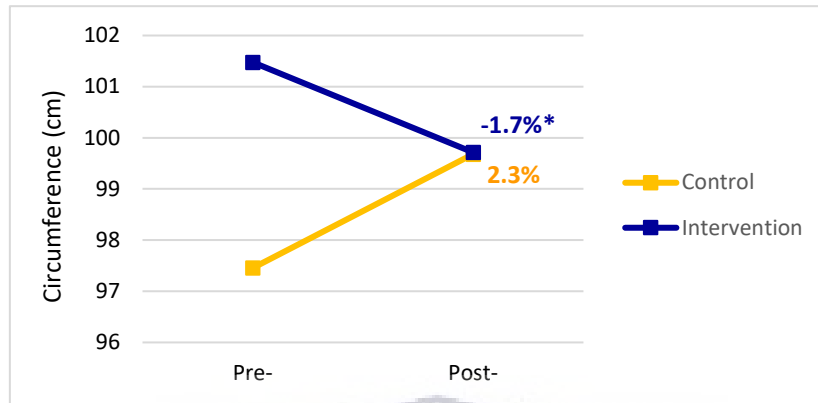


Figure 4.2 Change of Waist Circumference in the intervention and control group

Figure 4.3 depicts the changes in weight-to-height ratio from pre- to post-test for the control and intervention groups. The graph shows that waist-to-hip ratio decreased by 1.6% in the intervention group as a result of the intervention, but increased in the control group by 1.7% over the same period.

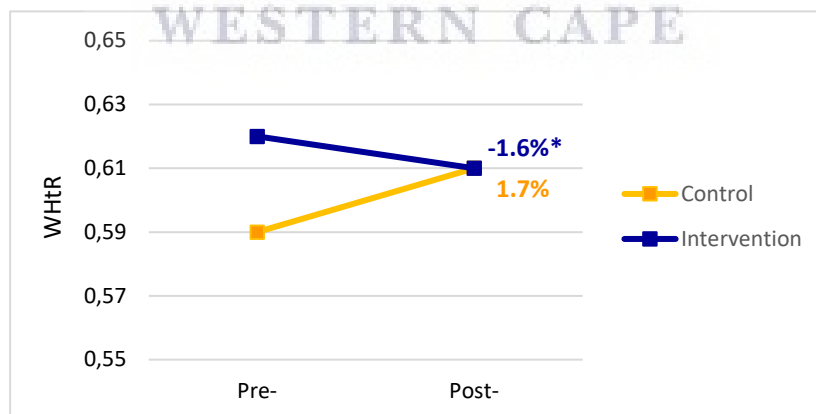


Figure 4.3 Change in Waist-to-height ratio of the intervention and control group

In terms of percent body fat, Figure 4.4 presents the change in each of the groups from pre-and-post intervention. As can be seen, body fat percentage for the intervention group decreased by 1.4% over the 12 weeks whilst it increased by 1.6% in the control group.

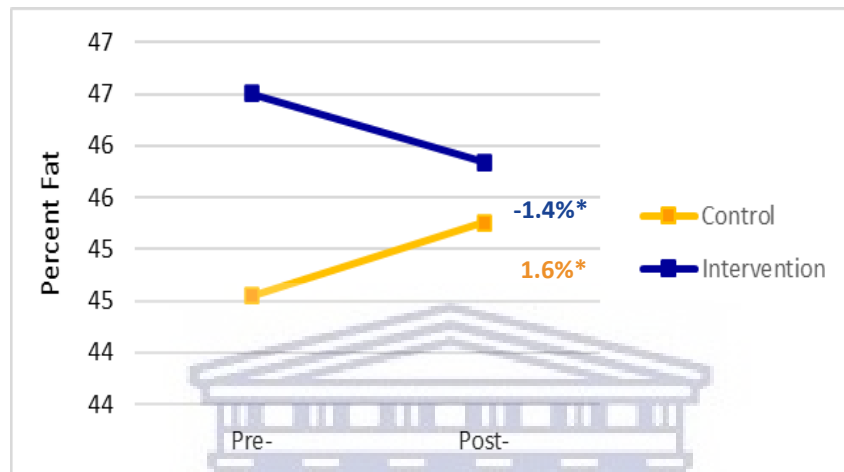


Figure 4.4 Change in Body fat percentage of the intervention and control group

4.6.2 Clinical variables

In this study, 12 weeks of aerobic exercise training resulted a change in the fasting blood glucose levels of the participants throughout the 12 weeks. The mean score of the participants before engaging in the exercise programme was $7.56 \pm 1.68 \text{ mmol}\cdot\text{L}^{-1}$ and after the intervention programme the mean score of their blood glucose level was $7.0 \pm 1.50 \text{ mmol}\cdot\text{L}^{-1}$ (mean difference = 0.53). Systolic blood pressure increased post aerobic intervention with a mean score of 114.57 ± 8.80 (difference of -1.38), diastolic blood pressure also increased with a mean score of 115.95 ± 7.65 (difference of -2.23) however both systolic and diastolic blood pressure were still within the normal range pre-and-post intervention.

The mean score of the participants before engaging in the intervention programme was 6.80 ± 0.60 to a mean score of 5.19 ± 0.72 (mean difference = 1.61). The increase in exercise intensity

resulted in a decrease in dyspnoea, dyspnoea decreased after the 12-week aerobic training programme. The results for the within group clinical variables comparisons are shown in Table 4.8.

Table 4.8 Within group comparisons of clinical variables - Intervention group

Variable (units): Time of assessment	Mean ± SD	Mean Difference	p-value	Effect Size (d)
Fasting Blood Glucose (mmol·L ⁻¹): Pre-test	7.56 ± 0.73	-0.53	0.082	0.45
Post-test	7.02 ± 1.50			
Systolic Blood Pressure (mmHg): Pre-test	114.57 ± 8.80	1.38	0.296	0.16
Post-test	115.95 ± 7.65			
Diastolic Blood Pressure (mmHg): Pre-test	68.90 ± 5.37	2.23	0.082	0.35
Post-test	71.14 ± 7.03			
Dyspnoea rating (/10): Pre-test	6.80 ± 0.60	-1.61	0.000*	2.38
Post-test	5.19 ± 0.74			

* significant at $p < 0.01$

The above table presents the mean difference of the clinical variables in the intervention group before and after the intervention programme. There was a decrease in fasting blood glucose and dyspnoea. The only significantly different ($p=0.000$) variable is for the dyspnoea ratings with a mean difference of -1.61, indicating the pre-test score was higher than the post-test score. Systolic blood pressure increased after the intervention with a mean difference of 1.38 mmHg and diastolic blood pressure also increased after the intervention programme with a mean difference of 2.23 mmHg, however they were still within the normal range (<120mmHg / <90mmHg).

The effect size for fasting blood glucose was 0.45 with an overlap of >65.4%, systolic blood pressure was 0.16 with an overlap of > 54%, the effect size for diastolic blood pressure was 0.35 with an overlap of > 61.7%. Lastly, dyspnoea had an effect size of 2.38 with an overlap

of > 92%. The effect size for fasting blood glucose and diastolic blood pressure test was medium after the intervention programme and the effect size of diastolic blood pressure was small.

The graph below (Figure 4.5) presents the comparison of results of the fasting blood glucose levels between the control and intervention groups from pre- to post-intervention. The decrease in the intervention group is clearly seen, although this change was not significant, whilst the FBG levels in the control group increased over the 12-week intervention period.

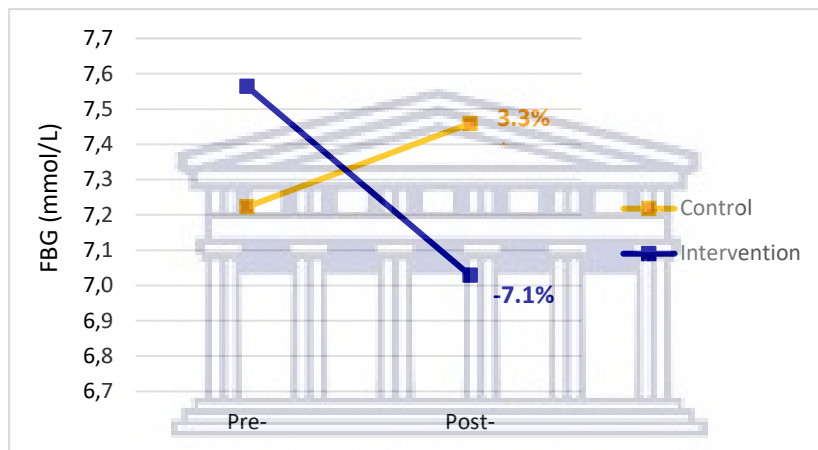


Figure 4.5 Change in Fasting blood glucose of the intervention and control group

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A comparison of means between the control and intervention group for systolic blood pressure is shown in Figure 4.6. Systolic blood pressure increased in both the control group (1.4%) and intervention group (1.2%). Blood pressure fluctuates all the time, even though systolic blood pressure increased in both groups, the values were still within the normal range.

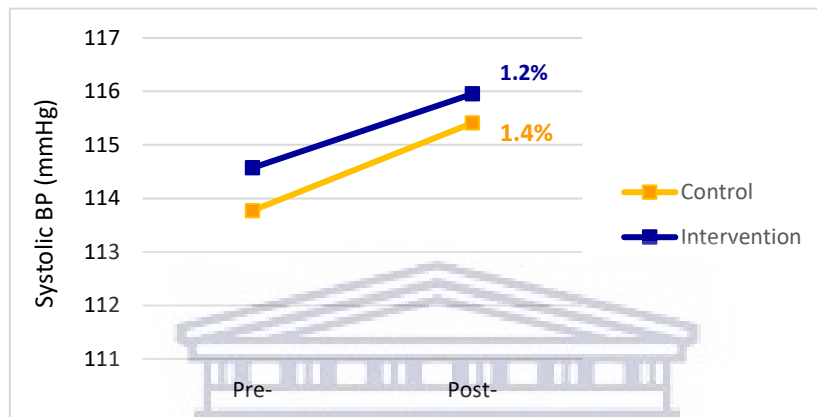


Figure 4.6 Change in Systolic blood pressure of the intervention and control group

The graph below presents the results for diastolic blood pressure from pre to post intervention (Figure 4.7). The results in the current study show that diastolic blood pressure decreased by -3.2% in the control group and increased by 3% in the intervention group, but both these measures were both still within the normal range.

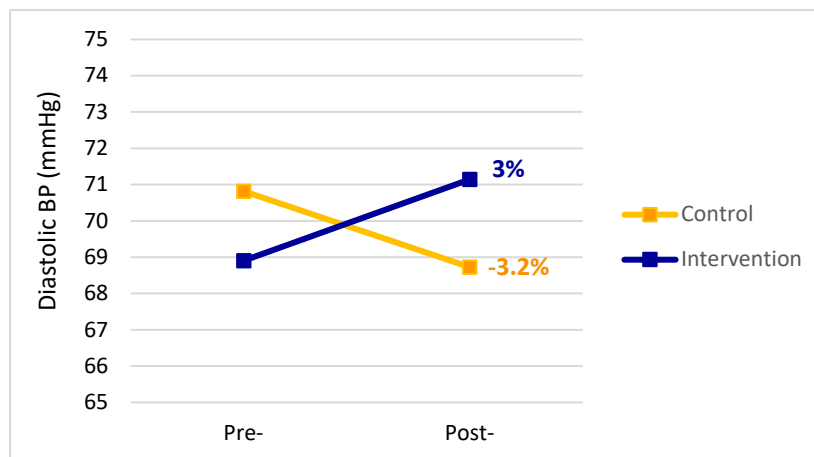


Figure 4.7 Change in Diastolic blood pressure of the intervention and control group

Figure 4.8 presents changes in ratings of dyspnoea in the control and intervention group from pre- to post-intervention. Ratings of dyspnoea decreased significantly ($p < 0.01$) in both groups, by 12% in the control group and by 24% in the intervention group, over the 12-week intervention period.

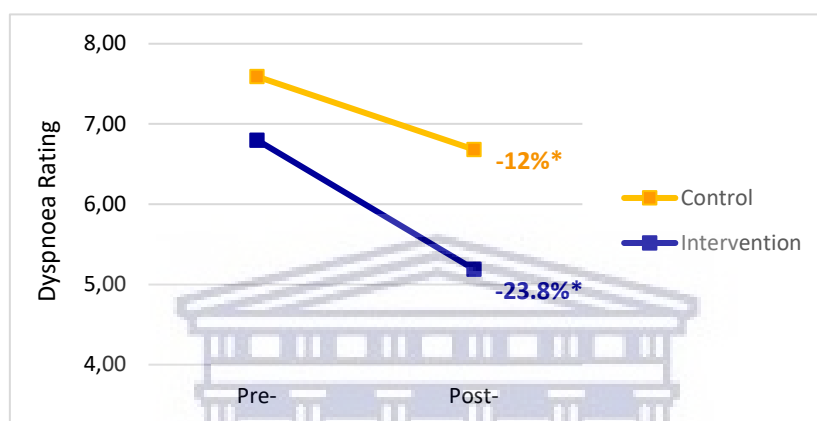


Figure 4.8 Change in Dyspnoea ratings of the intervention and control group

4.7. Summary

Based on the findings of this study, aerobic exercise has yielded positive outcomes on body mass index, body fat percentage, waist circumference, blood glucose levels and dyspnoea. The summary of the expected and observed changes from pre- to post-intervention are shown in Table 4.9.

Table 4.9 Summary of the expected and observed changes from pre- to post-intervention

Variable	Expected change post-intervention (Intervention Group)	Observed change	
		Control Group	Intervention Group
BMI (kg·m ⁻¹)	↓	↑ (2.5%)*	↓ (-1.9%)*
Waist-to-height ratio	↓	↑ (3.4%)*	↓ (-1.6%)*
Waist circumference (cm)	↓	↑ (2.3%)*	↓ (-1.7%)*
Body fat percentage (%)	↓	↑ (-0.8%)*	↓ (0.7%)*
Fasting blood glucose (mmol·L ⁻¹)	↓	↑ (3.3%)*	↓ (-7.1%)
Systolic blood pressure (mmHg)	↓	↑ (1.4%)	↑ (1.2%)
Diastolic blood pressure (mmHg)	↓	↓ (3.0%)	↑ (3.3%)
Dyspnoea rating (/10)	↓	↓ (-12%)*	↓ (-23.7%)*

Key: ↑ = increase; ↓ = decrease; * = significant at $p < 0.01$

The results in the current study provide evidence that, for the control group, all anthropometric variables increased, yet the same variables decreased in the intervention group over the same period, as a result of the exercise intervention. For the clinical variables fasting blood glucose and dyspnoea decreased post intervention whilst systolic and diastolic blood pressure increased after the intervention programme. Even though systolic and diastolic blood pressure increased post-intervention, they were still within the normal range

4.7.1 Hypothesis 1: Percentage body fat would decrease after the 12-week aerobic programme.

After the 12-week aerobic training programme, the percentage body fat significantly decreased with a mean difference of 0.66 ($p < 0.000^*$).

Hypothesis 1 is therefore supported.

4.7.2 Hypothesis 2: Fasting blood glucose levels would decrease after the 12-week aerobic programme.

There was a slight change in the fasting blood glucose (FBG) levels of the participants in the intervention group after 12-week aerobic exercise intervention. There was a significant decrease in the mean difference from pre-to-post intervention. The mean score for glucose before the intervention was 7.56 ± 1.68 and 7.02 ± 1.50 after the intervention programme (mean difference = 0.53).

Hypothesis 2 is therefore supported.

4.7.3 Hypothesis 3: Dyspnoea during exertion would improve after the 12-week aerobic programme.

The mean score of the participants before engaging in the intervention programme was 6.80 ± 0.60 to a mean score of 5.19 ± 0.74 after the intervention programme (mean difference = 1.61). Therefore, there was an improvement in dyspnoea after the 12-week aerobic training programme.

Hypothesis 3 is therefore supported.

Thus the findings of the current study present useful information for the design of future aerobic exercise intervention programme for university staff.

CHAPTER 5

Discussion

5.1 Introduction

The role of physical activity (PA) as an intervention to reduce the menace of chronic diseases caused by an unhealthy lifestyle cannot be underestimated. Globally, as the level of involvement in PA declines, people's health and quality of life are reduced, in direct proportion. In South Africa, more than half (51.1%) of the adult population is insufficiently active while many deaths are known to be caused by lifestyle-related risk factors such as the use of tobacco, excess body weight, alcohol consumption and physical inactivity (Krugar *et al.*, 2002). Many of these chronic diseases of lifestyle can be prevented and managed with aerobic physical activity/exercise interventions that improve fitness levels, reduce obesity, and improve overall wellbeing (Anderson & Durstine.,2019).

The participants in this study were all females aged between 40 and 63 years. The participants in both the control and the intervention group were diabetic and obese but they were not hypertensive. They did not have any other chronic conditions besides being diabetic. There were 43 participants in total, 22 participants in the control group and 21 participants in the intervention group. The control group were not allowed to engage in any exercise programme, whereas the other 21 participants that were involved in the intervention were supervised for a period of 12 weeks.

5.2 Between-group comparison: Pre-test and post-test

At baseline, the control group and the intervention group were essentially the same as there was only one significant difference (waist-to-hip ratio) in any of the variables between the two groups. The intervention group had a significantly larger waist-to-height ratio than the control

group, but had a much larger variance, which, accounts for the significant difference noted. It is important to understand the mechanisms that may explain the results, in order to get more insight.

The effect size for BMI was medium at 0.40, with an overlap of 65.4 %, waist circumference had an effect size of 0.35 with an overlap of 61.7%, body fat percentage had a medium effect of 0.29 with an overlap of between 58% and 61.7%, lastly waist-to-height ratio had an effect size of 0.04 with an overlap of 50% at baseline. There was 58% - 61.7 overlap in fasting blood glucose at baseline, 54 % overlap in systolic blood pressure, 61.7% overlap in diastolic blood pressure and 87.7 % overlap in dyspnoea at baseline.

There was an overlap of 65.4% in fasting blood glucose, an overlap of 50% in systolic blood pressure, diastolic blood pressure had an overlap of 58% whilst dyspnoea had an overlap of >92% after the intervention programme. The effect sizes for the anthropometric variables between the control and the intervention group were small after the intervention. This means that there was a 50% overlap in the mean score of their standard deviations. According to Cohen (1988), when the effect sizes are like this, they are too small to be differentiated by the naked eye. This, however, does not mean that there were no improvements after the intervention programme.

According to Booth *et al.* (2011) some of the mechanisms by which, inactivity causes chronic diseases is different from the mechanisms by which exercise acts as a key prevention for the same disease. Blümel *et al.* (2015) suggest that, although the prevalence of obesity is similar between males and females, it is important to consider that females undergo ovarian hormone decline as they age, which causes metabolic changes that significantly affect the risk of developing obesity. It is therefore important to consider the lifestyle that the participants might have been living prior to being involved in the intervention.

Results of a study by Bacopoulou, Efthymiou, Landis, Rentoumis and Chrousos (2015) suggest that there could be a positive impact on waist circumference, which is caused by peripheral fat accumulating predominantly in the lower body in females as compared to the upper body in males. Although, the study by Bacopoulou *et al.* (2015) focused on adolescents, the results provide a significant contribution to the current study. Although, participants in the current study's lifestyles were not evaluated, the results indicate common characteristics of both groups which can be witnessed by people living a sedentary lifestyle.

The baseline measurements of the participants in the current study portray characteristics of sedentary lifestyle living, which may lead to obesity, coronary heart disease, type 2 diabetes, breast cancer and colon cancer and other related chronic diseases (Kahan, 2015). Sedentary lifestyle is detrimental if interventions are not made in time. It therefore, becomes important to look at the data from the control group for the current study.

5.3 Within-group comparison: Control group

The results of the current study show that body mass index, waist circumference, waist-to-hip ratio and body fat percentage all increased in the control group from pre to post intervention. Increase in these variables can be attributed to the unhealthy lifestyle of these participants, which includes being sedentary. Fransen *et al* (2016) notes that unhealthy diet is associated with a higher risk of chronic diseases and mortality. It is therefore, worrisome that this could lead participants to serious chronic diseases and it is important for them to be made aware of their condition and the ominous consequences.

The effect size of BMI, waist-to-height ratio and fasting blood glucose were medium. There was an overlap of 61.7% in BMI, an overlap of 58% in waist circumference, an overlap of 65.4% in waist-to-height ratio and body fat percentage had an effect size of 54%. The above variables (BMI, waist-to-height ration and fasting glucose) had an effect size of 0.5 with a 33%

non-overlap, which means the intervention had a modest effect on the variables above. The effect sizes for systolic blood pressure, diastolic blood pressure, waist circumference and body fat percentage were small. Fasting blood glucose had an overlap of 61.7%. Systolic blood pressure had an overlap of between 54% to 58%. Diastolic blood pressure had an overlap of 58% and lastly, dyspnoea had an overlap of 81.2%. The variables (systolic blood pressure, diastolic blood pressure, waist circumference and body fat percentage) above had 0.2 effect size with a non-overlap of 15%, this means that the intervention had a weak effect on the variables above.

Hassapidou *et al.* (2013) looked at the association of physical activity and sedentary lifestyle patterns with obesity and cardiometabolic comorbidities in Greek adults. Result of their study indicated that watching TV leads to harmful habits such as passive snacking and a lust for sugar-sweetened drinks and energy foods. Franssen *et al.* (2016) noted that there is an association of smoking with an unhealthy diet, which is linked to educational level and in those from a lower socio-economic status. Although, the current study did not investigate the association of unhealthy diet, it is worth acknowledging that such factors exist. It is assumed that participants in the control group are not immune to such factors, which, could be an explanation for their results.

In healthy individuals, normal fasting blood glucose concentration is kept in the range of about 3.5-5.4 mmol·L⁻¹ (Güemes, Rahman, & Hussain, 2016). Results of the current study show that fasting blood glucose levels increased, systolic blood pressure increased, diastolic blood pressure decreased and dyspnoea decreased in the control group over the 12-week period. The results of the current study show that even though systolic blood pressure increased post-intervention in the control group, it was still within the normal range of <80/<120 mmHg, this is in line with literature (Güemes *et al.*, 2016).

5.4 Within group comparisons: Intervention group

After a 12-week aerobic exercise intervention, body mass index decreased significantly in the participants who were in the intervention group (mean difference of -0.66). Donnelly *et al.* (2013) noted in their study that a supervised exercise programme has sufficient improvements in weight loss compared with observed behavioral weight loss. Wong *et al.* (2008) conducted a study on obese adolescents with Down Syndrome for 12 weeks and found that there was a significant reduction ($p<0.05$) in BMI in the exercise group after the training programme. Furthermore, there was a significant weight increase in the control group ($p<0.05$).

The effect sizes for the anthropometric variables in the intervention group between pre and post intervention were small after the intervention. This means that there was a 50% overlap in the mean score of their standard deviations. There was a decrease in the anthropometric variables of the participants that were in the intervention programme. This supports the hypothesis which states that there will be a decrease in body fat percentage after the intervention programme. Fasting blood glucose decreased from pre to post-intervention in this study, by 7.1%, for the intervention group, but the decrease, although large, was not statistically significant. It is known that fasting blood glucose levels should decrease with an aerobic exercise programme because of the “double dose” of medication and exercise (Boule *et al.*, 2001; Boule *et al.*, 2005). The effect size for fasting blood glucose was 0.45 with an overlap of 65.4%. The effect size for fasting blood glucose was medium. The hypothesis that said fasting blood glucose levels would decrease after the 12-week aerobic programme is supported.

Aerobic exercise requires the use of muscles and muscles use glucose to produce ATP (for energy transfer). The more an individual uses the larger muscle groups during aerobic exercise, the higher the uptake of glucose from the blood will be by the muscles. Aerobic training

programmes along with routine medical management are more effective treatments in the management of fasting blood glucose levels, glycaemic control, plasma insulin levels and insulin resistance compared with routine medical management and a dietary plan in the management of T2DM (Shakil-ur-Rehman *et al.*, 2017).

Ryun Kwon *et al.* (2011) reported a decrease in the HbA1c levels of the intervention group that were involved in the aerobic training programme in their study. They noted that aerobic exercise was more beneficial than resistance exercise for improving endothelial function in T2DM. In addition, aerobic capacity was identified as a better predictor of changes in (Flow-Mediated Dilation) FMD than body weight and glycaemic control. Another study by Behboudi, Azarbayjani, Aghaalinejad & Salavati (2011) that focused on the effect of an 8-week aerobic exercise and whole-body vibration in male diabetic patients, found that the aerobic exercise group had a significant decrease in fasting glucose levels compared with the control group ($p=0.02$).

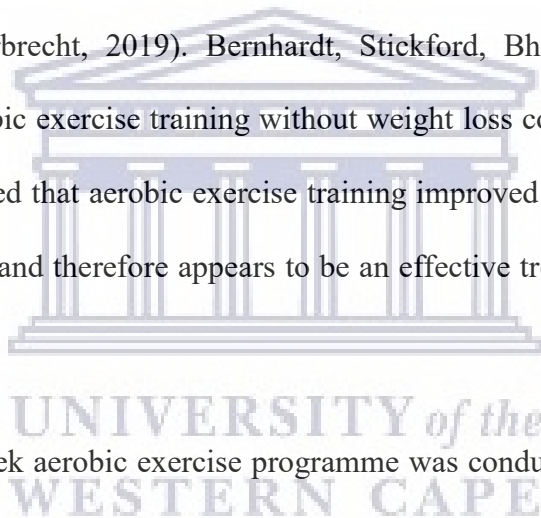
In the current study, systolic blood pressure increased post-aerobic intervention with a mean difference of -1.38, diastolic blood pressure also increased with a mean difference of 2.23, however both systolic and diastolic blood pressure were still within the normal range pre-and-post intervention (<120mmHg/<80mmHg). Systolic blood pressure had an effect size of 0.16, which is considered to be small with a 54% overlap and diastolic blood pressure had an effect size of 0.35, which is also considered to be small with an overlap of 61.7%. This means that the blood pressure readings of the participants remained within the normal range before and after the intervention programme.

The findings of the current study are contrary to the findings in a study by Kazeminia *et al.* (2020) who recorded a decrease in their participants for the same variables. However, Kazeminia *et al.* (2020) 's study focused on participants that were hypertensive. Despite the

increase in systolic and diastolic blood pressure in the current study, it remained within the normal range.

Dyspnoea ratings decreased significantly ($p < 0.01$) after the intervention programme with a mean difference in scores of 1.61. The effect size for dyspnoea was 2.38, which is considered to be large (Cohen.,1988), with a 92% overlap. The difference between the mean scores of the standard deviation post intervention is large. Therefore, we accept the hypothesis that dyspnoea during exertion would improve after the 12-week aerobic programme. The decrease in dyspnoea is due to the pulmonary adaptations that occur as a result of the aerobic training programme, including decreased breathing rate and a greater tidal volume (Luce., 1980; Kaeuffer, Ledoux & Herbrecht, 2019). Bernhardt, Stickford, Bhammar & Babb (2016) investigated whether aerobic exercise training without weight loss could reduce dyspnoea on exertion and they concluded that aerobic exercise training improved cardiorespiratory fitness and dyspnoea on exertion and therefore appears to be an effective treatment for dyspnoea on exertion in obese women.

In another study, a six-week aerobic exercise programme was conducted on individuals with chronic obstructive pulmonary disease (Borghesi-Silva *et al.*, 2009). The results of their study suggest that, at comparable submaximal exercise intensity, peak dyspnoea and perceived exertion were significantly reduced following aerobic training. No significant differences in any variables were observed in control group. The similar results of this study would support this finding. Stoner *et al.* (2016) reviewed data from 13 articles (15 trials) involving 556 participants (176 male, 193 female) and conducted a meta-analysis. They noticed that exercise interventions in general reduced BMI by $2.0 \text{ kg}\cdot\text{m}^{-2}$, body weight by 3.7 kg, body fat percentage by 3.1%, waist circumference by 3.0 cm, blood glucose by $\text{mmol}\cdot\text{L}^{-1}$ and systolic blood pressure by 7.1 mmHg.



5.5 Summary

In comparing the dependent variables between control and intervention groups, the findings of the current study indicate that the exercise intervention was a success, given the statistically significant changes which occurred in the intervention group. It is justified to state that the aerobic exercise training programme is an accepted therapeutic strategy in management of T2DM and obesity because of its shown beneficial effects. The results from the current study have been aligned to the available literature, which is encouraging for future studies with this population.



CHAPTER 6

Conclusion, Limitations and Recommendations for future research

6.1 Conclusions

Obesity is considered to be a major risk factor for many chronic diseases, but specifically for cardiovascular disease and diabetes (World Health Organization, 2018). Furthermore, obesity is linked with numerous health problems, which include T2DM, hypertension, stroke and heart attacks, sleep disordered breathing, and respiratory disorders (Jagsi *et al.*, 2017). However, studies have reported that regular physical activity can prevent or delay the onset of diabetes and its complications (Balducci *et al.*, 2006; Cohen *et al.*, 2008; Ghosh *et al.*, 2009). Yet, knowing this, most people with T2DM are still not active (Morrato, Hill, Wyatt, Ghushchyan & Sullivan, 2006).

This study aimed at investigating the effects of a 12-week aerobic exercise training programme on insulin resistant, obese female employees at a university in the Western Cape. It was hypothesised that a 12-week aerobic exercise programme would reduce the percentage of body fat, blood glucose and dyspnoea ratings in insulin resistant obese female university employees. The results of the study show support for all hypotheses stated, and to the current body of knowledge and literature. Therefore, it can be concluded that the implementation of an aerobic exercise programme will provide numerous health benefits in people with insulin resistance – exercise is medicine in this case.

6.2 Limitations

- These study findings cannot be generalised to the wider university staff in South Africa. It is recommended that future studies strive to obtain a broadly representative sample across South Africa in order to generalise the findings for future exercise intervention studies.
- Effort should be made when planning exercise intervention programmes to consider the barriers to physical activity so as to minimise dropout, most especially in university staff.
- Time constraints and family of the participants was seen as a barrier, which needs to be considered in future studies.
- The participants were not put on a structured diet plan, this should be considered in future studies for much clearer results.

6.3 Recommendations for Future Research

Future studies could further explore the limitations that were observed in this study, which, affected the staff participation rates, with, a view to reducing them. Potentially draw a representative sample across South Africa in order to obtain representative data for exercise interventions is a necessity. Dropout rate could be minimised by finding a means to educate people on the benefits to be gained through participating in physical activity intervention programmes. Once the population takes responsibility for their own health, compliance will be increased in intervention studies. In this study, the participants were not required to follow a specific diet plan, which might have delayed the improvement in weight-loss and glucose tolerance. Participants should be put on a healthy diet in future studies in order to achieve better results after the intervention programme.

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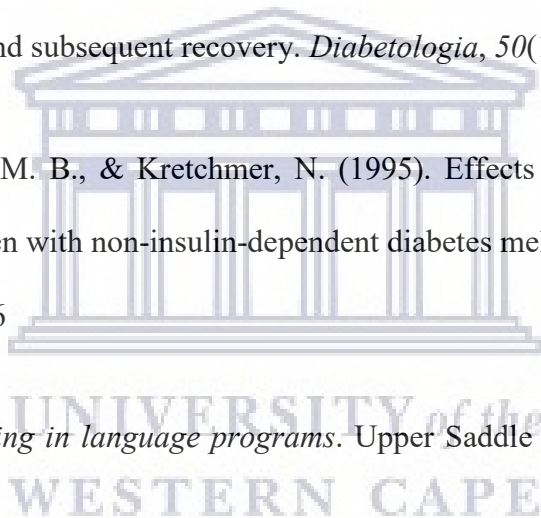
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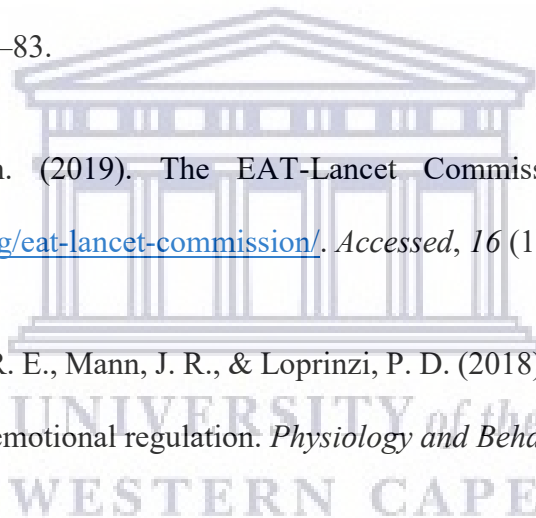
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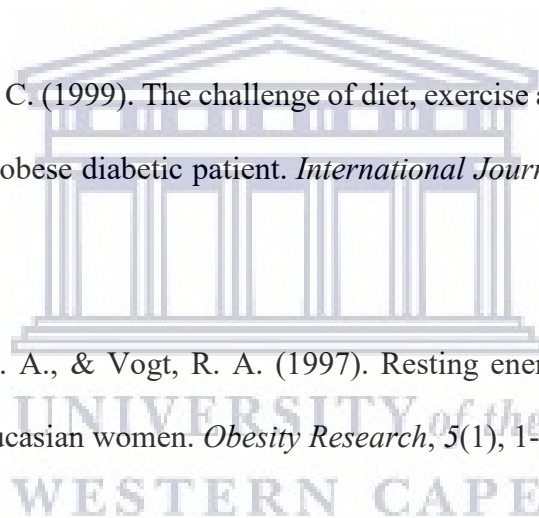
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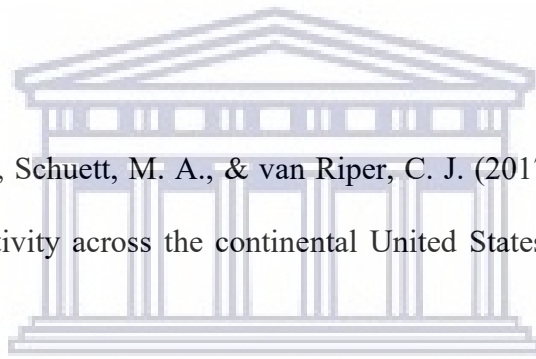
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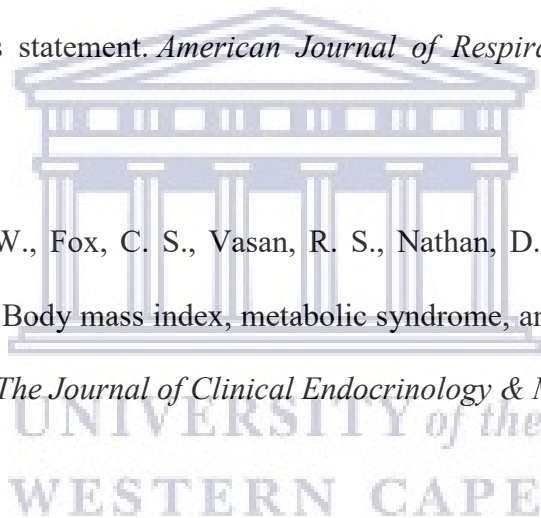
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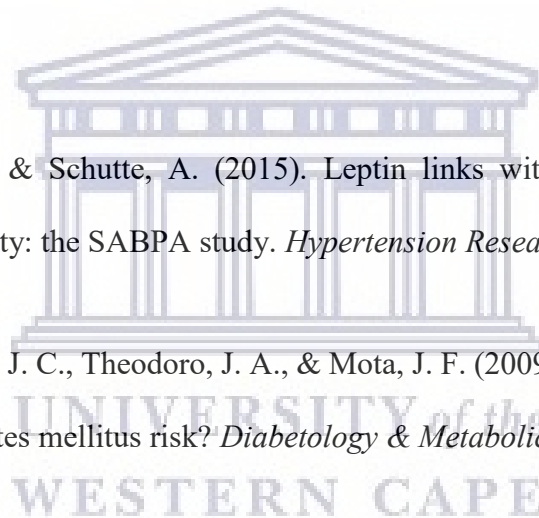
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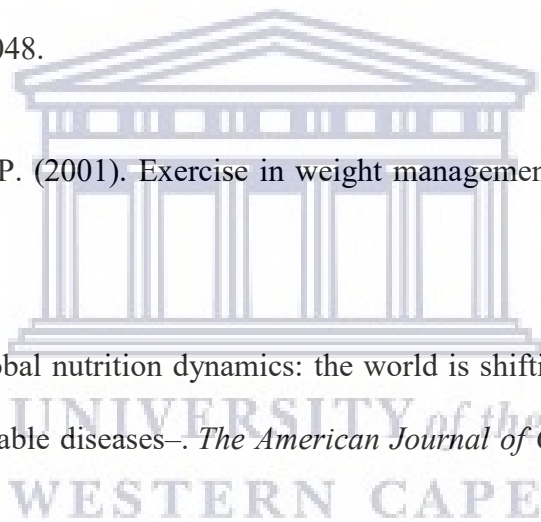
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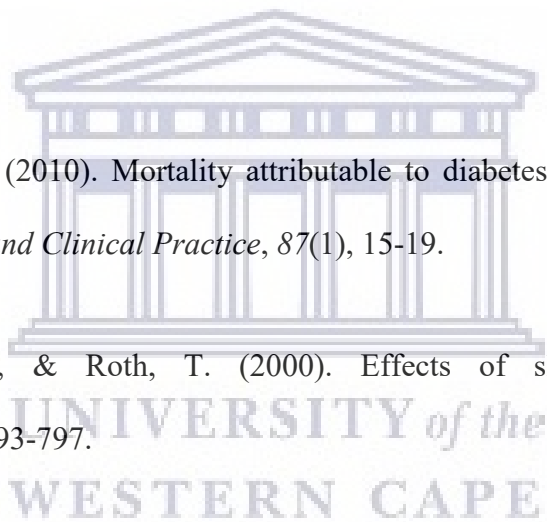
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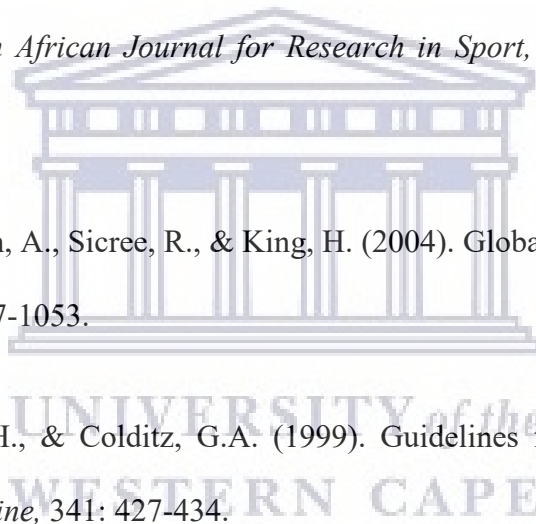
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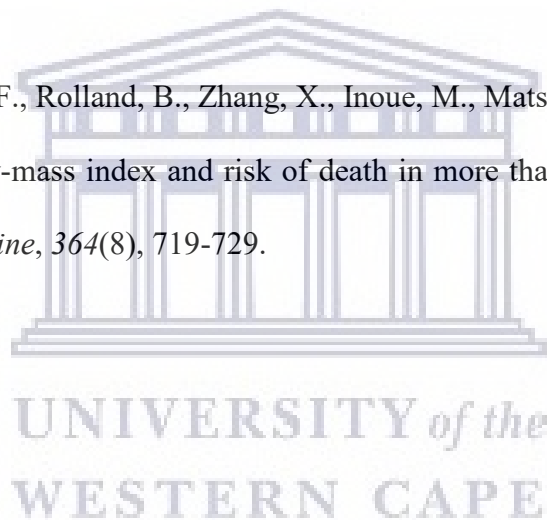
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APPENDIX A: INFORMATION SHEET

Project Title: Effect of a 12-week aerobic exercise programme on percentage body fat, fasting blood glucose and dyspnoea in insulin resistant, obese female university employees in the Western Cape.

What is this study about?

This is a research project being conducted by Maphoko Phindile Malema at the University of the Western Cape. We are inviting you to participate in this research project because you meet the study's inclusion criteria.

The purpose of this research project is to evaluate the effect of a 12-week aerobic training programme on percentage body fat, fasting blood glucose and dyspnoea in obese females in the Western Cape who are insulin resistant.

This study will add to the current body of knowledge and literature, furthermore, this study will lay down a foundation for South Africa stakeholders and health care practitioners to implement this programme and effectively get the country moving through Physical Activity.

What will I be asked to do if I agree to participate?

You will be asked to participate in a 12-week aerobic training programme for three days a week throughout the 12 weeks; the study will take place at the UWC Biokinetics clinic.

Blood glucose and blood pressure will be assessed before and after every training session for safety reasons of this study. Each training session will take 30-45 minutes long.

Would my participation in this study be kept confidential?

To ensure your anonymity, the researchers undertake to protect your identity and the nature of your contribution. To ensure this, the names of the participants will not be revealed at any time and a participant ID code will be allocated by the research supervisor from the consent forms, so all subsequent forms will only have their ID code on them. No names will be used on any publication emanating from this research. All information about the participants will be kept confidential with the data forms being kept in the supervisor's office in a locked cabinet and/or on a password protected computer. If we write a report or article about this research project, your identity will also be protected through never using the participants' names.

What are the risks of this research?

There may be some risks from participating in this research study. Before exercising, blood glucose levels will be measured in order to ensure that it is safe to continue with the exercise. After exercise, blood glucose and blood pressure will be measured to ensure that it is safe for the participant to leave the exercise venue. In terms of thermoregulation, the participants will



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exercise in an air-conditioned environment to prevent any possibility of heat illnesses in the summer, when data collection is expected to take place. Should the participants experience any signs and symptoms of heat illnesses, all activity will stop and the researcher (a qualified first aider) will provide immediate assistance.

Safety of the environment

Participants will be exercising in a safe environment free of loose weights and obstacles that might cause injuries. The risk of injury will be minimized by providing an adequate warm up before each exercise session (brisk walking and dynamic stretches) as well as a cool down involving slow walking and static stretches. If a participant does sustain an injury, the researcher will provide immediate first aid and the participant will be referred to an appropriate medical professional, or the campus emergency system will be activated. All human interactions and talking about self or others carry some amount of risks. We will nevertheless minimise such risks and act promptly to assist you if you experience any discomfort, psychological or otherwise during the process of your participation in this study. Where necessary, an appropriate referral will be made to a suitable professional for further assistance or intervention.

What are the benefits of this research?

The benefits to you include potential weight-loss, improved fasting blood glucose levels and lower dyspnoea. Although, this research is not designed to help you personally, the results may help the investigator learn more about exercise programmes for people with insulin resistance who are obese. We hope that, in the future, other people might benefit from this study through improved understanding of exercise aimed to decrease blood glucose, improve glucose sensitivity, and enhance weight loss.

Do I have to be in this research and may I stop participating at any time?

Your participation in this research is completely voluntary. You may choose not to take part at all. If you decide to participate in this research, you may stop participating at any time. If you decide not to participate in this study or if you stop participating at any time, you will not be penalized or lose any benefits to which you otherwise qualify.

What if I have questions?

This research is being conducted by Ms Phindile Phatlane, Department of Sport, Recreation and Exercise Science at the University of the Western Cape. If you have any questions about the research study itself, please contact Ms Phatlane at: phindilephatlane@gmail.com or 063 746 4656.

Should you have any questions regarding this study and your rights as a research participant or if you wish to report any problems you have experienced related to the study, please contact:



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This research has been approved by the University of the Western Cape's Biomedical Research Ethics Committee (REFERENCE NUMBER: BM17/8/11).

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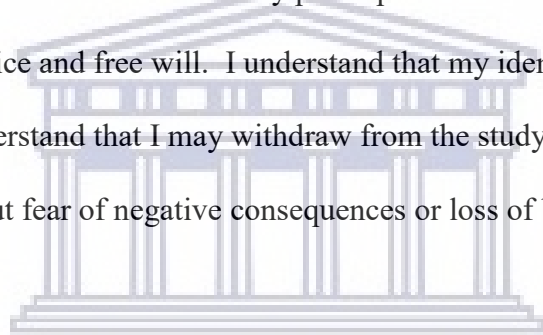
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APPENDIX B: CONSENT FORM

Title of Research Project: Effect of a 12-week aerobic exercise programme on percentage body fat, fasting blood glucose and dyspnoea in insulin resistant, obese female university employees in the Western Cape.

The study has been described to me in language that I understand. My questions about the study have been answered. I understand what my participation will involve and I agree to participate of my own choice and free will. I understand that my identity will not be disclosed to anyone. I understand that I may withdraw from the study at any time without giving a reason and without fear of negative consequences or loss of benefits.



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Participant's name.....

Participant's signature.....

Date.....

APPENDIX C: INTERVENTION TRAINING PROGRAMME

Exercise programme

Frequency	Intensity	Type	Time
3 DAYS A WEEK	Moderate (40% - 60% HRR)	Aerobic Exercises	30 minutes

Exercise Type	Exercises	Frequency	Intensity	Duration
Warm-up	-Brisk Walking	Before every session		5 minutes
Dynamic Stretches	-Arm swings (dynamic shoulder flexion, extension, circumduction etc.) -Leg swings (dynamic hip abduction, adduction, extension and flexion) -Hip rotations -Shoulder shrugs and rotations	Every session	-Prepare the body muscles for exercise and minimize the risk of injury.	5 minutes
Aerobic exercises	-Elliptical machine -Treadmill -Cycle ergometer -Stepper	Every session	40 – 60% HRR	7minutes 7minutes 7minutes 7minutes
Cool-down	-Cycling on a recumbent bike	Post exercise		5 minutes
Static stretches	-Pigeon -Cat stretch -Hamstrings stretch -Quadriceps stretch -Piriformis stretch -Glut medius stretch -Calf stretch -Soleus stretch -Triceps stretch	Post exercise	Stretch muscle beyond the normal range of motion.	5 minutes



APPENDIX D: ETHICS CLEARANCE

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12 April 2018

Ms MP Phatlane
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Science Ethics Reference Number:

BM17/8/11

Project Title: Effect of a 12-week aerobic exercise programme on percentage body fat, fasting blood glucose and dyspnoea in insulin resistant, obese female university employees in the Western Cape.

Approval Period: 09 April 2018 – 09 April 2019

I hereby certify that the Biomedical Science Research Ethics Committee of the University of the Western Cape approved the scientific methodology and ethics of the above mentioned research project.

Any amendments, extension or other modifications to the protocol must be submitted to the Ethics Committee for approval.

Please remember to submit a progress report in good time for annual renewal.

The Committee must be informed of any serious adverse event and/or termination of the study.

A handwritten signature in black ink, appearing to read 'Josias'.

*Ms Patricia Josias
Research Ethics Committee
Officer, University of the
Western Cape*

PROVISIONAL REC NUMBER -130416-050