The effect of an 8-week combined aerobic and suspension training programme on physical, performance, cardiovascular, and metabolic characteristics of overweight and obese inactive individuals

Master of Science (MSc) By Research

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Abstract

Title: The effect of an 8-week combined aerobic and suspension training programme on physical, performance, cardiovascular, and metabolic characteristics of overweight and obese inactive individuals.

Background: Overweight and obesity are serious global problems with 1.9 billion adults classified as overweight or obese in 2016 (World Health Organization, 2017). They are complex conditions and are associated with severe physical and metabolic health implications including cardiovascular disease, type II diabetes mellitus and premature mortality. In order to combat overweight and obesity and reduce the risk of associated non-communicable diseases there is a need to develop effective exercise interventions. Aerobic training and suspension training have been shown to be effective in treating obesity. However, no previous research has investigated the combination of aerobic and suspension training in one intervention. Therefore the aim of this study was to investigate the effect of an 8-week combined aerobic and suspension training programme on physical, performance, cardiovascular, and metabolic characteristics, in overweight and obese inactive individuals.

Methods: Eighteen (N = 2 male, N = 16 female) untrained, overweight and obese participants (age: 37 ± 11 yr, BMI: 30.3 ± 5.8 kg/m^2) participated in the study. Baseline physical (height, weight, percentage body fat (%BF) and body circumferences), performance (muscular strength, 20m sprint, timed up and go, plank time trial, and VO$_{2max}$), cardiovascular (high density lipoprotein cholesterol, low density lipoprotein cholesterol, total cholesterol, and triglycerides), and metabolic measurements (fasting blood glucose, and fasting insulin) were measured. The novel biomarker, myostatin was also recorded. An 8-week exercise intervention was then completed. Two 60min aerobic sessions, involving walking/jogging at intensities of 65-80% VO$_{2max}$, and two 60min suspension training sessions, involving a circuit of 10 exercises, performed 4 times with a 45s:15s work to rest ratio, were completed weekly. All baseline tests were repeated immediately following the 8-week intervention.

Results: In addition to significant improvements in BMI, body weight and %BF following the intervention, significant improvements were observed in all performance measures. There were also significant improvements in all cardiovascular and metabolic health markers except fasting insulin. Improvements in percentage body fat and 20m sprint were significantly correlated with fasted blood glucose. In addition, regression analysis indicated that improvements in body composition and performance measures explained the improvements in cardiovascular and metabolic health.

Conclusion: The novel aerobic and suspension training intervention was effective at improving physical, performance, cardiovascular, and metabolic characteristics of overweight and obese sedentary individuals. It is possible that the novel combination of the two exercise modalities in one intervention elicits improvements across a wider spectrum of physical, performance, and cardiovascular and metabolic factors compared to interventions involving just one training mode. Such novel training interventions have the potential to improve metabolic and cardiovascular health and reduce the incidence of overweight and obesity.

Declaration:

I hereby declare that this project is entirely the result of my own investigation and that appropriate credit has been given where reference has been made to the work of others. This work has not been submitted for any academic award, or part thereof, at this or any other establishment.

Signed: Date: 26/09/19

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Glossary of Terms

Aerobic Exercise - physical exercise that depends primarily on the aerobic metabolism process to meet energy demands.

Body Mass Index – a measure of weight status, calculated by dividing a person's weight in kilograms by the square of their height in metres.

Cardiovascular Disease - a class of diseases that involve the heart or blood vessels.

Cardiovascular Health – relates to health of the circulatory system, which comprises the heart and blood vessels.

Concentric Muscle Contraction - a muscle contraction in which the muscle is shortening under tension.

Eccentric Muscle Contraction - a muscle contraction in which the muscle is lengthening under tension.

Insulin Resistance - when insulin level produces a decreased biological effect on the body.

Maximal Oxygen Uptake (VO$_{2max}$) - the maximal amount of oxygen an individual can take up, transport and use at a cellular level.

Metabolic Health – health related to the anabolism and catabolism of molecules required for energy in the body.

Overweight and Obesity - abnormal or excessive fat accumulation that may impair health.

Physical Activity - any movement caused by skeletal muscles that results expended energy, which can be quantified in kilocalories.

Resistance Training - any exercise that causes the muscles to contract against an external resistance.

Sedentary Behaviour - any waking behaviour characterized by an energy expenditure ≤1.5 metabolic equivalents while in a sitting, reclining or lying posture.

Suspension Training - when the user utilises a set of ropes or webbing suspended from an overhead anchor in order to exercise against their own body weight.

List of Abbreviations

$300°.s^{-1}$ KEPT = $300°.s^{-1}$ knee extension peak torque

$300°.s^{-1}$ KETW = $300°.s^{-1}$ knee extension total work

$300°.s^{-1}$ KFPT = $300°.s^{-1}$ flexion peak torque

$300°.s^{-1}$ KFTW = $300°.s^{-1}$ knee flexion total work

3RM = 3 Repetition Maximum Strength Test

$60°.s^{-1}$ KEPT = $60°.s^{-1}$ knee extension peak torque

$60°.s^{-1}$ KETW = $60°.s^{-1}$ knee extension total work

$60°.s^{-1}$ KFTW = $60°.s^{-1}$ knee flexion total work

$60°.s^{-1}$ KEFT = $60°.s^{-1}$ knee flexion peak torque

BMI = Body mass index

CHD = Coronary heart disease

CVD = Cardiovascular disease

DBP = Diastolic blood pressure

FBG = Fasting blood glucose

HDL = High density lipoprotein cholesterol

LDL = Low density lipoprotein cholesterol

PA = Physical activity

RHR = Resting heart rate

SBP = Systolic blood pressure

T2DM = Type II diabetes mellitus

TC = Total cholesterol

TG = Triglycerides

TUG = Timed up and go

VO_{2max} = Maximal oxygen uptake

WC = Waist circumference

WHR = Waist hip ratio

Chapter 1: Introduction

1.1 Introduction.

The worldwide prevalence of obesity nearly tripled between 1975 and 2016 with more than 1.9 billion adults classified as either overweight or obese in 2016 (39% of men and 40% of women) (World Health Organisation, 2017). In an EU context, Ireland ranks above the EU average in terms of overweight and obesity with 62% of people overweight or obese in 2017 (Central Statistics Office, 2018).

Obesity is associated with many negative health implications and an increased risk of developing comorbidities such as insulin resistance, type II diabetes mellitus (T2DM), and cardiovascular disease (CVD), which in turn increases the risk of mortality (De Koning et al., 2010; Kitahara et al., 2014; Jung and Choi, 2014; Hall et al., 2014; De Koning et al., 2007). All of these conditions result in a large economic burden on the individual, families and nations and are associated with both direct costs, including in-patient and day patient costs, out-patient costs, GP costs and drug costs and indirect costs including productivity losses associated with work absenteeism and premature mortality (Dee et al., 2012). In 2014 the global economic impact of obesity was estimated to be two trillion US dollars or 2.8% of the global gross domestic product (Dobbs et al., 2014). In Ireland the estimated total costs attributable to obesity in 2009 were 1.13 billion euro, with 399 million euro attributed to direct costs and 729 million euro attributed to indirect costs (Dee et al., 2012).

Overweight and obesity can be defined as abnormal or excessive fat accumulation that may impair health (World Health Organisation, 2017). This excessive fat accumulation results from an imbalance between calorie intake, and caloric expenditure that results in a positive energy balance stored predominantly in adipose tissue as fat. Physical inactivity and dietary trends play a role in the development of obesity. Current global physical activity (PA) levels are inadequate with 31.1% of adults worldwide reported to be physically inactive (Hallal et al., 2012). In Ireland, in 2018 only 33% of adults achieved the PA levels recommended for health, set out by the American College of Sports Medicine (ACSM) (World Health Organization, 2018). Sedentary behaviour, defined as any waking behaviour characterised by an energy expenditure ≤1.5METs while in a sitting, reclining, or lying posture may also contribute to the global and national increasing prevalence of overweight and obesity (Van der Ploeg, and Hillsdon, M., 2017).

Matthews et al., (2016) investigated PA levels and sedentary behaviour of US adults and established that daily sedentary time was 8.2 hours. Six years post study, 700 deaths had occurred and compared with less-sedentary adults (adults with 6 sedentary hours per day), individuals with 10 sedentary hours per day had a 29% greater risk of death. Technological advances contribute toward this decrease in PA and development of new technology has enabled individuals to reduce the amount of PA required to complete tasks throughout their daily lives (Hallal et al., 2012). The quantity of calories consumed worldwide has vastly increased in recent decades also, influencing the increase of global obesity levels. Daily global calorie consumption for adults has risen from 2411 kcal in 1969, to 2950 kcal in 2015 (Kearney, 2010). The WHO does not have a recommended daily allowance for calories but states that in order to prevent weight gain and obesity, energy intake should be in balance with energy expenditure (World Health Organization, 2015). All of these factors result in weight gain which contributes towards significant health risks including cardiovascular and metabolic diseases. Compared with normal-weight adults, obese adults have at least a 20% significantly higher risk of dying of all-causes (Stiano et al., 2015).

1.2 Statement of problem

Various therapies are used in the treatment and management of obesity. The challenge that exists is to determine the most effective form of therapy, or combination of therapies. One such therapy is exercise, with aerobic exercise and resistance training being utilised extensively. Aerobic exercise, any activity that uses large muscle groups, can be maintained continuously and is rhythmic in nature (Wahid et al., 2016), has been shown to be effective in terms of weight loss (Donnelly et al., 2013; Lucotti et al., 2011). The energy expenditure associated with aerobic exercises contributes to this weight loss. In addition to weight loss, other favourable adaptations of aerobic training include increases in cardiovascular fitness (Bassett and Howley, 2000), which is an independent predictor of health (Myers et al., 2015), improvements in mitochondrial size, number, and function (Miller et al., 2011), increased activity of oxidative enzymes (Maltais et al., 1996), reductions in hypertension (Franz et al., 2015), increased blood glucose utilisation (Christ-Roberts et al., 2004), improvements in insulin sensitivity (Jenkins et al., 2011), and improved blood lipid parameters (Leon and Sanchez, 2001). These improvements

reduce the risk of developing conditions such as T2DM, CVD, insulin resistance, and mortality (Christ-Roberts et al., 2004; Ference et al., 2017; Jenkins et al., 2011).

Resistance training is any exercise that causes the muscles to contract against an external resistance. In addition to improving muscle mass, strength, and inducing muscular hypertrophy (Schoenfeld, 2010), other benefits of resistance training include improved insulin sensitivity and glucose tolerance (Sigal et al., 2007). Skeletal muscle mass is highly correlated with basal metabolic rate, which means the obese individual who participates in resistance training would burn more calories at rest, potentially improving body composition (Vaughn, 2013). Suspension training is a novel form of resistance training which has emerged in recent years. It appears to elicit similar physiological adaptations as regular resistance training, although studies published in this area are currently limited (Dolati et al., 2017; Smith et al., 2016; Janot et al., 2013). Suspension training involves the use of ropes or webbing suspended from an overhead anchor in order to exercise against a person's own body weight. It provides a greater range of motion than traditional resistance training, which provides a greater distance by which muscles can lengthen (eccentric contraction) and shorten under tension (concentric contraction), which could potentiate greater muscular hypertrophy (Schoenfeld, 2016). Compared with traditional resistance training, suspension training may elicit increased muscle activation (Calatayud et al., 2014; Byrne et al., 2014; Snarr at al., 2013; Snarr and Esco 2013; Mok et al., 2015), potentially leading to greater levels of strength and hypertrophy of selected muscle groups. In addition to increased motor unit recruitment, there is an increased emphasis on eccentric contractions. This increases muscle damage thus increasing energy expenditure post training, which may in turn facilitate more favourable improvements in body composition when compared to regular resistance training. Resting energy expenditure has been shown to be increased up to 72 hours after traditional resistance training involving an eccentric component (Hacknet et al., 2008). Due to the eccentric nature of suspension training exercise it is possible that resting energy expenditure would be increased following suspension training bouts also, perhaps to a higher degree when compared with traditional resistance training exercise.

As a result of the novel nature of suspension training, there are few studies that document the chronic benefits of this type of training on physical and metabolic health, particularly in obese individuals. Studies that have been performed note suspension training to be effective at improving metabolic (Smith et al., 2016), physical (Dolati et al., 2017), and cardiovascular health markers (Smith et al., 2016). The limited research that has been conducted to date has shown potential for increased energy expenditure and muscular damage post exercise, due to increased muscular activation (Mok et al., 2015; Snarr et al., 2014). No studies have yet examined a combined intervention of suspension training and aerobic training in the treatment and management of obesity. Likewise, no studies have examined the effect of this type of combined intervention on myostatin, a novel biomarker of insulin resistance. Given the potential benefits of both types of training, a combined intervention may be more effective in terms of improving physical, performance, cardiovascular and metabolic characteristics than previously examined exercise interventions. Examining the change in circulating concentration of novel biomarkers of insulin resistance in response to exercise interventions may also provide an important unique insight into the effect of the intervention on the metabolic health of overweight and obese individuals.

1.3 Aims

The aim of this thesis was to observe the effect of an 8-week combined aerobic and suspension training intervention on the physiological, physical performance, metabolic and cardiovascular characteristics, of overweight and obese inactive individuals.

1.4 Objectives

1. To examine the changes in physical characteristics of the participants post-intervention including changes in body mass index (BMI), body weight, body fat percentage, percentage muscle mass and body circumference measurements.

2. To assess the changes in physical performance characteristics of overweight and obese sedentary individuals including 20m sprint, 3 repetition maximum strength (3RM), isokinetic knee strength, maximum aerobic capacity (VO$_{2max}$) and plank time trial following the eight week combined intervention.

3. To determine the metabolic (fasting blood glucose (FBG) and fasting insulin), and cardiovascular (fasting lipid profile) adaptations to this novel intervention.

4. To determine the impact of this intervention on the circulating concentration of myostatin, a novel biomarker of insulin resistance.

5. To investigate the relationship between changes in physical and performance characteristics, with changes in metabolic and cardiovascular health characteristics post intervention.

6. To use regression analysis to determine the physical, performance, metabolic and/or cardiovascular variables that predict the improvements in cardiovascular and metabolic health post intervention.

1.5 Hypothesis

A combination of aerobic and suspension training will result in significant improvements in the physical, cardiovascular, and metabolic health of the participants including improvements in the circulating concentration of myostatin, a novel biomarker of insulin resistance

Chapter 2: Literature Review

2.1 Introduction

This chapter provides an overview of the available scientific literature examining obesity. Relevant literature of key areas pertaining to obesity and exercise is presented. Firstly, the causes, economic cost and prevalence of obesity are discussed. Secondly, the health consequences associated with obesity are critically examined. Lastly, current methods used to treat and manage obesity are critically appraised including surgical, dietary, and exercise interventions. The physiological and metabolic adaptations that occur in response to exercise interventions are examined in detail and the role of these adaptations in treating obesity and preventing obesity related comorbidities is also discussed.

2.1.1 Introduction to obesity

Obesity and overweight can be defined as having abnormal or excessive amounts of adipose tissue that may impair health (World Health Organisation, 2017). Body mass index is the most commonly used calculation to define and discuss overweight and obese adults. The BMI calculation consists of a person's weight in kilograms, divided by the square of their height in meters (kg/m^2). For adults, overweight and obesity are defined as a BMI greater than or equal to 25 kg/m^2; and greater than or equal to 30 kg/m^2 respectively (World Health Organization, 2017). Obesity can be further subdivided into obesity class I (30.0-34.9 kg/m^2), obesity class II (35.0-39.9 kg/m^2) and obesity class III (> 40 kg/m^2). Body mass index is an extensively utilised term when discussing body composition and obesity levels, due mainly to its simplicity, and it is a good indicator of health in inactive individuals (Yang et al., 2016). However, it does not take body composition into account, and instead considers the whole weight of the person regardless of muscle mass. Another measure by which obesity can be identified is that of body fat percentage. In general, a healthy total body fat percentage is between 12% and 15% for young men and between 25% and 28% for young women (Lohman and Going, 1993). Age related percentage body fat guidelines, for males and females, are provided by the American College of Sports Medicine (ACSM) and can be seen below in Table 2.1 and 2.2. The risk of certain diseases and health conditions such as CVD and T2DM dramatically increases as BMI and percentage body fat increases (De Koning et al., 2007; Nguyen et al., 2011).

Table 2.1: American College of Sports Medicine male body fat guidelines.

Percentile	Age (Male)				
	20-29	30-39	40-49	50-59	60+
90 (Excellent)	7.1%	11.3%	13.6%	15.3%	15.3%
80	9.4%	13.9%	16.3%	17.9%	18.4%
70 (Above average)	11.8%	15.9%	18.1%	19.8%	20.3%
60	14.1%	17.5%	19.6%	21.3%	22.0%
50 (Average)	15.9%	19.0%	21.1%	22.7%	23.5%
40	17.4%	20.5%	22.5%	24.1%	25.0%
30 (Below average)	19.5%	22.3%	24.1%	25.7%	26.7%
20	22.4%	24.2%	26.1%	27.5%	28.5%
10 (Poor)	25.9%	27. 3%	28.9%	30.3%	31.2%

(ACSM'S Health-Related Physical Fitness Assessment Manual, 2ndEd. 2008. pg 59

Table 2.2: American College of Sports Medicine female body fat guidelines.

Percentile	Age (Female)				
	20-29	30-39	40-49	50-59	60+
90 (Excellent)	14.5%	15.5%	18.5%	21.6%	21.1%
80	17.1%	18.0%	21.3%	25.0%	25.1%
70 (Above average)	19.0%	20.0%	23.5%	26.2%	27.5%
60	20.6%	21.6%	24.9%	28.5%	29.3%
50 (Average)	22.1%	23.1%	26.4%	30.1%	30.9%
40	23.7%	24.9%	28.1%	31.6%	32.5%
30 (Below average)	25.4%	27.0%	30.1%	33.5%	34.3%
20	27.7%	29.3%	32.1%	35.6%	36.6%
10 (Poor)	32.1%	32.8%	35.0%	37.9%	39.3%

(ACSM'S Health-Related Physical Fitness Assessment Manual, 2ndEd. 2008. pg 59).

2.2 Prevalence of obesity

The prevalence of obesity worldwide increased exponentially between 1975 and 2016. In 2016, 1.9 billion adults were either overweight or obese (39% of men and 40% of women), and of these over 650 million (11% of men and 15% of women) adults were obese (World Health Organization, 2017). When comparing past figures of obesity (in 1975 3% of men and 6% of women) versus statistics from 2016 (11% of men and 15% of women) it is clear to see the trend is escalating at an alarming rate (World Health Organization, 2017).

The United States of America (USA) has a particularly high prevalence of obesity. As of 2014, approximately 70% of the population over 20 years of age is overweight (Centre for Disease Control and Prevention, 2016). Flegal et al., (2016) found that for the years 2013-2014, the prevalence of obesity was 35.2% and 40.5% among men and women respectively, with class III obesity incidences being 5.5% and 9.9% for men and women, respectively. When compared to worldwide statistics, USA has a far higher incidence of obesity, however between the years of 2005 and 2014 the prevalence of overall obesity showed significant linear increases for women, but there were no increases for men (Flegal et al., 2016). Hales et al., (2018) noted overall obesity levels of 33.7% in 2008 rising significantly to 39.6% in 2015 ($P < 0.001$), showing overall obesity prevalence in the USA is high.

Within the European Union (EU), 51.6 % of the adult population was identified as being overweight in 2014, with no difference between genders (Eurostat, 2017). Several factors may contribute to this high prevalence of overweight individuals, and the Eurostat survey identified that the prevalence of overweight people in the EU has a positive correlation with age, and a negative correlation with education, with the proportion of overweight individuals falling as education level rises. Ireland ranks above the EU average in terms of overweight and obese men and women, with 69% and 52% being either overweight or obese, respectively. Of these, 26% of men and 21% of women are obese only (Irish Universities Nutrition Alliance, 2010). By the year 2030, the WHO predicts that 47% of both Irish men and women will be obese (World Health Organization, 2013).

2.3 Economic cost of obesity

Obesity incurs large economic burden on the individual, and on families and nations. In 2014 the global economic impact of obesity was estimated to be US $2.0 trillion or 2.8% of the global gross domestic product (Dobbs et al., 2014). This economic cost can consist of both direct and indirect measures, with direct costs involving hospital inpatient and outpatient visits, physician services, drug costs, health research and other health care directly related to obesity. Indirect costs consist of work absenteeism, early retirement and premature death relating to co-morbidities associated with obesity (Dee et al., 2014). When dividing economic cost by country, Anis et al., (2010) noted direct costs of $5.9 billion, and indirect costs of $5 billion in Canada, Finkelstein et al., (2010) noted $30.3 billion direct costs, and $42.8 billion indirect costs in the USA, and Konnopka et al., (2011) noted €4.8 billion direct costs, and €5 billion indirect costs in Germany. The differences between these studies lies in the definitions of direct and indirect costs with Anis et al., (2010) defining indirect costs as morbidity due to both long and short-term disability, Dinkelstein et al., (2010) as absence from work, and Konnofka et al., (2011) as sickness absence, early retirement and mortality. Direct costs were defined similarly between studies and involved inpatient and outpatient treatment, rehabilitation and non-medical costs (administration and research) (Anis et al., 2010; Dinkelstein et al., 2010; Konnofka et al., 2011)

Obesity in Ireland also incurs substantial costs. In Ireland the estimated total costs attributable to obesity in 2009 were €1.13 billion, and of this, €399 million were direct and €729 million were indirect costs (Dee et al., 2012). Direct costs in this study referred to in-patient and day patient costs, out-patient costs, GP costs and drug costs, while indirect costs referred to productivity losses associated with work absenteeism and premature mortality. In Ireland the hospital discharge frequency of obesity related conditions such as T2DM, CVD, and dyslipidemia increased significantly between 1997 and 2004 for both adults and children, with the relative length of stay (number of days in care for obesity related conditions per 1000 days of hospital care given) increasing from 1.47 days in 1997 to 4.16 days in 2004 for children and from 3.68 days in 1997 to 6.74 days in 2004 for adults (Vellinga et al., 2008). This contributed to annual hospital

costs of €4.4 million in 1997, increasing to €13.3 million in 2004, based on the 2001 figures for cost per inpatient bed per day (Vellinga et al., 2008).

2.4 Causes of obesity

Obesity occurs due to an imbalance between calorie intake and caloric expenditure that results in a positive energy balance. This positive energy balance is stored in adipose tissue and over time, body fat levels and body weight rise into the overweight and obese categories. Physical inactivity, dietary trends, and genetics can also influence the development of obesity.

2.4.1 Physical inactivity

Physical activity is defined as any movement caused by skeletal muscles that results in expended energy, which can be quantified in kilocalories and can result from various contributors in daily life including occupational, exercise, or household activities (Casperson et al., 1985). Exercise is a subsection of PA that is planned, structured and also involves the objective of improving or maintaining physical attributes such as strength or fitness (Casperson et al., 1985). Insufficient PA can contribute toward obesity as PA is necessary for caloric expenditure, and the current global PA levels are inadequate (Hallal et al, 2012). The current PA criteria recommended for health benefits from the WHO involves 150 minutes of moderate to vigorous intensity exercises weekly (Garber et al., 2011). Globally in 2016 27.5% of adults were not obtaining the recommended level of PA and were insufficiently physically active, improving marginally from 28.5% in 2001 (World Health Organization, 2019).

Among European countries PA levels vary substantially. Gerovasili et al., (2016) examined PA levels in 28 EU countries and reported that 28.6% of individuals were physically inactive. Of the participants surveyed 59.1% were categorised as highly active, which equates to achieving at least 300 minutes of moderate intensity PA (World Health Organization, 2010). Physical activity showed huge variability between countries with high incidences of inactive individuals noted in Cyprus (53.7%), Portugal (50.6%) and Malta (48.7%). However the incidences were much lower in other countries such as Sweden (12.4%), the Netherlands (14.9%), and Finland (15.9%). The highest incidences of highly active population occurred in Sweden (72.2%), Germany (71.5%) and Latvia

(71.4%). A systematic literature review by Loyen et al., (2016) further supports this variability between PA levels in European countries. Loyen et al., (2016) noted the lowest percentages of participants meeting the recommended PA levels (7% in males and 9% in females) were found in Georgia, while the highest percentage (96%) was found in Ukrainian females and Estonian males. Likewise, the same study noted the time spent in moderate to vigorous PA, which ranged from 45 minutes per week in Malta to 960 minutes per week in the Netherlands.

In Ireland PA data including the type, duration, intensity and frequency of exercise carried out by Irish people is collected and collated by The Irish Sports Monitor (ISM), a subsection of the Irish Sports Council. According to the ISM in 2013 only 31.3% of Irish adults met the ACSM criteria for PA levels for health (Irish Sports Council, 2015). The Irish national recommendation for PA levels for health is consistent with both the EU PA guidelines, and with the ACSMs recommended PA guidelines of 150 minutes of moderate to vigorous intensity exercise weekly (Garber et al., 2011).

Irish children are also not sufficiently active with only 19% of primary and 12% of post-primary school children meeting PA recommendations (60 minutes of moderate to vigorous PA daily (Woods et al., 2010). The 2016 Ireland North and South Report Card on Physical Activity for Children and Youth summarised available PA data pertaining to children and youth in Ireland and reported that 12% to 31% of children were meeting the recommended PA guidelines (Harrington et al., 2016). Likewise, only 34% of Irish adults over 50 years of age perform high levels of PA as quantified by The International Physical Activity Questionnaire (Barrett, et al 2011). Data from the Irish Sports Council, (2015), Woods et al., (2010) and Barrett et al., (2011), highlight that inactivity spans all age groups in Ireland. Due to the current levels of physical inactivity in Ireland a national PA plan "Get Ireland Active" was introduced in 2016, the aim of which is to increase the proportion of each age group participating in the required amount of PA each week by 1%. The plan also aimed at reducing the proportion of people in each age group participating in no PA each week by 0.5% (Get Ireland Active, 2016).

Sedentary behaviour, defined as any waking behaviour characterised by an energy expenditure ≤1.5 metabolic equivalents while in a sitting, reclining or lying posture, may

also contribute to the increasing prevalence of overweight and obesity (Van der Ploeg, and Hillsdon, M., 2017). Elevated sedentary time has been associated with a 112% increase in the relative risk of T2DM, a 147% increase in the relative risk of cardiovascular events, a 90% increase in the risk of cardiovascular mortality, and a 49% increase in the risk of all-cause mortality (Wilmot et al., 2012). Increasing amounts of time are being spent in sedentary environments such as sitting at work, at home, and in cars (Connor et al., 2017; Church et al., 2012). Technological advances contribute toward this decrease in PA and development of new technology has enabled individuals to reduce the amount of PA required to complete tasks throughout their daily lives (Hallal et al., 2012). Total screen time (use of computers, television, and video games) has also increased substantially in recent years (Connor et al., 2017). Connor et al., (2017) investigated sedentary behaviour in adults and noted a mean screen time of 14.8 ± 11.6 hours per week, accounting for 25% of total sedentary time (59.0 ± 25.8 hours per week). Screen time has also been positively associated with parameters such as BMI ($R^2 = 0.19$, $P < 0.05$), and waist circumference ($R^2 = 0.19$, $P < 0.01$) indicating excessive screen time has negative effects on health markers as a result of reduced energy expenditure. Matthews et al., (2016) also investigated PA levels and sedentary behaviour of US adults (n = 4840). On average, daily sedentary time was 8.2 hours. Six years post study, 700 deaths had occurred and compared with less-sedentary adults (6 sedentary hours per day), and individuals with 10 sedentary hours per day had a 29% greater risk of death. In addition to decreasing activity at home, decreasing work related PA is a large cause of insufficient PA. Church et al., (2012) examined PA in US employment and noted a significant increase in the prevalence of sedentary and light intensity PA occupations from 1960 to 2008 ($P < 0.001$), while the prevalence of moderate intensity PA occupations had decreased from 48% in 1960 to 20% in 2008 ($P < 0.001$). Clearly PA is insufficient on a global level, with sedentary behaviour being a major contributing factor.

2.4.2 Dietary trends contributing toward obesity
Excessive caloric consumption is a contributing factor to obesity. Several factors may influence this excessive caloric consumption, such as ease of access and increased consumption of calorie dense foods (Hawkes et al., 2007; Crino et al., 2015). The

quantity of calories consumed worldwide has vastly increased in recent decades which has influenced the increase of global obesity levels. Daily global calorie consumption has risen from 2411 kcal in 1969, to 2704 kcal in 1989, and to 2870 kcal in 2014 (Kearney, 2010; Statista, 2019). The WHO does not have a recommended daily allowance for calories but states that "energy intake (calories) should be in balance with energy expenditure" (World Health Organization, 2015). While caloric requirements can vary depending on factors such as gender and activity level, it is apparent from global obesity statistics that people consume above what is necessary.

In Ireland, the National Adult Nutrition Survey (2011) highlighted that 29% of men and 24% of women aged 18-64 years, and 24% of men and 10% of women aged over 65 years, consume above the maximum recommended weekly alcohol limit, and 63% of the population consume above the recommended fat intake. While overall caloric consumption is not listed, given the higher quantity of calories per gram in fat compared with protein and carbohydrates, this could influence body composition in a negative manner.

In addition to food preferences, ease of access to energy dense foods may influence dietary trends of individuals, leading to obesity. The worldwide quantity of McDonald's restaurant outlets increased from 951 in 1987 to 7,135 in 2002, and to 36,899 in 2016 (Statista, 2017). This large scale availability of processed fast food in recent years plays a part in influencing consumers' food choices. Multinational fast food companies often have large marketing budgets, and evidence reports that food marketing influences food choices and habits of people, especially children (Hawkes et al., 2007). This ease of access and marketing both contribute toward daily energy contributions up to 23% and 15% from fast food in males and females respectively, in the USA (Lachat et al., 2012). Typically, many energy dense foods contain high levels of fats and sugars which make them palatable but non-satiating, leading to consumption beyond energy requirements. This consistent consumption of food with a higher average energy density is associated with increased weight (Crino et al., 2015). Snacking behaviours are linked to weight status also, particularly when high frequencies of snacking are reported. Larson et al., (2016) reported adolescents consume a mean 4.3 snacks per day, and the measures of

snacking were directly associated (P < 0.01) with higher energy, lower fruit/vegetable, higher sugar-sweetened beverage, and more frequent fast-food intakes

2.4.3 Genetic link to obesity

The genetic make-up of individuals can influence the development of obesity. Some genetic links to obesity influence leptin, which plays an important role in regulating energy balance, as well as glucose, lipid and bone metabolism (Dalamaga et al., 2013). Leptin inhibits hunger to regulate energy balance and a leptin deficiency can lead to substantial over-eating, which has been known to cause early onset obesity (Montague et al., 1997). Other genetic links to obesity include mutations of the Melanocortin 4 receptor (MC4R) gene, which can also influence the likelihood of a person to develop obesity (Loos et al., 2008). Melanocortin 4 receptor gene acts on the hypothalamus in the brain to control appetite and satiety (Branson et al., 2003). While this can contribute to obesity, only 2.5% of people in the UK and Europe with a BMI greater than 30 kg/m^2 have pathogenic mutations in MC4R (Larsen et al., 2005). Leptin deficiencies are rare, and MC4R mutations typically only affect up to 2.5% of people with obesity (Larsen et al., 2005), so while these genetic factors may increase the chances of the person being obese, they are not a direct cause. A leptin deficiency and MC4R mutation may affect the persons' appetite, but it is the overeating that eventually leads to obesity. Evidence for genetic contributions to body composition generally arises from family and twin studies. Evidence for a genetic influence on body fat was suggested by Selby et al., (1989) who estimated heritability for subscapular skinfold thickness was strong (r = 0.77 P < 0.01). Malis et al., (2005) also noted strong positive relationships between regional body fat distribution in twins. Intra-class correlation between young and elderly twins revealed a genetic link for total fat (r = 0.83, and 0.86); trunk fat (r = 0.82, and 0.85); and lower body fat (r = 0.83, and 0.81) in both the young and elderly twins respectively. Though there are obvious similarities in body composition between families, these similarities however cannot be described by genetics alone. Environmental factors such as dietary habits may have been inherited from family and this could explain some of the similarities in body composition (Birch and Davison, 2001).

2.5 Health consequences of obesity

Obesity increases risk of negative health implications, and is associated with a number of comorbidities. Among the conditions influenced by obesity are mortality, hypertension, dyslipidemia, CVD, and T2DM.

2.5.1 Mortality

Obesity is one of the largest contributors to premature death, with increased risk of death associated with increases in several measures of body composition including BMI, waist to hip ratio (WHR) and waist circumference (WC) obesity (Kithara et al., 2014; Staiano et al., 2005; Seidell et al., 2010). Kitahara et al., (2014) conducted a meta-analysis involving 20 studies from the USA, Sweden, and Australia comparing mortality rates (deaths per 100,000 persons per year) for the class III obesity (BMI 40.0–59.9 kg/m^2) and normal-weight (BMI 18.5–24.9 kg/m^2) groups. Total annual mortality rates were significantly higher for individuals with class III obesity than for normal weight individuals, with differences of 509.3 and 382.5 deaths per 100,000 persons per year between groups for men and women, respectively. Individuals identified with class III obesity were at a much larger risk of death than normal weight individuals. Similarly, Borrell and Samuel, (2014) identified that obese adults had at least a 20% significantly higher rate of dying of all-causes, with BMI having a positive relationship with premature death.

Elevated WC and WHR have also been linked to all-cause mortality in a number of studies (Staiano et al., 205; Seidell et al., 2010). Waist hip circumference ratio is the ratio of the waist circumference to the hip circumference with >0.85 and >0.90 being considered high for males and females, respectively (World Health Organization, 2008). Staiano et al., (2005) reported that elevated BMI, WC and WHR (P < 0.05) were associated with increased risk of death, with WC being the most accurate predictor. However, at present there is no standard measure of WC that is widely accepted. Waist circumference is a measure of abdominal fat, which has reported to have severe negative health implications such as dyslipidemia and insulin resistance (Yajima et al., 2018). WC is a simpler measurement than WHR as it requires only one anthropometric measurement, therefore there is less chance of measurement error. Similarly, Seidell, (2010) identified that both WC and WHR are better predictors of death

than BMI, especially in elderly participants where often a negative correlation between BMI and mortality was observed. One potential reason for this result is that the BMI calculation does not take muscle mass into account, so a person with a high BMI could potentially have a healthy body fat range (Nuttall, 2015).

2.5.2 Hypertension

The WHO and International Society of Hypertension guidelines classify optimal blood pressure as <120/80 mmHg, with prehypertension 120–139/80–89 mmHg (Egan and Stevens-Fabry, 2015) and hypertension defined as >140/90 mmHg (Chalmers et al., 1999). Excessive weight is a major contributor to primary hypertension, accounting for up to 65–75% of the risk (Hall et al., 2015). Increased blood pressure during the development of obesity related hypertension may be due to decreased kidney function which can occur as a result of physical compression of the kidneys due to excess fat around the kidneys, and elevated sympathetic nervous system activity. Excess levels of adipose tissue around the kidneys can contribute to impaired sodium excretion, increased intra-renal pressures, and hypertension (Hall et al., 2014). In patients with visceral obesity, increased levels of intra-abdominal pressure as high as 35–40 mmHg have been detected (Sugarman et al., 1997) which can contribute to compression of the renal veins, lymph vessels, ureters and renal tissue. The Renin-Aldosterone-Angiotensin System also is a factor which may influence hypertension in obese populations. Overweight individuals often have increases in key regulators of blood pressure including plasma renin activity, angiotensin converting enzyme activity, angiotensinogen, angiotensin II, and aldosterone (Engeli and Shrma, 2001). Elevations of these systems may be due to increased pressure on the kidneys and increased sympathetic nervous system activation (Davy et al., 2004). Altered function of the autonomic nervous system also influences blood pressure in obese individuals (Hall, 2003). Renal sympathetic nerve activity is generally elevated in overweight compared to non-overweight participants (Davy et al., 2004), which initiates renin secretion and therefore sodium reabsorption. Consequentially this contributes to development and maintenance of obesity related hypertension (Hall et al., 2014).

2.5.3 Dyslipidemia

Adipose tissue plays a vital role both as an endocrine organ, and in fuel metabolism. Adipose tissue secretes a large quantity of proteins and other molecules, which can often be dysregulated as a result of obesity leading to atherogenic dyslipidaemia (AD) (Goossens, 2008). Atherogenic Dyslipidaemia refers to imbalances in some or all blood lipids, and is characterised by elevated levels of TG, LDL cholesterol, and TC, and low levels of HDL cholesterol (Manjunath et al., 2005). Total cholesterol concentrations of >5.17 mmol/L, LDL cholesterol levels of ≥3.36 mmol/L, TG levels of >1.69 mmol/L), and HDL cholesterol levels of <0.90 mmol/L indicate AD (Yang et al., 2012). Optimal levels of these lipids is of paramount importance as they play vital roles in maintenance of health. High-density-lipoprotein cholesterol protects against atherosclerosis by removing excess cholesterol from macrophages (Rosenson et al., 2016) and LDL cholesterol in the cell decreases synthesis of enzymes responsible for intracellular synthesis of cholesterol (Cox and García-Palmieri, 1990). Impaired lipid metabolism can occur in up to 60-70% of obese patients with AD (Feingold and Grunfeld, 2015). In AD, impairments in insulin signalling and insulin actions is due to increased secretion of cytokines and macrophages from adipose tissue leading to obesity-induced inflammation and insulin resistance (Jung and Choi, 2014). This in turn elevates the conversion of TG into free fatty acids (FFA), which in turn are transported to liver and muscles where they can be either oxidized to generate energy in the form of adensosine tri phosphate (ATP) or re-esterified for storage as TG (Mirana et al, 2005). Usually, insulin inhibits breakdown of fat in adipose tissue by inhibiting the intracellular lipase, however with obesity, this function is compromised. The majority of FFA are re-esterified to TG which along with Apolipoprotein B increases the assembly and secretion of LDL particles (Choi and Ginsberg, 2011). This in turn can lead to cardiovascular disease (CVD) risk, as increases in TG concentrations of 90 mg/dL are associated with a 32% increase of obtaining CVD (Abdel-Maksoud and Hokanson, 2002). Likewise, a 5 mg/dL decrement in HDL cholesterol is associated with a 14% greater risk of developing CVD (Gotto et al., 2000).

Central adiposity is particularly important as a key cause of insulin resistance, and thus abnormal hepatic metabolism. Central obesity is associated with elevated TG stores in non-adipose tissues such as liver, muscles, and pancreatic β-cells, which in turn

promotes the excretion of pro-inflammatory cytokines and macrophages (Bakker et al., 2000). Skeletal muscle is responsible for a major part of insulin stimulated whole-body glucose disposal and plays an important role in the pathogenesis of insulin resistance, as increased amount of intracellular lipids is associated with insulin resistance (Guebre-Egziabher et al., 2013). Furthermore, a correlation has been observed between obesity and ectopic lipid stores in cardiac muscle (Kankaanpää et al., 2006). Ectopic lipid storage in heart has been shown to be associated with left ventricular hypertrophy and cardiovascular cell insulin resistance (Davidoff et al., 2004). There is evidence that lipid accumulation causes activation of inflammatory markers and can trigger β-cell dysfunction and apoptosis in the pancreas, partly by inhibiting insulin signalling. (Kulkarni, 2002). Clearly ectopic lipid storage is a key driver of inflammation and insulin resistance. Consequentially, the quantity of FFA delivered to the liver as a product of visceral adipose tissue lipolysis rises as a result of visceral fat mass (Gastaldelli et al., 2007). In some cases of extremely overweight individuals, up to 50% of post-absorptive hepatic FFA delivery is from visceral fat, although on average this is approximately 20% (Nielsen et al., 2004). Therefore, individuals with excessive amounts of visceral fat are likely to have decreased hepatic metabolism.

2.5.4 Insulin resistance and Type II diabetes mellitus (T2DM)

Insulin is a peptide hormone produced by the β-cells on the islets of Langerhans in the pancreas. The function of insulin is to maintain blood glucose homeostasis which is achieved by regulating cellular glucose uptake, carbohydrate, lipid, and protein metabolism as well as promoting cell division, and differentiation (Riaz, 2014). Glucose is the primary stimulus for which insulin is released, though other macronutrients, hormones and neural factors may affect this response. Up to 0.25–1.5 units of insulin are secreted by the β-cells per hour during the fasting state in order to enable glucose insulin-dependent entry into cells (Kahn et al., 1997). This level prevents uncontrolled breakdown of triglycerides and limits gluconeogenesis which is the generation of glucose from non-carbohydrate substrates, in order to maintain normal fasting blood glucose levels (Kahn et al., 1997). In healthy individuals glucose stimulated insulin secretion occurs in two phases (Satin et al., 2015). The first phase of insulin release occurs within one minute of intravenous glucose administration, peaks at 3-5 minutes

and lasts approximately 10 minutes in duration, while the second phase is slower in nature, and though insulin release commences shortly after the glucose load, it is not apparent until 10 minutes later (Satin et al., 2015). This quantity of insulin released is proportional to the glucose concentration immediately prior to the administration (Satin et al., 2015).

The difference between the two phases is that the first phase of insulin secretion represents the release of insulin already synthesised and stored in the pancreas, while the second phase involves the release of a combination of stored and newly synthesised insulin (Wilcox, 2005). Insulin release depends on the total dose of glucose, and its rate of administration, with maximal pancreatic response occurring in response to 20g of glucose administered intravenously over three minutes (Wilcox, 2005). This occurs in healthy pancreatic cells functioning optimally, though this is not the case for a large percentage of obese individuals. Though the latter study referenced intravenous glucose administration, insulin secretion following oral glucose is much more variable and is affected by several factors such as, gastrointestinal motility, and gastric emptying. Optimal functioning of the insulin system is essential for glucose metabolism. Insulin release can continue sometime after glucose ingestion (Kahn et al., 1997).

Glycaemic control is maintained by the balanced relationship between insulin action and secretion. Fully functioning pancreatic β-cells adapt to changes in insulin action (Stumvoll et al., 2005). This includes upregulation or downregulation of insulin secretion. Therefore β-cell functioning is a vital component in the development of T2DM (Stumvoll et al., 2005). Type II diabetes mellitus is characterised by insulin deficiency caused by pancreatic β-cell dysfunction (Chatterjee et al., 2017). Insulin resistance also plays a role in the development of T2DM. Insulin resistance is defined as when a normal or elevated insulin level produces a decreased biological effect (Cafelu et al., 2001). Major areas of insulin resistance in the body include peripheral areas such as muscle and adipose tissue. Glucose uptake into muscle while resting is insulin dependent, and muscle accounts for about 60–70% of whole-body insulin mediated uptake, with adipose tissue accounting for approximately 10% (Smith, 2002).

Body composition has a profound effect on insulin secretion and in turn on T2DM, with a positive relationship existing between diabetes and body fat levels as noted by Nguyen et al., (2011). Mean fasting glucose and glycated haemoglobin (HbA1c) levels were highest for diabetics with a BMI >25.0 kg/m^2, suggesting a state of increased severity of T2DM in these individuals. Glycated haemoglobin is formed from the binding of glucose to haemoglobin and provides information on a person's average blood glucose control during the previous 8-12 weeks (Sherwani et al., 2016). Likewise, De Koning et al., (2010) and Narayan et al., (2007) concluded that an increase in BMI (P < 0.01), waist circumference (P < 0.01), or WHR (P < 0.01) significantly increased an individual's risk of developing T2DM.

There are a number of factors that link obesity to diabetes. Fat accumulation in all of the organs and tissues of metabolic importance cause insulin resistance as a result of increased secretion of cytokines and macrophages from adipose tissue which impair insulin signalling (Jung and Choi, 2014). Insulin cannot perform its normal metabolic actions (promote glucose disposal, inhibition of gluconeogenesis, inhibition of lipolysis) and the ultimate result is dysregulation of glucose and lipid metabolism (Miranda et al., 2005). The disposal of blood glucose is impaired and so glucose levels start to rise. Lipolysis is no longer inhibited so there is an influx of lipids into circulation and organs and tissues of metabolic importance which compounds the problem (Boden, 1997). The pancreas responds by producing and secreting more insulin (hyperinsulinemia), but over time this cannot be sustained and β-cells function decrease (Boden, 1996). At this point blood glucose levels rise into the clinical range of T2DM.

The International Diabetes Federation estimated that in 2015 over 336 million adults had T2DM globally (Zheng et al., 2018). Selvin et al., (2014) examined the prevalence of T2DM among overweight and obese populations from a representative sample of adults with diabetes participating in the National Health and Nutrition Examination Surveys between 1999 and 2006. Among the 21,205 surveyed participants, 2,894 had T2DM (13.6%). Of those with T2DM, 80.3% and 49.1% were considered overweight (BMI ≥ 25 kg/m^2) and obese (BMI ≥ 30 kg/m^2) respectively. The influence of weight on T2DM was

observed with only 8% of normal weight individuals having T2DM compared with 43% for individuals with class III obesity.

2.5.5 Cardiovascular disease (CVD)

Cardiovascular disease is an umbrella term for linked pathologies involving the heart or blood vessels and includes events such as coronary heart disease (CHD), cerebrovascular disease, peripheral arterial disease, rheumatic and congenital heart diseases and venous thromboembolism (Stewart et al., 2017). Stroke, angina and myocardial infarction (MI) are also included under the term CVD. Cardiovascular disease accounts for 31% of mortality worldwide, the majority of which consists of CHD (WHO, 2017). De Koning et al., (2007) conducted a meta-analysis investigating the relationship between WC, WHR and cardiovascular outcomes, with CVD defined as fatal and non-fatal CHD and stroke events. They concluded that a 1 cm increase in WC and a 0.01 increase in WHR was associated with a 2% and 5% increased risk of a CVD incident, respectively. Similarly, Yusuf et al., (2005) reported that WHR, WC and HC were significantly ($P < 0.01$) related with the risk of MI even after accounting for external risk factors (smoking, dietary habits and alcohol consumption). Obesity adversely affects cardiovascular structure and function, including increasing the incidences of left ventricle structural abnormalities such as ventricular and atrial enlargement, and leading to abnormalities in both systolic and left ventricle diastolic function (Lavie et al., 2013). The excessive fat accumulation caused by obesity results in greater demand for circulating blood volume and increased cardiac output (Brady, 2016). With increased intravascular volume, stroke volume is elevated resulting in increased work performed by the heart. The pressure against which the ventricles must work to eject blood rises in association with obesity also (Bastien et al., 2014). When these cardiovascular alterations are sustained cardiac remodelling occurs resulting in left ventricular hypertrophy (Brady, 2016). This in turn contributes toward the cardiovascular disease and events listed above.

2.6 Treatment of obesity

Several methods have been used previously to treat and manage obesity with varying degrees of effectiveness. Current methods include pharmacological interventions, surgical procedures, and lifestyle interventions involving dietary and exercise interventions.

2.6.1 Weight loss promoting drugs

Weight loss promoting drugs such as Orlistat and Sibutramine are prescribed to augment fat loss during lifestyle interventions. Methods by which these drugs work include increasing satiety by acting on the neurotransmitter receptors (Halford et al., 2005), and inhibiting lipase, which in turn reduces dietary fat absorption (Padwal et al., 2007). Orlistat, a lipase inhibitor has been shown to reduce weight by 2.9% more than when compared to a placebo in 6021 overweight or obese patients over a 1-year period. All participants were requested to follow a standardized low fat (less than 30% of caloric intake) hypocaloric diet and weight loss ranged from -3.29 to 10.30 kg with the use of Orlistat, while placebo weight loss ranged from -1.27 to 6.4 kg (Padwal et al., 2003). Sibutramine, a drug designed to increase satiety, has also been shown to reduce weight with a 4.6% decrease in bodyweight observed in 929 overweight or obese patients over a 1-year period also (Padwal et al., 2003). The effectiveness of these drugs in terms of weight loss is further highlighted by the fact that the percentage of patients achieving a 10% loss of body weight was 12% higher with Orlistat (Padwal et al., 2003) and 15% higher with Sibutramine (Padwal et al., 2007) compared to placebo.

Orlistat and Sibutramine also contribute toward improving physiological and metabolic markers of health, though this could be dependent on weight loss. Deroso et al., (2004) noted a significant increase in blood glucose control, as indicated by a decrease in HbA1c obtained after 6 ($P < 0.05$), 9 ($P < 0.05$), and 12 ($P < 0.01$) months in both Orlistat and Sibutramine groups. After 9 and 12 months, mean fasting blood glucose and post meal blood glucose levels were significantly decreased in both groups (both $P < 0.05$). Nguyen et al., (2011) has previously noted a positive relationship between body mass and HbA1c, so this decrease in HbA1c could be as a consequence of excess weight loss, and not the drug itself. A similar trial by Torgerson et al., (2004) compared the effect of Orlistat accompanied with a lifestyle intervention versus a lifestyle intervention in isolation on the delay or prevention of T2DM on 3300 obese patients, 21% of whom had impaired glucose tolerance. A 37% lower incidence of T2DM, with a 3.9 kg and 2.8 kg greater weight loss at 1 and 4 years post intervention respectively was observed in the Orlistat treated participants compared to those who completed just the lifestyle intervention. The only instructions participants received was to consume a reduced-

calorie diet (~800 kcal/day deficit) containing 30% of calories from fat, and to walk at least one extra kilometre per day in addition to their usual PA. Whether the augmented glucose metabolism is a result of the drug alone, or as a result of weight loss is unknown.

Though effective in terms of weight loss, significant side effects can occur while using weight loss promoting drugs such as Liraglutide and Naltrexone-bupropion. A meta-analysis by Khera et al., (2016) investigated the association of drug treatments of at least one year in duration for obesity with weight loss and adverse events. Overall, weight loss was 2.6 kg with Orlistat, 3.2 kg with Lorcaserin, 5.0 kg with Naltrexone-bupropion, 8.8 kg with Phentermine-topiramate, and 5.2 kg with Liraglutide when compared with placebo. In the meta-analysis, compared with placebo, all drug treated trials had 1.3 to 2.9 higher odds of being discontinued due to adverse events, such as hypoglycaemia, gastrointestinal distress, cardiac disorders and respiratory disorders (Khera et al., 2016). Other common side effects suffered as a result of Orlistat include fatty and oily stool, faecal urgency, and oily spotting which can occur in 15–30% of orlistat-treated participants (Padwal et al., 2007).

A common problem associated with weight loss drugs is the weight regain that has been noted following cessation of these drugs. In an experiment by James et al., (2000), 605 obese participants were assigned to a 6-month course of Sibutramine (10mg/day) and an individualised 600 kcal/day deficit programme. Of the 204 Sibutramine treated individuals in this study only 89 (43%) maintained 80% or more of their original weight loss, compared with nine (16%) of the 57 individuals in the placebo group. Similar weight regain was reported by Norris et al., (2005), Yanovski et al., (1996), and Davidson et al., (1999) suggesting that weight regain occurs in the majority of people after cessation of the drug treatment.

2.6.2 Surgical procedures

Surgical procedures can be used to treat extreme cases of obesity (Rosenthal et al., 2006), and include gastric bypass, sleeve gastrectomy, and gastric banding. Gastric bypass involves the surgeon creating a small pouch at the top of the stomach. This pouch is then the only part of the stomach that receives food and fluid (Park and Torquati, 2011). Gastric banding meanwhile involves decreasing the volume of the stomach with

a gastric band (O'Brien, 2010), while a sleeve gastrectomy involves separating and removing part of the stomach from the body. All three surgical procedures, although different in approach, limit the quantity of food that the stomach can hold.

Surgery for obesity has typically been reserved for patients with more severe obesity, and has been suggested for patients with a BMI ≥40 kg/m^2 (Yermilov et al., 2009). Several studies note the effectiveness of bariatric surgery, with Santos et al., (2014) reporting significant reductions in body weight (115.03 ± 22.39 to 99.90 ± 19.53 kg, P < 0.001) and BMI (43.10 ± 6.16 to 37.37 ± 6.28 kg/m^2, P < 0.001) in 46 patients before and 12 weeks post gastric banding surgery. Significant reductions in inflammatory markers including C-reactive protein (P < 0.05), and TG (P < 0.01) 12 weeks post-surgery were noted also. Likewise, Buchwald et al., (2004) analysed outcomes of 46 patients' evaluated 3 months post bariatric surgery. A mean percentage of excess weight loss (defined as the participants' current pre-operative weight minus the participants' ideal weight) of 61.2% (58.1%-64.4%) for all patients, 47.5% (40.7%-54.2%) for patients who underwent gastric banding, and 61.6% (56.7%-66.5%) for patients who underwent gastric bypass was reported. Other areas of metabolic health improved via surgery include T2DM, which was eradicated and improved in 76% and 86% of participants respectively, hyperlipidaemia, which was improved in 70% of patients and hypertension, which was resolved in 61.7% of patients and resolved or improved in 78.5% (Buchwald et al., 2004).

Surgical procedures can prove extremely effective in terms of overall weight loss, and reductions in mortality. Sjöström et al., (2007) conducted a study involving 4047 obese participants, 2010 of which underwent bariatric surgery and 2037 of which received conventional treatment. The conventional treatment was not standardised and varied between each health centre, ranging from lifestyle interventions and behaviour modification, to no treatment whatsoever. Ten years post-surgery weight losses were 25 ± 11% for gastric bypass, 16 ± 11% for vertical-banded gastroplasty, and 14 ± 14% for banding versus 2 ± 2% for the control group, with 101 deaths in the surgery group, with 129 deaths in the control group. Mortality was reduced massively, though MI and cancer were the most common causes of death. Weight regain has commonly been

noted in the months and years after surgery also, and remains a problem associated with surgery. Magro et al., (2008) reported BMI reduction (N = 782) was significant up to 18 months post gastric-bypass surgery (P < 0.001), but no longer significant at 24 months post-surgery. Weight regain was reported as being significant 48 months post-surgery (P < 0.01).

Though the aim of surgery is enhancement of health, all surgical procedures constitute a health risk. Morino et al (2007) studied the mortality rate after 14,000 bariatric surgery procedures including gastric bypasses, biliopancreatic diversions, gastric bandings, vertical banded gastroplasties, and biliointestinal bypasses from January 1996 to January 2006, with sixty day mortality reported at 0.25%. Similarly, Buchwald et al., (2004) reported operative mortality of 0.1% for 2297 patients undergoing gastric banding, and 0.1% in 749 patients undergoing gastroplasty, 0.5% in 5644 patients undergoing gastric bypass procedures, and 1.1% in 3030 patients undergoing biliopancreatic diversion or duodenal switch operations. Such statistics highlights that similar to all invasive procedures, bariatric surgery constitutes a risk to the individuals involved. Surgical procedures can be effective in terms of weight loss and metabolic health, but due to risk, high cost, and impracticality are not implemented on a wide scale.

2.6.3 Dietary interventions

Dietary interventions are common in those attempting to reduce body fat level, as caloric restriction is vital in treating obesity. Ross et al., (2000) conducted a 12-week trial involving 52 obese men (mean BMI 31.3 ± 2.0 kg/m^2) that induced a 700 kcal energy deficit daily. A mean weekly weight loss of 0.6 kg and total mean weight loss of 7.4 kg were observed over the 12-week period, as well as a significant reduction in total fat (-4.8 ± 1.2 kg, P < 0.001), and abdominal fat levels (-1.5 ± 0.5 kg, P < 0.001). Likewise, Redman et al., (2007) reduced calorie intake in order to induce a 25% energy reduction from baseline in 25 overweight male participants (BMI 25-30 kg/m^2), and noted participants reduced bodyweight by approximately 10% (−8.3 ± 0.8 kg, P < 0.01), fat mass by approximately 24% (-5.8 ± 0.6 kg, P < 0.01), and abdominal visceral fat by 27% (-0.9 ± 0.2 kg, P < 0.01) post-study. The effectiveness of dietary interventions at improving body weight is again shown in a systematic review by Curioni et al., (2005)

involving 9 dietary interventions consisting of 142 participants in total, with the length of the interventions varying from 10 to 52 weeks. Diet included very low energy diets and low energy diets, with no information provided regarding macronutrient distribution. The average weight loss per person was -9.9 ± 9.6 kg, and the average reduction in percentage body fat was -10 ± 3.6%.

Dietary interventions can improve metabolic factors also, such as glucose tolerance and insulin sensitivity. The previously mentioned study by Ross et al., (2000) noted glucose disposal was significantly improved ($P > 0.01$) when compared to controls following a 12-week 700 kcal per day deficit. Brinkworth et al., (2004) examined the effect of a high-protein, low-carbohydrate diet (HP) on glycaemic indices in T2DM patients. Participants were assigned to one of two groups, either a standard protein (SP) (15% protein, 55% carbohydrate) or HP (30% protein, 40% carbohydrate) diet, during 3 months of caloric restriction (1600 kcal consumption per day) and 4 weeks of energy maintenance (2000 kcal consumption per day). Both diets significantly decreased fasting insulin ($P < 0.01$), insulin resistance (measured by HOMA IR) ($P < 0.05$), and C reactive protein levels ($P < 0.05$), and increased HDL cholesterol concentrations ($P < 0.001$), with no significant difference observed between groups ($P > 0.05$). While weight loss does indeed improve metabolic health, there appears to be a dose response necessary in order to elicit positive changes. Steven et al., (2016) conducted a study on participants with T2DM (n = 30) that followed a very low calorie diet (VLCD) (700 kcal per day) for 8 weeks, followed by an isocaloric weight maintenance diet for 6 months. Isocaloric intake was individual to each person and was calculated from resting energy expenditure measured by indirect calorimetry. Body weight reduced from 98.0 ± 2.6 kg to 83.8 ± 2.4 kg ($P < 0.001$) following the 8-week VLCD, and did not change over the following 6 months (84.7 ± 2.5 kg, $P > 0.05$). In the 13 responders, fasting plasma glucose decreased by 8.9 ± 0.7 to 6.2 ± 0.1 mmol/L ($P < 0.05$) and remained constant following the isocaloric diet. In the 17 non-responders, fasting plasma glucose decreased 13.2 ± 0.6 to 10.9 ± 1.1 mmol/L ($P > 0.05$) post-VLCD and remained constant following the isocaloric diet. Achieving a blood glucose measure of <7 mmol/L is the standard threshold for remission of diabetes and was used to define the group of responders. All T2DM medication was stopped at

baseline in this study, highlighting the effectiveness of body weight loss, induced by dietary interventions at improving blood glucose metabolism.

There has been some debate as to whether altering the macronutrient ratio of weight-loss diets benefits body composition or metabolic health. Farnsworth et al., (2003) examined a HP diet (27% protein, 44% carbohydrate, and 29% fat) to a SP diet (16% protein, 57% carbohydrate, 27% fat) during a study involving 3 months of energy restriction (30% energy restriction) followed by 1 month of energy balance. Weight loss for all participants (-7.9 ± 0.5 kg, $P < 0.01$), total fat loss (-6.9 ± 0.4 kg, $P < 0.01$), fasting LDL cholesterol ($P < 0.01$), and HDL cholesterol ($P < 0.05$), were all significantly improved, with no difference between groups ($P > 0.05$). In addition, fasting insulin for all participants decreased by $33 \pm 3.3\%$ at week 12 ($P < 0.001$) and by $29 \pm 3.4\%$ at week 16 ($P < 0.001$), while the HOMA index for insulin resistance decreased by $32 \pm 4\%$, from 4.3 at week 0 to 2.5 at week 12 ($P < 0.001$), and by $27 \pm 4\%$ to 2.8 at week 16 ($P < 0.001$), with no difference between groups ($P > 0.05$). Though a HOMA result of <1.0 is classed as optimal, the improvements noted in the study were substantial. The results also suggest that in terms of metabolic health, diet composition does not matter once the total calorie deficit is equal.

Noakes et al., (2005) also investigated an energy-restricted HP diet versus a conventional high-carbohydrate, low-fat diet (HC) on weight loss in obese women. The HP diet consisted of 34% of energy from protein, 20% from fat, and 46% from carbohydrate, while the HC diet consisted of 17% of energy from protein, 20% from fat, and 64% from carbohydrate. One hundred women with a mean BMI of 32 ± 6 kg/m^2 and age of 49 ± 9 years completed the 12-week study, and were randomly assigned to one of the two isocaloric 1350 kcal dietary interventions. A significant weight loss was observed with both the HP diet (-7.6 ± 0.4 kg; n = 52) and the HC diet (-6.9 ± 0.5 kg; n = 48), but there was no significant difference in weight loss between the groups. Significant improvements were also reported in LDL-cholesterol, HDL-cholesterol, and glucose concentrations, independent of the type of diet. Diet type did affect triacylglycerols, which decreased by 8% with the HC diet and by 22% with the HP diet ($P < 0.05$). The previously mentioned study by Brinkworth et al., (2004) examined the effect of a HP diet

on glycaemic control in T2DM participants resulting in significantly increased HDL cholesterol concentrations (P < 0.001), decreased fasting insulin (P < 0.01), decreased insulin resistance (P < 0.05), and reduced C reactive protein levels (P < 0.05), with no significant difference between groups (P > 0.05). Such results suggest that metabolic health will improved regardless of the macronutrient breakdown once the calorie deficit is equal between diets. A meta-analysis by Johnston et al., (2014) analysed 48 randomized controlled studies (n = 7286) comparing various diets with no diet. All diets were superior in terms of total weight loss compared to no diet at 6-month follow-up (P < 0.05). Compared with no diet, low-carbohydrate and low fat diets had a median difference in weight loss at 6 months of -8.73 kg and -7.99 kg respectively. Therefore, it appears that when calories are matched between groups, altering the ratio of protein, carbohydrates and fats provides no additional benefit in terms of weight and fat loss.

Weight regain following cessation of dietary interventions is common, especially when the calorie intake with the intervention is particularly restrictive (Larsen at al., 2010). A meta-analysis by Franz et al., (2007) investigated weight-loss studies with a 12-month follow-up. Dieting participants reduced bodyweight by -4.9 kg (5%) at 6 months, and maintained a mean weight loss of -4.6 kg, -4.4 kg, and -3.0kg (4.6%, 4.4%, 3.0%) at 12, 24, and 48 months, respectively. Very low energy diets (<800 kcal/day) resulted in a -17.9 kg (16%) mean weight loss at 6 months, -10.9 kg (10%) at 12 months and -5.6 kg (5%) by 36 months indicating that weight regain occurs over time. Decreases in fat free mass has a considerable contribution to this weight regain. Menzzzi et al., (2000) investigated the effect of a 3-week low-energy diet (700 kcal/day) on ninety-three obese female participants and observed a significant reduction in body weight (-3.3 kg, P < 0.01), fat free mass (-1.9 kg, P < 0.01) and fat mass (-1.2 kg, P < 0.01). The low energy diet also resulted in a significant decrease in resting metabolic rate, (P < 0.01), with variations in metabolic rate before and after the dietary intervention explained by fat free mass (r^2 = 0.79 and 0.80, respectively). This shows that losses in fat free mass correlate with downregulation of metabolic rate. Neuroendocrine disturbances, such as thyroid status, are affected by diet induced weight loss, and this may play a role in the reduction of resting metabolic rate (Knuth et al., 2014). Pelletier et al., (2002) conducted a study on 16 obese participants on an exergy restricted diet, and noted significant

decreases (P < 0.05) in serum triiodothyronine over the course of the 15-week study. Triiodothyronine is a hormone produced by the thyroid and plays a role in regulation of metabolism and energy expenditure. The same study noted a decrease in resting metabolic rate also (−13%, P < 0.001). This clearly shows that losses in fat free mass correlate with downregulation of metabolic rate, which means that maintenance of weight loss over time is difficult. Exercise training could offset this loss of muscle and prevent the down regulation of metabolic rate (Dolati et al., 2017).

Diet interventions in overweight or obese adults have frequently been found to result in weight loss of less than 5% and do not result in positive metabolic adaptations, but a weight loss of greater than 5% appears necessary for beneficial effects on glycated haemoglobin, lipids, and blood pressure. Franz et al., (2015) concluded however that achieving greater than 5% weight loss requires intense interventions, including restriction of energy intake, regular exercise, and frequent contact with health professionals.

2.7 Exercise interventions
Exercise interventions are commonly used to treat and manage obesity, though the literature is somewhat inconclusive regarding the efficacy of such interventions. Various types of exercise interventions have been examined previously including aerobic exercise (Thorogood et al., 2011), resistance training (Schoenfeld et al., 2016), high intensity interval training (Talanian et al., 2007), and suspension training (Dolati et al., 2017). A review of all exercise interventions is beyond the scope of this literature review and therefore this review will focus on aerobic training and suspension training.

2.7.1 Aerobic exercise interventions
Aerobic exercise is defined as any activity that uses large muscle groups, can be maintained continuously and is rhythmic in nature (Wahid et al., 2016). Aerobic exercise is used extensively to treat obesity (Anderson et al., 1999; Johnson et al., 2009; Okura et al., 2005; Heijden et al., 2010; Dengel et al., 1996). Franz et al., (2007) performed a meta-analysis investigating clinical weight loss trials with a 12-month follow up. This meta-analysis included 4 different types of study designs involving aerobic exercise alone (frequency 2-5 times per week, intensity 50-74% VO_{2max} or 60-75% maximum

heart rate, and duration 15-45 minutes) with participants instructed to maintain normal dietary habits. These exercise interventions combined resulted in a mean -2.4 kg (2.7%) weight loss at 6 months and a mean -1.0 kg (1.0%) weight loss at 24 months indicating that this exercise mode was unsuccessful in terms of weight loss, and the maintenance of that weight loss over an extended time period. One limitation of this review is that aerobic exercise may potentially be under prescribed in the trials included in this review. Moderate to vigorous PA of 150 minutes weekly has been suggested as a guideline for health in recent years (World Health Organization, 2010), but weight loss may require exercise far in excess of this. For additional health benefits such as weight or fat loss, the WHO suggest that adults should engage in 300 minutes per week moderate-intensity aerobic PA, or 150 minutes of vigorous-intensity aerobic PA per week, or an equivalent combination of both (Garber et al., 2011). In addition to this, resistance training should be performed on 2 or more days a week (World Health Organization, 2010). Some of the trials included in this study fell short of this, with Donnelly et al., (2000) only prescribing 90 minutes of exercise per week for participants. Lastly, a major limitation of the meta-analysis was that none of the studies involved provided body composition changes in their results, but rather reported total weight loss. Whether this weight loss consisted of loss of fat mass or muscle mass is unknown.

Thorogood et al., (2011) conducted a meta-analysis of 14 aerobic exercise training trials involving 1847 overweight healthy participants. The aerobic exercise programs ranged from 12 weeks to 12 months in duration and consisted of 135-240 minutes of exercise at an intensity of 40-85% maximum heart rate or 40-70% VO_{2max} per week. Results were noted as weighted mean difference, the difference between the mean change in the exercise group and the mean change in the control group for both 6-month and 12-month programmes. Six month programs resulted in weight decreases of -1.6 kg and waist circumference decreases of -2.12 cm. Twelve month programs also resulted in weight reductions of -1.7 kg and waist circumference reductions of -1.95 cm. At 6-months, aerobic exercise resulted in the lowering of systolic and diastolic blood pressure by -1.8mm Hg and -2.92mm Hg, respectively, and total cholesterol decreases of -1.54mg/dL. This review concluded that aerobic training in isolation was not an effective mode of treatment for obesity, however some limitations of the meta-analysis were

identified. Exercise adherence was noted as one limitation, as intention to treat trials were used in the study. Intention to treat analysis means all patients who were recruited are included in the analysis regardless of withdrawals from the study or non-compliance. Secondly, the increase in exercise by participants may have been offset by an increased caloric intake, of which there was no record. Thirdly, like that of Franz et al., (2007) aerobic exercise may be under prescribed in the trials included in this review also as some of the trials included in this study fell short of WHO weekly recommendations for exercise, with two consisting of 120 minutes per week (Abe et al., 1997; Posner et al., 1992) with no measure of aerobic fitness being reported also. Finally, and the fourth major limitation, no body composition analysis was included, with waist circumference being the only measure analysed collectively.

Donnelly et al., (2013) however did prove the efficacy of aerobic exercise in the reduction of body weight and improvement of body composition. Participants were randomly allocated to exercise 5 days per week, for 10 months under supervision at either 400 or 600 kcal per session groups or to a control group. Weight loss decreased significantly post intervention in the 400 kcal per session group (-3.9 ± 4.9 kg, $P < 0.05$) and the 600 kcal per session group (-5.2 ± 5.6 kg, $P < 0.05$) but no change was observed in the control group. Likewise, significant improvements were observed in percent body fat post intervention for both the 400 kcal per session ($-2.9 \pm 3.9\%$, $P < 0.05$) and 600 kcal per session groups ($-4.4 \pm 4.4\%$, $P < 0.05$), with no change observed in the control group ($-0.6 \pm 2.4\%$, $P > 0.05$). When comparing exercise groups, there was no significant difference between the 600 kcal per session group and the 400 kcal per session group with respect to changes in percentage body fat and total weight loss ($P > 0.05$). The author concluded that supervised exercise is effective in achieving clinically significant weight loss. However, it is clear to see from the aforementioned studies that the quantity of overall exercise is important as under prescribing exercises results in no changes in body weight or body composition.

2.7.1.1 Physiological adaptations to aerobic exercise

Aerobic exercise leads to increased aerobic fitness which is measured by maximal oxygen uptake (VO_{2max}). VO_{2max} is defined as the maximum rate at which oxygen can be inhaled and utilized by the body during exercise (Bassett and Howley, 2000), and is a

product of the Fick equation. The Fick equation is as follows: $VO_{2max} = Q \times A\text{-}VO_2$ difference, with Q being cardiac output, and $A\text{-}VO_{2max}$ difference representing the arteriovenous oxygen difference. $A\text{-}VO_{2max}$ difference is augmented by aerobic exercise, which can result in increases in VO_{2max}, and this occurs due to increased capillarisation of skeletal muscle tissue (Prior et al., 2014). This occurs alongside increased blood volume with aerobically trained males and females exhibiting higher blood volume when compared with untrained individuals, which occurs due to elevated blood plasma and erythrocyte levels (Sawka et al., 2000). The increased number of capillaries and blood volume, combined with an increase in red blood cell count facilitates an increased extraction of oxygen by the exercising muscle (Jones and Carter, 2000).

Cardiac output is the quantity of blood the heart pumps in any one minute, and is expressed in litres per minute (Vincent, 2008). Cardiac output is a product of heart rate, the number of times the heart beats per minute multiplied by stroke volume, the amount of blood pumped per beat (Lee and Oh, 2016). Stroke volume is increased by aerobic exercise, due to a variety of reasons including adaptations in the structure and function of cardiac muscle. Lee and Oh, (2016) noted significant differences in left ventricular end-diastolic volume (EDV) ($P < 0.05$), left ventricular mass ($P < 0.05$), and left ventricular stroke volume ($P < 0.05$) between aerobically trained and non-trained groups. EDV refers to the amount of blood in the ventricles of the heart at the end of diastole and increases with aerobic exercise, predominantly due to increases in blood volume. The ventricles respond to this increased EDV with a more forceful contraction and a greater stroke volume ejected per beat of the heart. This is known as the Frank Starling mechanism (Delicce and Makaryus, 2017). Increases in diastolic expansion are in proportion to the strength of the heart's systolic contraction (Plotnick et al., 1986). Since cardiac output is a function of stroke volume and heart rate, and stroke volume increases with aerobic exercise, this in turn results in a simultaneous reduction in heart rate at rest and during submaximal exercise intensities (Kang et al., 2016). This also allows trained individuals heart rate to return to baseline, and recover faster after exercise (Darr et al., 1988).

Other adaptations that occur as a result of chronic aerobic exercise include adaptations to muscle mitochondria and enzymes. Mitochondria oxidize nutrient substrates to generate adenosine tri-phosphate (ATP) and, are primarily responsible for meeting the energy demands of prolonged exercise (Drake et al., 2016). Aerobic exercise elicits pronounced improvements in mitochondrial size, number, and function through several highly coordinated adaptive processes, including synthesis and incorporation of new mitochondria (Miller et al., 2011) and structural separation and joining of mitochondria (Youle and Van Der Bliek, 2012). These effects increase the overall efficiency by which mitochondria meet energy demands. Enzyme activity involved in bioenergetics processes are augmented with chronic aerobic exercise and also lead to increases in cardiovascular fitness (Maltais et al., 1996). Maltais et al., (1996) observed increased activity of the oxidative enzymes, citrate synthase ($P < 0.05$) and 3-hydroxyacyl-CoA dehydrogenase, ($P < 0.01$), both of which play a role in generation of ATP for energy consumption. Spina et al., (1996) also reported improvements in oxidative enzyme activity in twelve participants who completed 2 hours of cycling per day for either 7 (n = 5) or 10 days (n = 7) at intensities of 60-70% of VO_{2max}. Citrate synthase, mitochondrial thiolase, beta-hydroxyacyl-CoA dehydrogenase and carnitine acetyltransferase activity were elevated by 39%, 38%, 23%, and 47% in response to training, respectively ($P < 0.05$) with a 9% increase in VO_{2max} (from 2.97 ± 0.16 to 3.24 ± 0.17 l/min), while blood lactate levels were lower at the same work-loads than pre-training values. The increased activities of oxidative enzymes adapt rapidly in response to exercise training, which may contribute to increased VO_{2max} and lower submaximal lactate levels.

Blood pressure is lowered as a result of aerobic exercise due to decreases in systemic vascular resistance, decreased plasma norepinephrine, and lowered plasma renin activity (Fagard and Cornelissen, 2007). Systemic vascular resistance is the resistance the left ventricle must overcome to pump blood through the systemic circulation, and is defined as the pressure difference between the mean arterial pressure and the central venous pressure divided cardiac output (Dellinger et al., 2004). Norepinephrine is a neurotransmitter produced by the sympathetic nervous system, and an increase in sympathetic activity is one mechanism by which blood pressure can be elevated (Esler, 2000). Reductions of this neurotransmitter such as is seen with aerobic exercise result

in decreased vasoconstriction, leading to decreased systolic blood pressure. Renin is a hormone secreted by the kidney that plays a role in regulating blood pressure through the renin–angiotensin system. Renin breaks down angiotensinogen secreted from the liver into the peptide angiotensin I, which is then converted into angiotensin II, a potent vasoconstrictor of blood vessels (Weir and Dzau, 1999). Decreases in renin result in decreased systolic blood pressure. A meta-analysis by Cornelissen et al., (2005) noted reductions in resting systolic blood pressure of up to -6.9 mmHg in 31 mildly hypertensive study groups containing 492 participants in response to aerobic exercise training. Aerobic exercise training sessions involved 40-minute durations, intensities of 65% heart rate reserve, frequencies of three times per week, and intervention length of 12 weeks on average.

These physiological adaptations are vital for an overweight population as adaptations to mitochondria and muscle capilarisation contribute toward increases in cardiovascular fitness which has been reported as a strong predictor of all-cause mortality (Harber et al., 2016). Likewise, reductions in blood pressure and increases in cardiac output result in a lower risk of developing CVD, which accounts for up to 31% of mortality worldwide (World Health Organization, 2017).

2.7.1.2 Metabolic and cardiovascular adaptations to aerobic exercise
Aerobic exercise can positively influence the metabolic health of individuals who are overweight and obese as well as their physiological health. Positive metabolic adaptions to aerobic exercise include increased glucose disposal (Short et al., 2003) increased lipid turnover in skeletal muscle (Jeukendrop, 2002), improved insulin sensitivity (Jenkins et al., 2011), reduced risk of developing T2DM, reduced secretion of pro-inflammatory hormones, and improved blood lipid parameters resulting in a decreased risk of CVD (Leon and Sanchez, 2011).

2.7.1.2.1 Blood glucose utilisation
Increased blood glucose utilisation is one method by which metabolic health is altered with aerobic training (Short et al., 2003), with this increased utilisation manifesting itself in various ways, including increases in GLUT4, increased insulin sensitivity, and elevated glycogen synthase activity (Christ-Roberts et al., 2004; Ebeling et al., 1993). Glucose

transporter type 4 is a glucose transporter protein that facilitates greater uptake of blood glucose into skeletal muscle (Tremblay et al., 2003). Increases in GLUT4 concentrations have been observed in response to aerobic exercise in both healthy participants (Short et al., 2003) and populations with T2DM (Christ-Roberts et al., 2004). In the aforementioned studies participants exercised on a cycle ergometer at 70-80% of maximum heart rate, for 20-40 minutes 3-4 times a week per session for 16 weeks (Short et al., 2003), 3-4 times a week for 20-40 minutes per session at 70-80% of maximum heart rate for 16 weeks (Short et al., 2003), and 3-4 times per week, for 30-45 minutes per session at 60-70% of VO_{2max} for 8 weeks (Christ-Roberts et al., 2004). Insulin independent glucose uptake also occurs as a result of exercise with glucose uptake by contracting skeletal muscle occurring by facilitated diffusion (Samuel and Shulman, 2016). However, this is dependent on the presence of GLUT4 in the surface membrane and an inward diffusion gradient for glucose (Richter and Hargreaves, 2013). In addition to increases in GLUT4, aerobic exercise training decreases insulin resistance. Jenkins et al., (2011) conducted a 6-month aerobic exercise study, entailing exercising at 50-70% VO_{2max} for 20-40 minutes three times a week, that resulted in decreased insulin resistance among obese individuals ($P < 0.05$). This change manifested itself as a 16% decrease in the HOMA-IR test, which is a measure of insulin resistance.

Aerobic exercise also increases glycogen synthase activity (Ebeling et al., 1993). Glycogen synthase is an enzyme utilised in glycogenesis, the conversion of glucose into glycogen. When comparing trained versus untrained individuals Ebeling et al., (1993) noted that muscle glycogen synthase activity was 33% greater in trained individuals compared with untrained participants ($P < 0.05$). Christ-Roberts et al., (2004) also conducted a study into blood glucose utilisation and noted increases of 46 ± 17% and 45 ± 12% in glycogen synthase activity in nondiabetic and diabetic participants, respectively, in response to aerobic training ($P < 0.01$) with training consisting of 16 weeks, 3-4 times per week, for 30-45 minutes per session on a cycle ergometer at 60-70% of VO_{2max}. Increases in glycogen synthase activity is important because this increases glucose disposal.

2.7.1.2.2 Blood lipids

Blood lipid parameters are impacted positively by aerobic exercise also. Leon and Sanchez, (2001) performed a meta-analysis consisting of 51 interventions involving 3 months or more of aerobic exercise (n = 4,700). Aerobic exercise was performed three to five times weekly for 30 minutes or more per session of varying intensities from 50-85% VO_{2max}, and 65 to 90% of maximum heart rate. While TG and LDL cholesterol decreased by 3.7% and 5% (P < 0.05), respectively, HDL cholesterol increased by 4.6% (P < 0.05), and TC remained unchanged. Increased LDL cholesterol, and decreased HDL cholesterol levels have been strongly linked with CHD risk (Ference et al., 2017; Sharrett et al., 2001). The ratio of these lipids to each-other is perhaps more important than the absolute values, as Manninen et al., (1992) suggested that the LDL/HDL ratio was a strong indicator of adverse cardiac events. Likewise, the serum TG level along with this ratio revealed that participants with LDL/HDL ratio >5 and triglycerides >2.3 mmol/l had an increased relative risk of cardiac events when compared with those with an LDL/HDL ratio <5 and triglyceride concentration 2.3 mmol/l. Bleda et al., (2012) also conducted a study observing the TC/HDL cholesterol ratio and noted that improving this ratio results in improved endothelial functioning in individuals with peripheral artery disease. The improvements in these blood lipid parameters may potentially be as a result of increased fat oxidation and increased enzyme expression.

Exercise can acutely elevate fat oxidation, and aerobic exercise has the potential to increase the capacity to oxidize fat (Jeukendrop, 2002). This suggests that regular aerobic exercise could potentially contribute to the loss of fat mass and improvements in metabolic health via increased fat oxidation (Jeukendrup, 2002). This is of vital importance given that overweight and insulin resistant individuals may have diminished ability to oxidize fatty acids as a consequence of this insulin resistance (Kelley and Goodpaster, 2001).

Although the mechanism of exercise-induced lipid changes is unclear, exercise itself may increase enzyme activity involved in blood lipids, helping regulate blood lipids (Earnest et al., 2013). One such enzyme is lipoprotein lipase, which is elevated substantially post exhaustive or glycogen depleting exercise, but which is unchanged during moderate intensity exercise (Melanson, 2009). Lipoprotein lipase plays a critical role in oxidation

of FFA in the body, and in chylomicron and LDL hydrolysis. Miyashita et al., (2010) observed that 12 weeks of jogging training increased lipoprotein lipase concentrations in overweight men compared to a walking group where no change was noted. This suggests exercise intensity is an important parameter in regulation of lipoprotein lipase activity. Aerobic exercise increases intracellular lipid content in parallel to increases in oxidative capacity (Pruchnic et al, 2004). Pruchnic et al., (2004) noted increased intracellular lipid levels following 12 weeks of aerobic exercise. However, the oxidative capacity of muscle as determined by succinate dehydrogenase staining, significantly increased ($P < 0.05$) along with the percentage of type I fibres ($P < 0.05$).

Fat oxidation during exercise increases in a linear fashion with exercise intensity up to 55-65% of VO_{2max}, but decreases at higher exercise intensities when carbohydrates become the primary fuel source (Achten and Jeukendrop, 2004). The intensity at which maximal fat oxidation occurs varies according to a variety of factors including training status and gender, with maximal fat oxidation occurring at higher intensities in trained athletes, and in women (Achten and Jeukendrop, 2004). The ability of females to utilize greater levels of fat during exercise may be as a result of several physiological reasons, such as differences in levels of circulating hormones, a higher percentage of oxidative muscle fibres, an elevated sensitivity to catecholamine initiated lipolysis, or elevated levels of hormone-sensitive lipase (Melanson et al., 2009). Hormone-sensitive lipase is a vital regulator in the mobilization of fatty acids from acylglycerols (Holm, 2003). The mode of exercise performed also influences the extent of fat oxidation with rates being higher when walking and running compared to cycling. This may be as a result of recruitment of a smaller muscle mass and a lower catecholamine response during cycling (Achten et al., 2003).

Aerobic training induces elevated fat oxidation during submaximal exercise in response to moderate intensity (60-75% of VO_{2max}) exercise training interventions of 6-12 weeks in duration (Melanson et al., 2009). A study by Warren et al., (2009) compared short versus long-duration ergometer cycling exercise (30 minutes vs. 90 minutes) matched for intensity, and low versus high-intensity cycling (50% vs. 85% of VO_{2max}) matched for energy expenditure. Altering exercise duration did not affect exercise VO_2 or RER ($P >$

0.05). However, RER was lower and fat oxidation was higher during the post-exercise period for the 90-minute group vs. 30-minute group (P < 0.05). The 85% of VO$_{2max}$ group resulted in significant elevations in energy expenditure and fat oxidation post exercise (P < 0.01) compared with the lower intensity group. This elevation of energy expenditure possibly occurs as a result of an oxygen debt caused by glycogen synthesis from lactate post exercise (Børsheim, and Bahr, 2003). These results demonstrate that by altering exercise intensity and work duration, post-exercise energy expenditure and fat oxidation can be increased, although the amount of fat oxidized after exercise may be minimal compared with that oxidized during the exercise bout. While exercising at an intensity of 85% VO$_{2max}$ may lead to greater energy expenditure post-exercise than exercising at 50% VO$_{2max}$, it would be easier to sustain exercise at the lower intensity for a longer duration which may lead to more fat oxidised during the exercise session, and overall. This is vital to consider when designing aerobic exercise programmes with the aim of improving body composition and metabolic health in overweight and obese individuals.

2.7.2 Suspension training

Suspension training involves utilising a set of ropes or straps suspended from an overhead anchor in order to exercise against a person's own body weight. The degree of resistance experienced during suspension training depends on the manipulation of gravitational pull, generally expressed as a percentage of the individual's body mass. This means that by altering the length of the straps, angles of pull, and body positions the resistance encountered during a certain movement can quickly be increased or decreased (Melrose and Dawes, 2015). This manipulation of angles and resistance facilitates for an increased range of motion when compared with standard barbell movements. This is because the handles allow the body to pass through the point of contact (the handles), which a standard barbell does not allow.

The literature regarding the effects of suspension training on body weight and body composition is inconclusive, though several studies note significant changes in both (Dolati et al., 2017; Smith et al., 2016). Janot et al., (2013) reported no change in body weight, WC, or abdominal skinfold measurements (P > 0.05). There were however significant (P < 0.05) differences in abdominal flexor, back extensor, side bridge

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endurance, flexibility, balance, and lower body strength following three training sessions per week for 7 weeks. Participants aged 18-54 years (n = 23) performed ten exercises for two sets of ten repetitions, at an RPE of 5-7 (hard to very hard). However, information was not provided as to the ratio of males to female participant, training status in other exercises modes, or weight status. Positive adaptations were also observed by Smith et al., (2016) following the performance of 3 suspension training sessions per week for 8 weeks, though no details were provided about the training sessions. This study involved sixteen participants (mean ± SD: age, height, weight, percentage body fat, and VO_{2max} = 40.1 ± 13.5 years, 165.3 ± 8.2 cm, 64.2 ± 11.9 kg, 23.0 ± 5.0%, and 41.2 ± 7.3 mL/kg/min, respectively) and resulted in significant ($P < 0.05$) improvements in the following parameters: systolic and diastolic blood pressure, waist circumference, body fat percentage, one-repetition maximum for leg press and bench press, and push-ups. Like that of Janot et al., (2013) this suspension training protocol proved effective in terms of increasing physical performance, and also resulted in improved body composition also, with a decrease in body fat percentage (-1.56%, $P < 0.05$) as measured by hydrostatic weighing. Bodyweight was not statistically different post intervention with a -0.7 kg reduction ($P < 0.05$). Dolati et al., (2017) conducted a suspension training study with 24 cases of overweight female participants (age: 29 ± 4.48 years, height: 162 ± 4.97 cm, weight: 73.4 ± 5.47 kg and BMI: 27.85 ± 2.02 kg/m^2). The exercise program consisted of an 8 week intervention with three training sessions per week, with an intensity of between 50-80% of maximum heart rate. The number of the exercises increased from 12 exercises including four lower body, four upper body and four abdominal exercises in week one to 30 exercises including 10 lower body, 10 upper body and 10 by week 8. After eight weeks of training, upper body muscular strength, lower body strength, and VO_{2max} were all significantly improved from pre-intervention ($P < 0.05$). Likewise body fat percentage reduced from 39.8 ± 5.4 to 36.9 ± 4.9% ($P < 0.001$) and body fat mass decreased from 28.2 ± 4.5 to 26.3 ± 4.1 kg ($P < 0.05$) while total body weight remained unchanged ($P > 0.05$). Such findings highlight the efficacy of suspension training in improving body composition and increasing physical performance characteristics.

2.7.2.1 Physiological adaptations to suspension training

Suspension training potentially provides several advantages over regular weight bearing or body weight resistance training. This includes the potential to induce greater muscular damage, and increased electrical activity of muscle groups.

2.7.2.1.1 Muscular damage

The greater range of motion achieved with suspension training compared with traditional resistance training provides a greater distance by which muscles can lengthen (eccentric contraction) and shorten under tension (concentric contraction), which could potentiate greater muscular hypertrophy (Schoenfeld, 2016). This is due to the performance of eccentric actions eliciting the greatest disruptions to contractile, structural, and supportive elements (Enoka, 1996). During an eccentric contraction skeletal muscle is forcibly stretched under tension, which induces significant damage to cellular components (Schoenfeld et al., 2015) and signalling pathways for muscle hypertrophy are activated in response to this. Muscular hypertrophy induced by exercise is regulated by a number of signalling pathways, including Akt/mammalian target of rapamycin (mTOR), mitogen-activated protein kinase (MAPK), and calcium dependent pathways (Schoenfeld et al., 2010). Each of these pathways are heavily involved in modulating hypertrophy in muscular tissue. As eccentric contractions produce higher levels of mechanical tension than concentric contractions, muscular damage is more pronounced with eccentric contractions. This muscle damage results in muscular tenderness or soreness known as delayed onset muscle soreness (DOMS) (MacIntyre et al., 1995) that becomes progressively worse from 24 to 48 hours post exercise. Damage to the muscle cell sarcolemma results in the accumulation of calcium ions in the cell, resulting in inflammation and circulation of neutrophils. Intracellular components and markers of muscle damage, such as creatine kinase, diffuse into the plasma and macrophages peak in quantity at 48 hours post-training and sensitise nerve endings to stimulation resulting in pain. This results in reduced muscle strength but post resistance training exercise the myocytes adapt, and increase in size (Schoenfeld et al., 2015). Increasing muscle mass has previously been associated with elevated resting metabolic rate which accounts for a large percentage of an individual's total daily energy expenditure (Dolezal and Potteiger, 1998). Increasing resting metabolic rate could

influence body composition in a positive manner as an individual is burning more calories while resting.

Both hormones and cytokines are vital in the hypertrophic response, and in regulating anabolic actions. Increased anabolic hormone concentrations result in an increased potential for receptor interactions, which regulate protein metabolism and muscle growth (Crewther et al., 2006). Anabolic hormones involved can include testosterone, growth hormone, and Insulin like Growth Factor 1 (IGF1). Scheet et al., (2011) observed an elevation in the testosterone:cortisol ratio by as much as 67% 120 minutes post a suspension training bout. This could potentially promote anabolism both by increasing the rate of protein synthesis and inhibiting protein breakdown (Buresh et al., 2009). Ahtiainen et al., (2003) reported significant correlations between increases in testosterone and increases in muscle cross-sectional area ($r = 0.76$, $P < 0.05$). This suggests that acute exercise induced elevations in testosterone may play an important role in regulating muscle hypertrophy. Dudgeon et al., (2011) showed an elevated growth hormone response to a single suspension training bout also, which is of vital importance as it is a peptide hormone that stimulates growth, cell reproduction, and cell regeneration in humans and other animals. Essentially, growth hormone stimulates the growth of all tissues of the body. An exercise induced elevation in growth hormone has previously been highly correlated with the magnitude of muscular hypertrophy in both type I ($r = 0.71$) and type II ($r = 0.74$) muscle fibres (McCall et al., 1999). It could be inferred from this that the elevation in growth hormone caused by suspension training could potentially cause increased muscular hypertrophy.

2.7.2.1.2 Muscle activation

Neural adaptations to resistance training play an important role in muscle strength gains and can occur in the absence of muscular hypertrophy (Moritani and DeVries, 1979). Increased electrical activity is evident in the early stages of resistance training, resulting in significant improvements in muscular strength, without changes in muscle mass or physical muscle architecture being observed (Moritani and DeVries, 1979). These neural adaptations are any changes within the nervous system that result in an individual activating working muscles in specific movements to a greater degree, or better coordination in the activation of all utilised muscles, resulting in a greater force

production in the movement (Sale, 1988). Increased excitability of motor neurons, changes in motor unit firing rate, muscle fibre conduction velocity are all improved as a result of resistance training and ultimately result in an increase in the rate of force production and muscular strength post-resistance training (Del Vecchio et al., 2019).

A number of authors have reported increased muscle activation with suspension training when compared with similar barbell and bodyweight resistance training exercises (Calatayud et al., 2014; Byrne et al., 2014; Snarr at al., 2013; Snarr and Esco 2013; Mok et al., 2015). Snarr et al., (2013), found increased agonist electrical activity for suspension training push up exercises when compared with bodyweight push ups, as measured by maximum voluntary contraction (MVC). The mean peak electromyographic values were significantly higher for pectoralis major (69.54 ± 27.6 vs. 63.62 ± 16.4% MVC, $P < 0.05$), anterior deltoid (81.13 ± 17.77 vs. 58.91 ± 20.3% MVC, $P < 0.05$) and triceps brachii (105.83 ± 18.54 vs. 74.32 ± 16.9% MVC, $P < 0.05$) during the suspension push-up versus the traditional push-up. It appears that agonist muscle activation is increased, when the stability of that muscle or joint is challenged, as is observed with suspension training exercises. Snarr et al., (2013) also noted increased activity of the rectus abdominis during the suspended push up exercise, when compared with a push up from the floor ($P < 0.05$). In the same study the rectus abdominis showed similar activations levels in the suspended push up and floor based crunch exercises. When normalized for MVC, rectus abdominis activity during the suspension push up, bodyweight push up, and floor based crunch were 68.0 ± 16.5%, 21 ± 16.6%, and 52 ± 28.7% of MVC, respectively. The raw and %MVC values were lower during bodyweight push up, compared to suspension push up and floor based crunch ($P < 0.05$). The authors concluded that when body-weight resisted movements of the upper body are performed on unstable devices, as is the case with suspension training, there is a greater muscular force production that leads to an increased rectus abdominis activity in order to retain stabilisation of the body.

This increased activation of motor units associated with suspension training could potentially lead to greater levels of strength and hypertrophy of selected muscle groups versus traditional resistance training methods. This is due to muscle hypertrophy being

dependant on recruitment of as many motor units as possible in working muscles, and high firing rates in these motor units for a sufficient period of time (Wernbom et al., 2007). This potential for hypertrophy associated with suspension training is of vital importance for the obese population as skeletal muscle mass is highly correlated with basal metabolic rate, and increasing muscle mass should increase metabolic rate (Vaughn, 2013). Increased numbers of recruited motor units should lead to developments in muscular strength as force output is directly correlated to amount of motor units recruited (Yao, 2004). Given the unstable nature of suspension training and the increased muscular activation, this should lead to increases in strength levels transferrable to everyday life for obese individuals.

2.7.2.2 Metabolic response to suspension training
Due to the eccentric nature of suspension training and the potential augmented muscular damage provided by this type of training there is a potential for increased post exercise energy expenditure. Likewise, though this varies depending on the structure of a training session, due to the intensity that suspension training is performed at there is a potential for increases in oxidative enzymes and elevated fat oxidation.

2.7.2.2.1 Energy demands of suspension training
Snarr et al., (2014) investigated the energy demands of suspension training. Twelve physically active participants (age: 24.67 ± 2.90 years, body weight: 78.92 ± 8.50 kg, body fat percentage: $13.87 \pm 7.31\%$) completed a suspension training bout consisting of nine exercises performed for 30 seconds each, interspersed with a 15-second rest period, followed by a 1-minute cardiovascular sprint. The exercise bout elicited an average heart rate of 148.0 ± 12.0 beats per minute ($82.99 \pm 4.20\%$ maximum heart rate) and a mean VO_2 of 24.34 ± 3.24 ml/kg/min, ($55.97 \pm 5.82\%$ VO_{2max}), while total caloric expenditure for the exercise bout was 96.98 ± 19.49 kcals. According to Achten et al., (2004) this exercise intensity is within the optimal fat oxidation zone (range of intensities with fat oxidation rates are highest) which is located between 55 ± 3 and $72 \pm 4\%$ VO_{2max}. Though the results obtained by Snarr et al., (2014) are on the lower end of the fat oxidation spectrum it could be assumed that exercise of this intensity would stimulate activity of aerobic enzymes with Grandjean et al., (2000) demonstrating that levels of lipoprotein lipase remained elevated for up to 48 hours post aerobic exercise of a similar intensity.

This is an important benefit as increased fat oxidation is desirable for an overweight and obese population. Smith et al., (2016) however reported an exercise intensity of 45.9 ± 8.6% VO_2 reserve (VO_{2max} minus resting VO_2), an average heart rate corresponding to 59.9 ± 10.2% of heart rate reserve, and a total energy expenditure of 398.1 ± 114.1 kcal per class. This involved sixteen participants (mean ± SD: age, height, weight, percentage body fat, and VO_{2max} = 40.1 ± 13.5 years, 165.3 ± 8.2 cm, 64.2 ± 11.9 kg, 23.0 ± 5.0%, and 41.2 ± 7.3 mL/kg/min, respectively). No details were provided regarding the exercise session. Based on previous research, resting VO_2 for individuals is approximately 3.5ml/kg/min (Sergi et al., 2011) and given that VO_2 reserve consists of VO_{2max} minus resting VO_2 this allows us to draw some comparison between the Smith et al., (2016) study and other studies. Based on the recommendations by Achten et al., (2004) this would be below the optimal fat oxidation zone, though energy expenditure for the entire session was four times greater than that of Snarr et al., (2014). Potential reasons behind the inconsistency in exercise intensity and energy expenditure between bouts include the session length (9 minutes vs. 60 minutes) and participant selection (body fat percentage of 13 vs. 23%), which vary hugely between studies. However, given that VO_{2max} and VO_2 reserve are both different measures, it is difficult to draw a clear conclusion as to energy demands of suspension training

The respiratory exchange ratio (RER) (CO_2 production/O_2 uptake) is a measure of fuel usage during exercise and determines the contribution of carbohydrate and lipids to overall energy expenditure (Simonson and DeFronzo, 1990). A high RER (closer to 1.0) indicates that carbohydrates are the primary fuel source, whereas a low RER (closer to 0.7) suggests lipid oxidation. During a suspension training bout involving 23 exercises, performed with a work to rest ratio of 30 seconds to 60 seconds Dudgeon et al., (2015) reported that RER ranged from 0.85 ± 0.01 during the rest interval to 1.03 ± 0.01 during the work intervals suggesting that substrate sources vary throughout the workout from fat oxidation to carbohydrate oxidation.

2.7.2.2.2 Post-exercise oxygen consumption
Resting energy expenditure has been shown to be increased up to 72 hours after traditional resistance training involving an eccentric component (Hackney et al., 2008). Hackney et al., (2008) induced DOMS using eight sets of six repetitions of 8 exercises

performed with a 1-second concentric and 3-second eccentric contraction. Resting energy expenditure was significantly higher at 24, 48, and 72 hours post training compared with baseline values for both trained and untrained participants ($P < 0.05$). Resting energy expenditure can represent up to 60-70% of an individual's total daily energy expenditure, with the rest attributed to the thermic effect of food and PA (Levine et al., 2001). Due to the eccentric nature of suspension training exercise it could be inferred that resting energy expenditure would be increased following a suspension training bout also (Hackett et al., 2008), perhaps to a higher degree when compared with traditional resistance training exercise. Raising resting energy expenditure is of particular importance to overweight and obese populations as this enables them to expend more calories while resting.

2.7.3 Summary of physical, cardiovascular and metabolic benefits of aerobic exercise and suspension training

In summary aerobic exercise contributes to weight loss and improved body composition (Donnelly et al., 2013), enhanced cardiac function (Lee and Oh, 2016), increased mitochondrial size number and function, (Miller et al., 2011), increased enzyme activity (Maltais et al., 1996), reduced hypertension (Cornelissen et al., 2005), improved blood glucose utilisation (Christ-Roberts et al., 2004), decreased insulin resistance (Duncan et al., 2003), improved blood lipid parameters (Leon and Sanchez, 2001), and increased fat oxidation capacity (Jeukendrup, 2002). Suspension training meanwhile leads to significant improvements in body composition (Dolati et al., 2017), muscular strength (Janot et al., 2013), muscular endurance (Dolati et al., 2017), balance ability (Janot et al., 2013), systolic and diastolic blood pressure (Smith et al., 2016), and VO_{2max} levels (Dolati et al., 2017). In addition to this suspension training may have the potential to increase muscular hypertrophy and resting metabolic rate to a greater degree than standard resistance training. Due to the greater eccentric emphasis and the unstable nature of suspension exercises, suspension training may lead to greater levels of muscular damage and increased muscular activation. Due to the benefits of aerobic and suspension training in isolation, combining both modes into a singular intervention could potentially prove more effective than these modes in isolation at treating and managing obesity

and related comorbidities such as insulin resistance, T2DM, dyslipidemia, elevated blood pressure, cardiovascular disease, and ultimately mortality.

2.8 Biomarkers

A biomarker is an objectively measured and quantified indicator of biological functioning, pathogenic processes, or pharmacologic responses to an intervention (Strimbu and Tavel, 2010). Biomarkers are produced in various tissues across the body such as adipose tissue, pancreas, and muscle tissue, and can be pro-inflammatory or anti-inflammatory in nature (Graham et al., 2017). Myokines are an example of biomarkers produced in muscle tissue, while adipokines are produced in adipose tissue and monitoring established biomarkers is crucial for detection and monitoring of various conditions such as dyslipidemia, T2DM, and CVD. Circulating concentrations of biomarkers are influenced by factors such as body weight, fitness levels, dietary habits and body fat levels. Examples of established biomarkers include cholesterol, and insulin, which play an extensive role in carbohydrate and lipid metabolism, and therefore cardiovascular and metabolic health. In addition to these well-established biomarkers, novel biomarkers are continually emerging and their potential roles in obesity and metabolic health are currently under investigation. Myostatin is one such novel biomarker.

2.8.1 Myostatin, a novel biomarker of insulin resistance

Myostatin is a myokine, and a transforming growth factor that is a regulator of skeletal muscle growth (Lee and McPherron, 2001). The primary function of myostatin is to inhibit skeletal muscle size, and mutations of the myostatin gene downregulate myostatin production and can cause increases in skeletal muscle, though much of the literature in this area pertains to animals (Lee, 2007). Substantial increases in skeletal muscle mass have been noted in mice with the myostatin gene removed, with individual muscles weighing twice as much as mice with the gene (Lee and McPherron, 2001). During embryonic development and during adult life of mice myostatin is expressed in skeletal muscle cells and works to limit both the final number of muscle fibres that are formed and limits cross sectional area of muscle fibres formed (Guo et al., 2009).

Human research has shown increased production of myostatin in the skeletal muscle of obese women compared to lean women ($P < 0.05$) (Hittel et al., 2009), suggesting muscular growth could potentially be inhibited as a result of excess fat mass. Weight loss induced by partial gastrectomy has also been observed to result in a significant lowering of muscle myostatin in obese participants ($P < 0.05$) and these changes in myostatin mRNA were also positively correlated ($r = 0.83$) with changes in fat free mass induced by weight loss (Milan et al., 2005). Myostatin also potentially plays an important role in metabolic health. Amor et al., (2018) investigated serum myostatin concentrations and noted positive correlations with estimators of insulin resistance and β-cell function. Myostatin positively correlated with area under the curve for insulin during the oral glucose tolerance test ($r = 0.47$) and HOMA-IR ($r = 0.43$). Furthermore myostatin serum concentrations were negatively correlated with the composite insulin sensitivity index ($r = -0.42$), indicating lowered insulin sensitivity with elevated myostatin concentrations. The role of myostatin in blood glucose regulation is further supported by McPherron and Lee, (2002) who reported that mice with the myostatin gene removed exhibited dramatically improved glucose metabolism.

Exercise appears to play a positive role in myostatin regulation, though research is lacking in the area. Hittel et al., (2010) examined myostatin levels of 10 insulin-resistant, middle aged (53.1 ± 5.5 years) men before and after 6 months of moderate aerobic exercise aerobic consisting of 1,200 kcal per week expended at 40–55% peak VO_2. Exercise modes and frequency were unique to each participant and included cycle ergometer, treadmill, and elliptical trainers. Hittel et al., (2009) noted both muscle protein and plasma myostatin levels decreased ($P < 0.05$) in response to the 6 months aerobic exercise. Strong correlations have also been reported between insulin sensitivity and plasma myostatin levels ($r = 0.82$). Myostatin mRNA levels have been reported to decrease acutely in response to aerobic exercise as well, with a 3–4 fold reduction in gastrocnemius biopsies 4, 8 and 12 hours after a single 30-minute running bout at 75% VO_{2max} in physically active individuals (Allen et al., 2011). Resistance training also appears to elicit favourable effects on myostatin concentrations with Kim et al., (2005) observing decreases of 44% in myostatin ($P < 0.005$) post resistance training consisting of 3 sets of 8-12 repetitions in three leg exercises.

Myostatin is affected by obesity (Hittel et al., 2009), and in turn can impact metabolic health (Amor et al., 2018). Research investigating the effects of exercise training on myostatin has indicated that improvements in body weight, body composition and metabolic health are linked with favourable alterations in the production of myostatin. The mechanisms causing these adaptations are not yet understood, therefore there is a need to study the interaction between myostatin and exercise further.

2.9 Conclusion

Obesity is a global problem and incidences are alarmingly high (World Health Organization, 2019), with insufficient PA (World Health Organization, 2018), excessive energy consumption (Statista, 2019) and genetic factors (Dalamaga et al, 2013) contributing to obesity. Obesity is linked to the development of conditions such as CVD (De Koning et al., 2007), T2DM (Nguyen et al., 2011), and dyslipidemia (Goossens et al., 2008), which contribute to premature mortality (Kithara et al., 2014). Surgical and pharmacological interventions have been implemented to treat and manage obesity, however, due to high cost and side effects these are not feasible to perform on a wide scale (Padwal et al., 2003; Rosenthal et al., 2006). Lifestyle interventions such as diet and exercise have proven effective in the reduction of obesity, and improvement of fitness levels (Noakes et al., 2000; Franz et al., 2007). Aerobic exercise has been extensively used to treat obesity and has proven effective in improving metabolic health (Christ-Roberts et al., 2004). There are limited studies pertaining to the effect of suspension training at treating and managing obesity, however the results are promising from studies that do exist (Dolati et al., 2017; Janot et al., 2013; Smith et al., 2016). Aerobic exercise and suspension training target different fitness qualities and so combining both into a single intervention could prove more effective at improving metabolic health and body composition. The effect of exercise on myostatin is not conclusive, as much of the research pertains to animals. Since myostatin is a novel biomarker of insulin resistance, the effect of exercise with respect to myostatin should be examined closely.

Chapter 3: Methodology

3.1 Participants

An email (Appendix 1) was sent to all staff and students in AIT with a copy of the plain language statement (Appendix 2) outlining the nature of the study. A private meeting was arranged with each potential participant that responded and all elements of the study were discussed in detail including the nature of the study, the tests involved, the interventions involved, and all risks and benefits associated with the study. The informed consent form (Appendix 3) was explained to the potential participants and participants were given an opportunity to ask any questions they had. Potential participants were advised to re-read the informed consent form in their own time and return a signed copy if they wished to take part in the study. Upon receiving the informed consent form, each participant then completed a physical activity readiness questionnaire (Appendix 4) to confirm their suitability for the study. Participants were then screened by having their height and weight measured in order to ensure their eligibility for the study, as per the inclusion criteria outlined below. Twenty three participants volunteered for the study. Of these individuals, 5 participants dropped out of the study due to scheduling commitments.

Inclusion criteria included any healthy, injury free males or females aged 18-50 years with a body mass index of 25-35 kg/m^2 and a history of physical inactivity for the previous 6 months. Physical inactivity was defined as not meeting the American College of Sports Medicines recommended 150 minutes of moderate to vigorous intensity exercise per week (Garber et al., 2011). Exclusion criteria included any musculoskeletal injury/disability that prevented participation in physical activities, any illness or condition that may affect the person's ability to perform maximal and near maximal exercise, any person suffering from uncontrolled hypertension, uncontrolled cardiovascular disease, and pregnant women. This study was approved by the Athlone Institute of Technology Ethics Committee and conformed to the Declaration of Helsinki.

3.2 Study Design

Baseline testing was completed over a one week period prior to commencing the 8-week exercise intervention. The testing days were structured as shown in Figure 3.1 below. A period of 48 hours was allocated between testing days and the exercise intervention commenced 48 hours following testing day 3. All testing occurred at the same time each

day to avoid any diurnal variation. Post-intervention data collection commenced 48 hours after ceasing the exercise intervention and occurred in the same order and same time of day as pre-testing. Due to scheduling issues, blood testing for one participant occurred ten days post intervention.

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┌─────────────────────────────────────────────────────────────┐
│                  Pre-intervention testing                     │
└─────────────────────────────────────────────────────────────┘

┌──────────────────┐  ┌──────────────────┐  ┌──────────────────┐
│     Day 1        │  │                  │  │     Day 3        │
│ Resting heart rate│ │                  │  │ Intravenous blood │
│ Blood pressure   │  │                  │  │     sample        │
│ Height           │  │     Day 2        │  │      FBG          │
│ Weight           │  │  VO2max Test     │  │ Fasting lipid     │
│ BIA              │  │                  │  │   profile         │
│ Body circumferences│ │                  │  │                  │
│ Sit and reach    │  │                  │  │                  │
│ TUG              │  │                  │  │                  │
│ Plank time trial │  │                  │  │                  │
│ 20m sprint       │  │                  │  │                  │
│ Isokinetic strength│ │                  │  │                  │
│ 3RM strength testing│ │                 │  │                  │
└──────────────────┘  └──────────────────┘  └──────────────────┘

┌─────────────────────────────────────────────────────────────┐
│                 8-week exercise intervention                  │
└─────────────────────────────────────────────────────────────┘

           ┌─────────────────────────────────────┐
           │      Post-Intervention Testing      │
           │       (Repeat of all pre-tests)     │
           └─────────────────────────────────────┘
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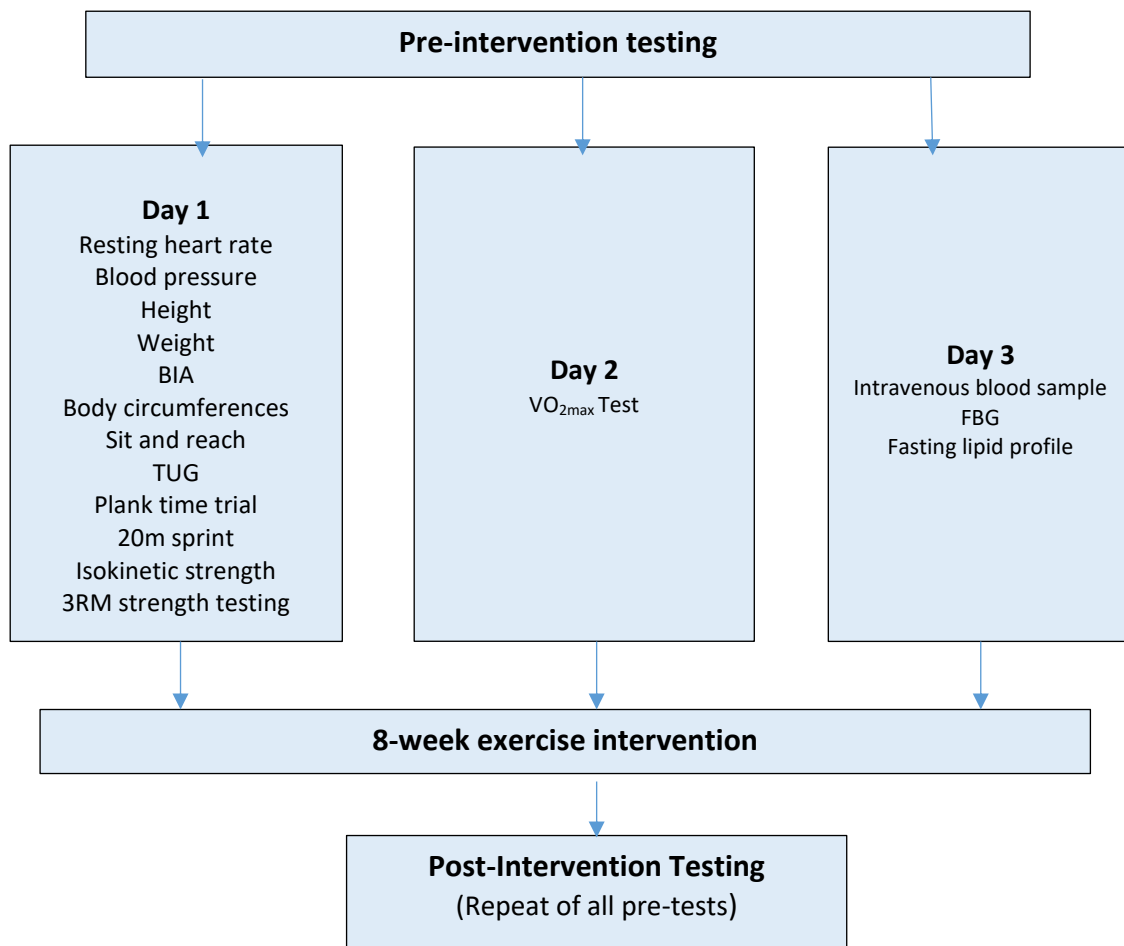
Figure 3.1: Order and Layout of Pre and Post-Intervention Testing.

3.3 Physical Characteristics
Physical characteristics investigated included bodyweight, BMI, body composition and circumference measurements and were measured on day 1 of baseline testing.

3.3.1 Height

Height was recorded after a deep inhalation to the nearest 0.1 cm using a stadiometer (SECA, Hamburg, Germany). Participants removed their shoes and stood against the backboard of the stadiometer with body weight evenly distributed and both feet flat on the platform.

3.3.2 Weight

Weight was measured to the nearest 0.1 kg using an electronic scale (SECA, Hamburg, Germany). Participants removed their shoes and any heavy items of clothing, and stood in the centre of the scale platform with weight evenly distributed.

3.3.3 Body Mass Index

Body mass index was calculated as: weight (kg) divided by height (m) squared (kg/m^2). The criteria for the current study included overweight individuals (BMI: 25-30 kg/m^2) and individuals with class I obesity (BMI: 30-35 kg/m^2) (Le et al., 2016).

3.3.4 Bio-electrical Impedance Analysis

Bio-electrical Impedance Analysis (BIA) was used to assess body fat in relation to lean body mass. Participants removed shoes and socks and stood in the centre of the scale platform (Model TBF-310, Tanita Corporation of America, Inc, Arlington Heights, IL). A low level, imperceptible electrical current was sent through the body. The BIA determined the resistance to flow of the current as it passed through the body, and then provided an estimate of percentage body fat and percentage muscle mass.

3.3.5 Body Circumferences

The participant stood in the anatomical position and a measuring tape was used to measure the circumferences of the hip, waist, upper arm, calf, and thigh to the nearest 0.1 cm. A description of the body circumference measurements used is provided in Table 3.1.

Table 3.1 Body Circumference Measurements (Santos et al., 2014).

Body part	Description
Arm	Mid-point of acromion on shoulder and lateral epicondyle of elbow.
Hip	Widest portion of buttocks, with tape parallel to floor.
Waist	Midpoint between lower margin of the last palpable rib and top of iliac crest.
Thigh	Midpoint between greater tuberosity and lateral condyle of femur.
Calf	The largest circumference.

3.4 Performance Testing

Following a warm-up, physical performance tests were performed to assess a wide variety of physical attributes including muscular strength, muscular endurance, aerobic endurance, dynamic balance, speed, and flexibility.

3.4.1 Warm-up Protocol

A 10-minute warm-up protocol, as described by Turki et al., (2012), was performed prior to the physical performance tests in order to minimise injury risk. The 10-minute dynamic stretching protocol involved 5 exercises, as outlined in Table 3.2 to stretch the lower body, and were all demonstrated by the lead researcher. Using a randomized selection procedure, the participants completed each of the 5 exercises for a period of two minutes. Participants were instructed to perform each exercise at a controlled speed. A rest period of 10 seconds was allowed between exercises before returning to the start position.

Table 3.2. Dynamic Warm-up Exercises (Turki et al., 2012)

Exercise	Description
Gluteals	The selected knee was lifted toward the chest, while walking and elevating the body on the toes of the opposite extended leg.
Hamstring Kick	While walking, the selected knee was extended outward while flexing the hip. The knee was extended until a stretch was felt in the hamstring muscle. Each leg was alternated.
Hurdlers Knee Raise	While traveling forward, the selected leg was raised and placed into an abducted and externally rotated position, and moved forward as though the participants were stepping over an object and returned to normal walking stride position.
Heel Ups	Heels were rapidly kicked toward buttocks while moving forward.
Tip-Toe Walking	While traveling forward alternating plantarflexion was performed (tip-toe) with every step. The aim was to raise the body as high as possible through tip-toeing.

3.4.2 Timed Up and Go (TUG).

The TUG test, depicted in Figure 3.2, was used to assess dynamic balance. The participant started in a seated position. Upon the testers command the participant stood up, walked 3 meters as quickly as possible, turned around a cone, walked back to the chair and sat down (Podsiadlo and Richardson 1991). The time stopped when the participant was seated.
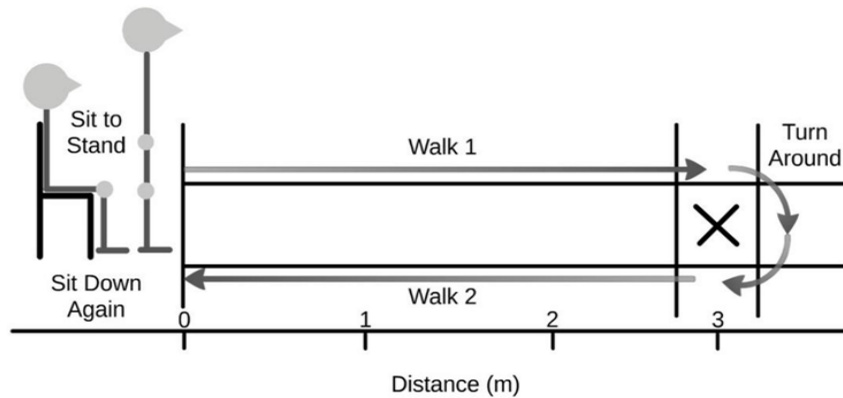
Figure 3.2: Diagram of the Timed up and Go test (Caballer, 2016).

3.4.3 Sit and Reach Testing

The sit and reach, as described by López-Miñarro et al., (2009) was used to assess flexibility of the legs and lower back. As seen in Figure 3.3, participants sat with their feet in contact with the testing box at approximately hip width apart. With both knees fully extended, the participants placed their right hand over their left and slowly slid their hands along the measuring board as far as they could. The distance reached was recorded to the nearest 1 cm.

Figure 3.3: Sit and Reach test

3.4.4 Plank Time Trial

The plank time trial is a measure of muscular endurance. The participant placed the forearms on the ground with the elbows aligned below the shoulders, and arms parallel to the body shoulder-width distance. The participant then raised their body off the ground on the go command (Learman et al., 2015). The hip was lifted off the floor

creating a straight line from head to toe, as shown in Figure 3.4. As soon as the participant was in the correct position, timing began for as long as the participant could maintain the correct position. The test was over when the participant was unable to hold the back straight and the hip was lowered.

Figure 3.4: Plank Time Trial

3.4.5 20 Metre Speed Test

Sprint speed was assessed using the 20 metre sprint test. The participant readied themselves on the start-line positioned behind the first timing gates (Brower Timing Systems, Draper, UT), in a standing split-stance start position. Each participant completed three maximal sprints, each separated by a 3-minute rest. Five, 10 and 20 metre split times were recorded, and the average of the three trials was used for analysis (Fletcher and Jones, 2004).

3.4.6 Isokinetic Strength Testing

Isokinetic knee extensors and flexors peak torque of both legs were measured using a Biodex System III dynamometer as seen in Figure 3.5 (Biodex Medical Systems, Shirley, New York). Isokinetic strength testing involves a muscular contraction applied against a dynamometer moving at a constant velocity (Baltzopoulos and Brodie, 1989). The force applied against the dynamometer is measured. Maximum force is measured (peak torque), and the ability to sustain this force over a number of repetitions (total work) is measured also. Movement velocity of isokinetic dynamometry can be changed in order to simulate different activities, with slower velocities being associated with absolute strength, and faster velocities being linked to explosive power (Baltzopoulos and Brodie,

1989). Peak torque of the knee extensors (KEPT) and peak torque of the knee flexors (KFPT) was recorded at 60°.s^{-1} and 300°.s^{-1} to simulate absolute strength and explosive power respectively. Participants were tested in a seated position with straps placed over their waist, both shoulders, and distal thigh for stabilisation. The tibial pad was positioned and secured approximately 3 cm proximal to the medial malleolus on the leg. One set of five maximal repetitions were performed at each speed per leg with 5 minutes rest been sets (Sandberg et al., 2012). Participants performed a self-selected number of trial repetitions prior to each speed. The total work performed in the exercise, and the highest peak torque value attained during the test was used for data analysis. Total work is measured in Joules, and calculated as the sum of work in all repetitions performed. The average value for both legs was calculated and used for statistical analysis (English et al., 2006).

Figure 3.5: Isokinetic Leg Extension and Flexion.

3.4.7 3-repetition Maximum (3RM) Strength Testing

The 3RM strength testing, as described McCurdy et al., (2004), was used to measure upper and lower body strength. The participant was instructed to complete a three part warm-up which began with a light resistance that easily allowed 5-10 repetitions of the exercise, followed by a 1-minute rest. The warm-up then progressed to a load that allowed the individual to complete 5-7 repetitions, followed by a 2-minute rest. Finally, a near maximum load that the participant could complete for 4-5 repetitions was performed, and 3 minutes rest was provided. Once the warm-up was completed, the load was then increased incrementally until the participant could complete only 3 repetitions with correct execution. Ideally this was achieved within 5 testing sets. This procedure was conducted for the leg press (Figure 3.6) and the chest press (Figure 3.7). This was conducted under the supervision of the lead researcher, a trained strength and conditioning professional.

Figure 3.6: The Leg Press exercise
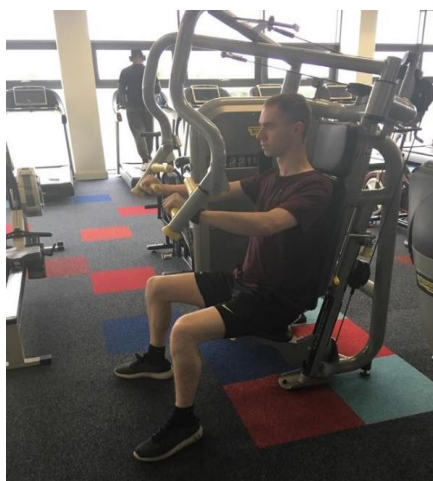
Figure 3.7: The Chest Press exercise

3.4.8 Maximum aerobic capacity (VO$_{2max}$)

A graded exercise protocol was used to identity participants' VO$_{2max}$ and was performed on day 2 of testing. A 5-minute warm-up was completed on a treadmill (HP Cosmos Pulsar, Hamburg, Germany) walking at 4 km/h for 5 minutes. The speed was then increased to 5 km/h for 3 minutes. Succeeding this the speed was increased to 6 km/h for 3 minutes. The grade was then increased by 2% every 3 minutes following this. During the test, heart rate was continuously monitored and recorded using a heart rate monitor (Polar T31 transmitter monitor, Polar Electro, Kempele, Finland). Submaximal

heart rate values were recorded once every 60 seconds. Rate of perceived exertion (RPE) was taken during the last ten seconds of each stage using the 6-20 point Borg scale (Appendix 8). Expired air was continuously analysed for O_2 and CO_2 using a metabolic cart (Moxus, AEI Technologies, Pittsburgh, PA) and mean values over 30s were recorded. Prior to each test the analyser was calibrated with gases of a known concentration (4% CO_2, and 16% O_2) and the pnemotach was calibrated with a 3 litre syringe. VO_{2max} was identified as the mean of the two VO_2 values in the final minute of the test (Loe et al., 2016). A plateau of VO_2 despite increased workload, a respiratory exchange ratio >1.00, a heart rate greater than or equal to age-predicted maximum and volitional exhaustion were used as criteria for reaching the VO_{2max} (Edvardsen et al., 2014). The participant then cooled down by walking slowly at a self-selected pace for 5 minutes followed by static stretching of the major muscle groups. Submaximal heart rate and submaximal VO_2 values were the average of the two readings per minute and were plotted against each other and heart rates corresponding to 65%, 70%, 65% and 80% VO_{2max} were then calculated using linear regression for use in training sessions.

3.5 Cardiovascular Characteristics

Cardiovascular characteristics investigated included resting heart rate, SBP, DBP, and blood lipids (TC, HDL cholesterol, LDL cholesterol, and TG). Blood lipid testing took place on testing day 3 of pre and post testing, in the 72 hours prior to the exercise intervention, and 72 hours post-intervention, with the exception of one participant whose testing occurred 10 days post intervention. Pre and post measurements were taken at the same time of day in order to avoid any diurnal variation, and participants were requested to fast for 10 hours prior to testing. Gloves were worn during blood analysis and all testing occurred in a designated sterile area of the sports science laboratory.

3.5.1 Resting Heart Rate

The sensor (electrode) area on the heart rate monitor (Polar T31 transmitter monitor, Polar Electro, Kempele, Finland) chest strap was moistened with water and placed firmly on the front of the lower chest of the participant with the elastic strap. Resting heart was recorded while sitting quietly for three minutes, and the value at the end of the three minutes was noted as resting heart rate.

3.5.2 Blood Pressure

The blood pressure monitor (Omron 705IT HEM-759-E, Omron Corporation, Kyoto, Japan) was attached to the upper arm of the participant and the participant was instructed not to talk for the duration of the monitoring. After a period of three minutes sitting quietly blood pressure was recorded.

3.5.3 Fasted Blood Lipids

A finger prick test was used to analyse fasted blood lipids. The fasting blood lipid profile included TC, HDL cholesterol, LDL cholesterol, and TG. The skin was pierced using a sterile, single use captive lancet. The first drop of blood was wiped away with an alcohol wipe and the second drop of blood was collected for analysis using a micro pipette (CardioChek PA SafeTec Pipettes, Polymer Technology Systems, Inc., Indianapolis, USA) then analysed using a CardioChek PA Analyser (Polymer Technology Systems, Inc., Indianapolis, USA).

3.6 Metabolic Characteristics

All fasted blood samples examining metabolic health were taken in a fasted state. This testing took place under the same conditions as blood lipid measurements as described in 3.5.

3.6.1 Fasted Blood Glucose (FBG)

A finger prick test was used to analyse fasted blood glucose. The skin was pierced using a sterile, single use captive lancet. The first drop of blood was wiped away with an alcohol wipe and the second drop of blood was collected for analysis using the Accu-Chek Performa (Roche Diagnostics, Basle, Switzerland) blood glucose meter. A plaster was placed on the piercing site following the testing.

3.6.2 Collection, Handling and Storage of Intravenous Blood Samples

Intravenous blood samples were taken to examine fasting plasma insulin and myostatin concentrations. Prior to the blood analysis participants had an intravenous catheter placed into a prominent forearm vein for blood sampling by a trained phlebotomist. Samples for fasting insulin and myostatin determination were collected in red top serum tubes (BD Vacutainer®). The red top vacutainers were allowed to stand for 20 minutes at room temperature before centrifugation (Dash, Drucker Diagnostics, USA) at 3000

rotations per minute for 15 minutes at 4°C. Following this process plasma aliquots were stored at -80°C for further analysis.

3.6.3 Elisa Analysis of Plasma Insulin and Myostatin

Commercially available ELISA kits were used to determine plasma insulin (Insulin Human ELISA Kit, Invitrogen, UK) and myostatin (GDF-8/Myostatin Quantikine ELISA Kit, R&D Systems, USA) concentrations according to the manufacturer's instructions. The optical density of each well was read within 30 minutes of assay completion in order to determine concentrations of plasma insulin and myostatin. Plasma samples were defrosted in the hour before beginning the assay.

3.7 Exercise Intervention

Following all baseline testing an 8-week intervention consisting of 4 training sessions per week was implemented. Each session was one hour in duration with two aerobic training sessions and two suspension training sessions completed per week. This consisted of a total of 160 minutes exercise per week, which was selected given the energy expensive eccentric nature, increased muscle damage and the potential for increased resting energy expenditure associated with suspension training, and to meet the American College of Sports Medicines recommended 150 minutes of moderate to vigorous intensity exercise for health. All session were instructed (suspension training) or supervised (aerobic training) by the principle investigator. On average all participants completed $94 \pm 2\%$ of all training sessions.

3.7.1 Aerobic Exercise Prescription

Each aerobic exercise session consisted of walking/jogging at a pre-determined heart rate zone based off a percentage of VO_{2max}. For weeks 1 and 2 participants exercised at a heart rate corresponding with 65% of VO_{2max}, at 70% VO_{2max} for weeks 3 and 4, at 75% VO_{2max} for weeks 5 and 6 and at 80% VO_{2max} for weeks 7 and 8. Each session consisted of a 10-minute warm-up, depicted in Table 3.2, involving dynamic exercises designed to reduce injury risk during the session. This was followed by the main body of the exercise session which lasted 40 minutes. Each session then ended with a cool down period of 10 minutes involving slow walking and static stretches of all muscle groups held for a period of 20 seconds each. Participants wore a heart rate monitor for each aerobic training session, and were informed of their target heart rate prior to each session. This

was monitored by the principal investigator throughout all training sessions. If participants' heart rates were lower than required, the participant was instructed to increase speed in order to achieve the desired heart rate. Likewise, if the participants heart rate was higher than required, then the participant was instructed to decrease speed in order to achieve the desired heart rate.

3.7.2 Suspension Training Prescription

Similar to the aerobic training sessions, each suspension training session commenced with a 10-minute warm-up involving dynamic exercises. This was followed with 40 minutes of specific suspension training, and a 10-minute cool down involving slow walking and static stretches. The 40-minute main body of the session consisted of a circuit of 10 exercises incorporating upper body, lower body and core musculature, and this was completed four times. Each set involved performing repetitions of an exercise for 45 seconds, followed by a 15-second rest. The suspension training session was conducted at a 15 (hard) to 17 (very hard) rate of perceived exertion using the 6-20 point Borg scale (Appendix 5). If the participants RPE was above or below the desired range, the exercise was adapted by the lead researcher in order to achieve the desired level of difficulty. The participants were informed of the target RPE before the training session. A full list and description and all suspension training classes is provided in Appendix 5.

3.8 Post Intervention Testing

Post intervention testing commenced 48 hours following the exercise intervention and all testing procedures were performed within 7 days, with the exception of one participant whose blood testing occurred 10 days post intervention. A period of 48 hours was allocated between testing days. All testing occurred at the same time and in the same order as pre intervention testing.

3.9 Statistical Analysis

All data was analysed using the IBM SPSS 25 statistical package (IBM Software, Inc. Chicago, IL) with statistical significance set at $P < 0.05$. Mean and standard deviations were calculated for all variables. For non-normally distributed variables median and interquartile ranges were calculated. Normality of all data was tested using the Shapiro-Wilk test. Paired samples t-tests were used to investigate the effects of the intervention

with normally distributed variables values, while Wilcoxon signed-rank test was used with non-normally distributed variables.

Pearson correlations were used to measure the strength of relationship between two normally distributed variables, while Spearman's correlation was used for non-parametric variables. Percentage change in metabolic and cardiovascular variables were correlated against percentage changes in physical, and performance variables. Correlations were performed in a step by step approach whereby each individual metabolic and cardiovascular variable were correlated separately against physical and performance variables. A Bonferroni correction was conducted to reduce the likelihood of a type I error occurring (Vasilopoulos et al., 2016). Correlations were interpreted as r = 0.00 to 0.09 (trivial), 0.1 to 0.29 (small), 0.30 to 0.49 (moderate), 0.50 to 0.69 (strong), 0.70 to 0.89 (very strong), 0.90 to 0.99 (nearly perfect), 1.0 (perfect) (Hopkins, 2000). Statistical significance was set at $P < 0.05$. Cohen's d effect size was used to measure the magnitude of the effect of the exercise intervention on the measured variables. Cohen's d effect size can be interpreted as; $d \leq 0.2$ = 'small' effect size, $d \leq 0.5$ = 'medium' effect size, and $d \leq 0.8$ = 'large' effect size (O'Sullivan and Feinn, 2012).

A multiple backwards stepwise linear regression was used to obtain a model to predict the change in the metabolic variables from the changes in physical performance, anthropometric and physiological variables. Multiple backwards stepwise regression eliminates each independent variable one by one until it ultimately determines the independent variable or variables that most significantly predicts the percent change in the dependent variable. To avoid multi-collinearity and to decide which variables to include in the regression analysis, Pearson correlation, Spearman's correlation, variance inflation factor and tolerance statistics were utilised whereby all variables had to have a variance inflation factor of less than 10, and a tolerance statistic of greater than 0.1 (Field, 2005). If two variables showed high inter-correlation ($r > 0.80$) only one was entered into regression analysis (Field, 2005). The variable entered into the analysis was determined by identifying the variable which had the greatest effect on the regression analysis in terms of R^2 and coefficient β. A variable was removed from the analysis when it had little effect on the predictive capabilities of that regression analysis. The

probability of change is indicated by R^2 with an R^2 of 1.0 indicating a perfect prediction model. The strength of each regression analysis was assessed using an ANOVA and statistical significance was set at $P < 0.05$. Independent variables that were not significant predictors were removed from the model. Coefficient β was also used to assess the strength of the effect of each individual independent variable on the dependant variable in that model (Freedmann, 2009).

Chapter 4: Results

4.1 Physical Characteristics

Table 4.1 presents the physical characteristics of all eighteen participants (N = 2 male, N = 16 female; age: 37 ± 11 years). A paired samples t-test revealed significant improvements in BMI, body weight, percentage body fat, percentage muscle mass, as well as waist, hip, arm circumferences, and WHR.

Table 4.1: Descriptive characteristics pre and post-intervention.

Variable	Pre	Post	% Change	D	P Values
BMI (kg/m^2)	31.26 ± 5.61	30.45 ± 5.94	-2.95 ± 2.95	0.14	0.02
Weight (kg)	86.8 ± 17.6	85.3 ± 18.3	-1.80 ± 2.40	0.08	0.03
Height (m)	1.66 ± 7.50	1.66 ± 7.50	0.00 ± 0.00	0.00	0.10
BF (%)	37.2 ± 8.1	34.5 ± 8.9	-7.90 ± 9.6	0.31	0.001
Muscle (%)	58.8 ± 8.6	61.5 ± 6.7	4.5 ± 0.55	0.35	0.001
Thigh (cm)	60.34 ± 7.9	59.17 ± 7.6	-2.07 ± 3.36	0.15	0.23
Waist (cm)	90.5 ± 13.9	86.1 ± 12.7	-5.05 ± 6.6	0.33	0.003
Hip (cm)	111.3 ± 13.1	108.9 ± 12.5	-2.16 ± 2.02	0.18	0.001
Arm (cm)	33.1 ± 5.2	32.1 ± 4.5	-3.02 ± 4.31	0.20	0.009
Calf (cm)	41.1 ± 5.1	39.7 ± 3.6	-3.41 ± 7.04	0.31	0.35
WHR	0.83 ± 0.07	0.78 ± 0.06	-6.50 ± 3.40	0.61	0.002

Data is presented as mean ± standard deviation. Note: BMI = Body mass index, BF = percentage body fat, Muscle = percentage muscle mass, WHR = waist to hip ratio.

4.2 Physical Performance Characteristics

The physical performance characteristics (N = 18) pre and post-intervention are displayed in Table 4.2. Significant reductions were observed in TUG, 5m, and 10m and 20m sprint times while significant increases were noted in VO$_{2max}$, and plank time trial, leg press and chest press 3RM.

Table 4.2: Physical performance characteristics pre and post-intervention.

Variable	Pre	Post	% Change	D	P values
TUG (s)	4.71 ± 0.55	3.87 ± 0.55	-17.31 ± 7.1	1.52	0.001
5m sprint (s)	1.42 ± 0.26	1.30 ± 0.20	-8.45 ± 0.27	0.51	0.038
10m sprint (s)	2.45 ± 0.31	2.31 ± 0.27	-5.71 ± 0.5	0.48	0.03
20m sprint (s)	4.56 ± 0.52	4.27 ± 0.50	-7.8 ± 6.6	0.56	0.001
VO_{2max} (ml/kg/min)	22.73 ± 5.4	26.29 ± 5.80	19.3 ± 26	0.62	0.03
Plank time trial (s)	65.1 ± 29.2	123.9 ± 54.8	90.32 ± 8.9	1.33	0.001
Leg Press 3RM (kg)	125 ± 33	155 ± 33	24 ± 18	0.90	0.001
Chest Press 3RM (kg)	35 ± 17	40 ± 18	14.2 ± 0.2	0.28	0.001

Data is presented as mean ± standard deviation. Note: TUG = timed up and go, 3RM = three repetition maximum.

4.3 Isokinetic strength testing

The isokinetic strength testing data (N = 18) is presented in Table 4.3. A Wilcoxon signed-rank test noted significant increases $60°.s^{-1}$ KEPT, $60°.s^{-1}$ KFPT, and $60°.s^{-1}$ KETW. A paired samples t-test revealed that there was significant increases in $60°.s^{-1}$ KFTW, $300°.s^{-1}$ KEPT, $300°.s^{-1}$ KFPT, $300°.s^{-1}$ KETW, and $300°.s^{-1}$ KFTW post intervention. Percentage improvements in isokinetic strength measures are displayed in Figure 4.1.

Table 4.3: Isokinetic strength testing pre and post-intervention

Variable	Pre	Post	D	P Values
$60°.s^{-1}$ KEPT (N)	140.5 (106.8-168.6)	178 (140.5-198.6)	0.63	0.001
$60°.s^{-1}$ KFPT (N)	80.7 (59.25-104)	106.2 (82-127.7)	0.87	0.003
$60°.s^{-1}$ KETW (J)	580.6 (475-627)	684.7 (536-767)	0.50	0.001
$60°.s^{-1}$ KFTW (J)	376.3 (253-475)	434.3 (343-514)	0.50	0.012
$300°.s^{-1}$ KEPT (N)	75.8 (56-88)	100.1 (80-116)	1.09	0.001
$300°.s^{-1}$ KFPT (N)	60.1 (46-77)	83.2 (63-99)	1.16	0.001
$300°.s^{-1}$ KETW (J)	333.2 (237-412)	401.6 (301-468)	0.53	0.001
$300°.s^{-1}$ KFTW (J)	213.7 (166-281)	281.6 (223-342)	0.79	0.001

Data is presented as mean (range). Note: $60 °.s^{-1}$ KEPT = $60°.s^{-1}$ knee extension peak torque, $60°.s^{-1}$KEFT = $60°.s^{-1}$ knee flexion peak torque, $60°.s^{-1}$ KETW = $60°.s^{-1}$ knee extension total work, $60°.s^{-1}$ KFTW = 60°/s knee flexion total work, $300°.s^{-1}$ KEPT = $300°.s^{-1}$ knee extension peak torque, $300°.s^{-1}$ KFPT = $300°.s^{-1}$ flexion peak torque, $300°.s^{-1}$ KETW = $300°.s^{-1}$ knee extension total work, $300°.s^{-1}$ KFTW = $300°.s^{-1}$ knee flexion total work
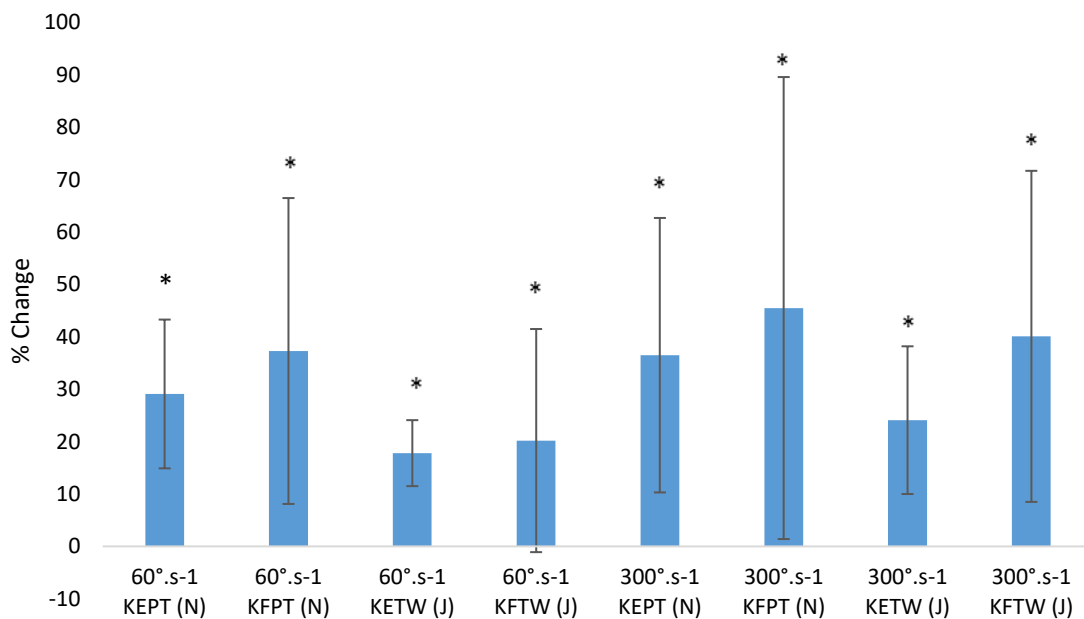
Figure 4.1 Percent change in isokinetic leg extension and flexion. Data presented as mean ± SD. *P < 0.05. Note: $60°.s^{-1}$ KEPT = $60°.s^{-1}$ knee extension peak torque, $60°.s^{-1}$ KEFT = $60°.s^{-1}$ knee flexion peak torque, $60°.s^{-1}$ KETW = $60°.s^{-1}$ knee extension total work, $60°.s^{-1}$ KFTW = $60°.s^{-1}$ knee flexion total work, $300°.s^{-1}$ KEPT = $300°.s^{-1}$ knee extension peak torque, $300°.s^{-1}$ KFPT = $300°.s^{-1}$ flexion peak torque, $300°.s^{-1}$ KETW = $300°.s^{-1}$ knee extension total work, $300°.s^{-1}$ KFTW = $300°.s^{-1}$ knee flexion total work.

4.4 Metabolic and Cardiovascular Characteristics

Table 4.4 presents the fasting metabolic characteristics of the participants (N = 18) pre and post-intervention. Significant decreases were noted in LDL cholesterol, TC:HDL ratio, resting heart rate, and DBP after a paired samples t-test was performed. A Wilcoxon signed-rank test revealed a significant reduction in FBG, TC, TG, and myostatin. This was accompanied by a significant increase in fasting HDL cholesterol. No significant difference was noted for fasting insulin concentrations post intervention. Percentage changes for metabolic characteristics are represented in Figure 4.2.

Table 4.4: Metabolic and cardiovascular characteristics pre and post-intervention

Variable	Pre	Post	d	P Values
FBG (mmol/L)	5.27 (5-5.6)	4.74 (4.3-5.1)	1.40	0.001
TC (mmol/L)	4.64 (4.25-5.25)	4.34 (4.1-5)	0.50	0.001
HDL (mmol/L)	1.09 (0.7-1.26)	1.32 (1.09-1.67)	0.95	0.001
LDL (mmol/L)	2.47 (2.05-2.5)	2.38 (1.99-2.4)	0.18	0.04
TC:HDL Ratio	4.1 (3.4-5.1)	3.2 (2.5-4.2)	0.72	0.002
TG (mmol/L)	1.75 (1.3-2.1)	1.56 (1.2-1.9)	0.52	0.001
RHR (bpm)	89 (77-101)	79 (71-95)	0.74	0.01
SBP (mmHg)	131 (122-140)	127 (118-131)	0.33	0.100
DBP (mmHg)	86 (79-91	83 (80-87)	0.42	0.015
Insulin (pmol/L)	178 (173-191	176 (172-189)	0.19	0.51
Myostatin (pg/ml)	3419 (1264-8588)	2726 (1220-7300	0.41	0.04

Data is presented as mean (range). Note: FBG = fasting blood glucose, TC = total cholesterol, HDL = high density lipoprotein cholesterol, LDL = low density lipoprotein cholesterol, TG = triglycerides, RHR = resting heart rate, SBP = systolic blood pressure, DBP = diastolic blood pressure.
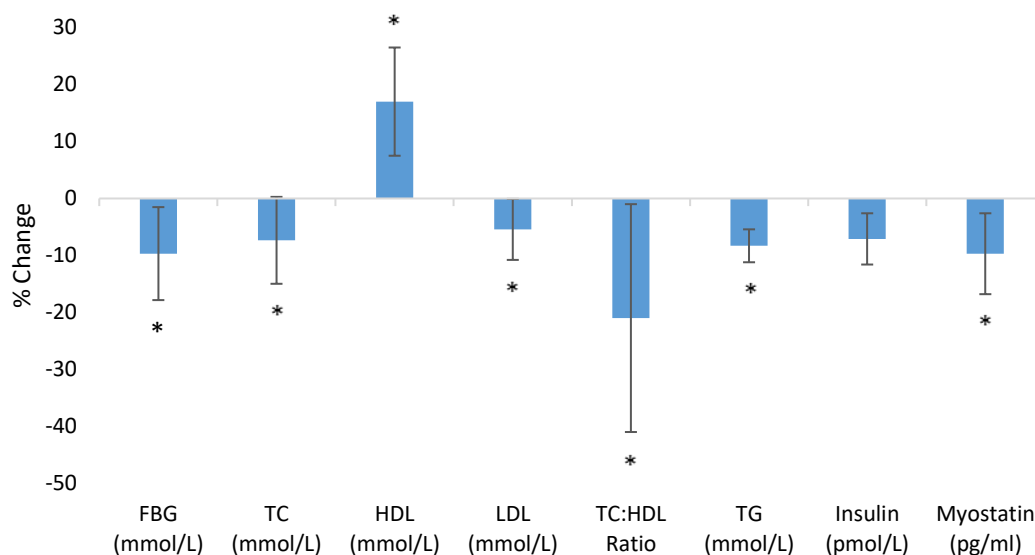
Figure 4.2: Percent change in metabolic health and blood lipid characteristics. Data presented as mean ± SD. *P < 0.05. Note: FBG = fasting blood glucose, TC = total cholesterol, HDL = high density lipoprotein cholesterol, LDL = low density lipoprotein cholesterol, TG = triglycerides.

4.5 Pearson's and Spearman's Correlation summary

The results of the correlation analysis which correlated percentage change in metabolic characteristics against percentage change in physical and performance characteristics, are presented in Table 4.5. This analysis revealed that the percent change in FBG following the exercise interventions had a very strong, significant correlation with both percentage body fat (P = 0.001) and 20m sprint (P = 0.001). No other significant correlations were noted.

Table 4.5: Pearson and Spearman's correlations between physical and metabolic characteristics.

	FBG	TC	HDL	LDL	TC:HDL	TG	INS	MSTN
BMI	r = 0.216	r = 0.081	r = 0.108	r = 0.010	r = -0.422	r = 0.043	r = 0.091	r = 0.323
Weight	r = 0.154	r = 0.036	r = 0.243	r = 0.133	r = 0.275	r = 0.249	r = -0.023	r = -0.002
BF	r = 0.806*	r = 0.029	r = -0.682	r = 0.490	r = -0.273	r = 0.260	r = -0.012	r = 0.048
%Muscle	r = 0.167	r = 0.145	r = 0.347	r = 0.125	r = -0.111	r = 0.113	r = 0.039	r = -0.038
Waist circumference	r = 0.217	r = 0.107	r = -0.351	r = 0.206	r =- 0.308	r = 0.410	r = 0.032	r = -0.031
Hip circumference	r = 0.032	r = 0.313	r = -0.215	r = 0.255	r = 0.341	r = 0.100	r = 0.200	r = 0.326
Thigh circumference	r = 0.243	r = 0.206	r = -0.119	r = 0.297	r = 0.116	r = 0.049	r = 0.401	r = 0.152
Arm circumference	r = 0.167	r = 0.062	r = -0.348	r = 0.340	r = 0.106	r = 0.147	r = 0.040	r = 0.115
Calf circumference	r = 0.167	r = 0.326	r = -0.192	r = 0.040	r = 0.303	r = 0.336	r = 0.034	r = 0.112
WHR	r = 0.265	r = -0.185	r = -0.190	r = 0.013	r = -0.257	r = -0.220	r = -0.090	r = 0.618
5m	r = 0.122	r = 0.201	r = -0.333	r = 0.104	r = 0.297	r = 0.183	r = 0.221	r = 0.025
10m	r = 0.131	r = 0.145	r = -0.050	r = 0.185	r = 0.335	r = 0.030	r = 0.296	r = 0.153
20m	r = 0.838*	r = 0.126	r = -0.612	r = 0.462	r = 0.205	r = 0.230	r = -0.299	r = 0.188
VO$_{2max}$	r = 0.68	r = 0.249	r = 0.355	r = 0.096	r = 0.186	r = 0.338	r = 0.183	r = -0.485
Plank time trial	r = 0.161	r = 0.303	r = 0.108	r = 0.149	r = 0.315	r = 0.343	r = 0.052	r = 0.336
TUG	r = 0.121	r = 0.213	r = 0.162	r = 0.108	r = 0.146	r = 0.342	r = 0.303	r = 0.112
Leg Press 3RM	r = 0.112	r = 0.902	r = 0.225	r = 0.225	r = -0.087	r = 0.161	r = 0.388	r = -0.165
Chest Press 3RM	r = 0.102	r = 0.296	r = 0.184	r = 0.049	r = 0.221	r = 0.043	r = 0.317	r = -0.053
60°.s^{-1} KEPT	r = 0.004	r = 0.369	r = 0.192	r = 0.192	r = -0.319	r = 0.031	r = -0.029	r = -0.411
60°.s^{-1} KFPT	r = 0.277	r = 0.503	r = 0.062	r = 0.496	r = -0.429	r = 0.198	r = 0.176	r = -0.163
60°.s^{-1} KETW	r = 0.107	r = 0.021	r = 0.221	r = 0.112	r = -0.095	r = 0.147	r = -0.205	r = -0.207
60°.s^{-1} KFTW	r = 0.213	r = 0.258	r = 0.030	r = 0.292	r = -0.200	r = 0.005	r = 0.105	r = -0.040
300°.s^{-1} KEPT	r = 0.219	r = 0.039	r = 0.131	r = 0.169	r = -0.049	r = 0.303	r = 0.119	r = -0.182
300°.s^{-1} KFPT	r = 0.032	r = 0.390	r = 0.550	r = 0.297	r = -0.538	r = 0.145	r = 0.204	r = -0.335
300°.s^{-1} KETW	r = 0.120	r = 0.336	r = 0.221	r = 0.340	r = -0.373	r = 0.116	r = -0.100	r = -0.032
300°.s^{-1} KFTW	r = 0.025	r = 0.152	r = 0.326	r = 0.243	r = -0.297	r = 0.206	r = 0.140	r = -0.106

r represents the strength of the relationship between two variables. *$P < 0.05$ is significant. Note: BMI = Body mass index, BF = % body fat, Muscle = % muscle mass, RHR = resting heart rate, SBP = systolic blood pressure, DBP = diastolic blood pressure, TUG = timed up and go, 3RM = three repetition maximum, FBG = fasting blood glucose, TC= total cholesterol, HDL= high density lipoprotein cholesterol, LDL= low density lipoprotein cholesterol, TG = triglycerides, 60°.s^{-1} KEPT = 60°.s^{-1} knee extension peak torque, 60°.s^{-1} KEFT = 60°.s^{-1} knee flexion peak torque, 60°.s^{-1} KETW = 60°.s^{-1} knee extension total work, 60°.s^{-1} KFTW = 60°.s^{-1} knee flexion total work, 300°.s^{-1} KEPT = 300°.s^{-1} knee extension peak torque, 300°.s^{-1} KFPT = 300°.s^{-1} flexion peak torque, 300°.s^{-1} KETW = 300°.s^{-1} knee extension total work, 300°.s^{-1} KFTW = 300°.s^{-1} knee flexion total work, INS = fasting insulin, MSTN = myostatin.

4.6 Multiple backwards stepwise regression

Following analysis of Pearson's and Spearman's correlations, variance inflation factor and tolerance statistics, multi-collinearity was identified and the following parameters were removed from the regression analysis: percentage muscle mass, thigh, waist, hip, arm, and calf circumferences, BMI, $60°.s^{-1}$ KEPT, $60°.s^{-1}$ KFPT, $60°.s^{-1}$ KFTW, $300°.s^{-1}$ KEPT, and $300°.s^{-1}$ KFTW.

Stepwise backward multiple regression analysis was performed with the following variables: $60°.s^{-1}$ KETW, body weight, percentage body fat, leg press 3RM, TUG, chest press 3RM, VO_{2max}, 20m sprint, $300°.s^{-1}$ KFPT, WHR, and plank time trial with FBG, TC, HDL, LDL, TC:HDL, TG, fasting insulin and myostatin the independent variables for each of the respective analyses. Results of the analysis can be seen in table 4.6.

Backwards stepwise regression revealed that the percent change in 20m sprint was the most significant contributor to the percent change in FBG and accounted for 70.2% of the variance. 20m sprint also had the greatest influence on FBG with a standardised coefficient β of 0.838. The percentage chance in TC was significantly predicted by $60°.s^{-1}$ KFPT, which was responsible for 25.3% of the variance in TC post intervention. $60°.s^{-1}$ KFPT was identified as the parameter with the greatest influence on TC with a standardised coefficient β of -0.503. The results indicate that the percent change in LDL was significantly predicted by $60°.s^{-1}$ KFPT which accounted for 24.6% of the variance. $60°.s^{-1}$ KFPT also had the greatest influence on LDL with a standardised coefficient β of -0.496. Backwards stepwise regression revealed that the percent change in VO_{2max}, weight, and $300°.s^{-1}$ KFPT were the most significant contributors to the percent change in HDL and accounted for 70.1% of the variance. VO_{2max} was identified as the parameter with the greatest influence on HDL with a standardised coefficient β of 0.585. The percent change in VO_{2max} was the most significant contributors to the percent change in TG and was responsible for 30.3% of the variance. VO_{2max} also had the greatest influence on TG with a standardised coefficient β of 0.551. Plank time trial, WHR, and chest press 3RM significantly predicted 82.3% of the variance in myostatin post intervention. WHR was identified as the parameter with the greatest influence on myostatin with a

standardised coefficient β of -0.653. TC:HDL, and fasting insulin showed no significant relationship to any variable.

Table 4.6 Backwards stepwise regression analysis.

Dependant Variable	Independent Variables	B	Standard Error	*B*	P Value
FBG	Constant	41.431	5.134		
	20m sprint	1.322	0.209	0.838	0.001
R^2 = 0.702; F = 40.027; P = 0.001*					
TC	Constant	50.151	11.763		
	$60°.s^{-1}$ KFPT	-0.567	0.252	-0.503	0.039
R^2 = 0.253; F = 5.086; P = 0.039*					
LDL	Constant	17.268	3.273		
	$60°.s^{-1}$ KFPT	-0.176	0.080	-0.496	0.043
R^2 = 0.246; F = 4.907; P = 0.043					
HDL	Constant	22.890	46.304		
	VO_{2max}	0.411	0.100	0.585	0.001
	Weight	1.643	0.799	-0.286	0.047
	$300°.s^{-1}$ KFPT	-0.126	0.044	0.401	0.014
R^2 = 0.701; F = 13.961; P = 0.001*					
TG	Constant	11.028	2.006		
	VO_{2max}	0.176	0.069	0.551	0.022
R^2 = 0.303; F = 6.525; P = 0.022*					
Myostatin	Constant	31.674	3.725		
	Chest press 3RM	-0.603	0.139	-0.572	0.001
	WHR	-2.536	0.470	-0.653	0.001
	Plank	0.100	0.027	0.507	0.003
R^2 = 0.823; F = 20.59; P = 0.001*					

R^2 represents the extent to which the change in 20m sprint predicted the change in the FBG. P < 0.05 is significant. Note: FBG = fasting blood glucose, $60°.s^{-1}$ KFPT=$60°.s^{-1}$ knee flexion peak torque, TC=total cholesterol, LDL = low density lipoprotein cholesterol, $300°.s^{-1}$ KFPT = $300°.s^{-1}$ knee flexion peak torque, HDL= high density lipoprotein cholesterol, chest press 3RM = chest press three repetition maximum, WHR = waist hip ratio, plank = plank time trial, TG = triglycerides.

Chapter 5: Discussion

5.1 Introduction

The main findings of this study were that a combined aerobic and suspension training intervention resulted in improvements in physical, performance, metabolic, and cardiovascular characteristics of overweight and obese sedentary individuals.

Combining exercise modes into a singular intervention appears to be a more effective method of improving body composition for overweight and obese individuals than interventions involving one exercise mode in isolation (Willis et al., 2012). Previous research has shown that combining exercise modes, especially when one of these training modes emphasises eccentric muscle contractions, may facilitate chronic increases in resting metabolic rate resulting in increased overall energy expenditure in overweight and obese individuals (Willis et al., 2012; Moon et al., 2018, Franklin et al., 2000). This may potentially result in improved loss of fat mass and greater increases in metabolic health over a period of time (Wilson et al., 2012). Aerobic exercise contributes to weight loss and improvements in body composition (Donnelly et al., 2013), cardiac function (Lee and Oh, 2016), mitochondrial size, number, and function (Miller et al., 2011), enzyme activity (Maltais et al., 1996), blood glucose utilisation (Christ-Roberts et al., 2004), blood lipid parameters (Leon and Sanchez, 2001), and fat oxidation capacity (Jeukendrup, 2002), while also reducing hypertension (Cornelissen et al., 2005) and insulin resistance (Duncan et al., 2003). Resistance training meanwhile can lead to improvements in insulin resistance, resting metabolic rate, glucose metabolism, blood pressure, body fat and strength levels (Winett and Carpinelli, 2001), while suspension training can elicit significant improvements in body composition (Dolati et al., 2017), muscular strength (Janot et al., 2013), muscular endurance (Dolati et al., 2017), balance ability (Janot et al., 2013), systolic and diastolic blood pressure (Smith et al., 2016), and VO_{2max} levels (Dolati et al., 2017). Suspension training has previously shown to also increase muscular activation compared with standard resistance training exercises of the same type (Mok et al., 2015; Snarr et al., 2014). The current study combined suspension training with aerobic training. This combination of exercise modes may potentially be a more effective training intervention in terms of improving physical, metabolic, and cardiovascular health than the standard concurrent aerobic and resistance training structure typically examined in much of the scientific literature as it

capitalises on all of the known positive adaptations to aerobic exercise and resistance training and possibly augments them due to the high eccentric component associated with suspension training.

5.2 Physical Characteristics

In the current study, the intervention resulted in a significant improvement in physical characteristics investigated, indicating the effectiveness of the training intervention at improving bodyweight, BMI, body composition, and circumference measurements.

Physical characteristics monitored during the current study included bodyweight, BMI, percentage body fat, percentage muscle mass, and circumference measurements.

5.2.1 Bodyweight and BMI

The current study resulted in a 1.5 kg (d = 0.08) decrease in bodyweight. In terms of body weight, 0.7 to 0.8 kg decreases have been reported post 8-week suspension training intervention involving three sessions per week for eight weeks in overweight participants (Smith et al., 2016; Dolati et al., 2017), however Janot et al., (2013) reported no difference in bodyweight following three sessions per week for 7 weeks on healthy individuals. Janot et al., (2013) had no direct measure of body composition but it could be inferred that any losses in body fat were potentially offset by gains in lean muscle mass causing weight to remain stable. Meta-analyses have previously identified that aerobic training interventions of 4-12 weeks, with frequencies of 3-5 sessions per week, at exercise of intensities of 60–75% of maximum heart rate or 50–65% of VO_{2max} resulted in a 2 kg decrease in bodyweight (Wewege et al., 2017) while combined aerobic and resistance training incorporating the same frequency of sessions over an 8-12 week period resulted in 2.3 kg reduction (Schwingshackl et al., 2013). The meta-analysis by Schwingshackl et al., (2013) involved 15 trials of between 10 weeks and 6 months in duration, intensities of 50-75% VO_{2max} or 60-85% maximum heart rate. The meta-analysis mentioned above included an aerobic intervention by Sawyer et al., (2016) involving 8 weeks of 3 sessions per week that resulted in no change in weight post intervention. Likewise, an 8-week combined aerobic and resistance training intervention observed no change to bodyweight following 3 sessions a week for 8 weeks. The 1.5 kg decrease in bodyweight, observed in the current study is within the ranges observed in

the literature mentioned above. This can have clinically significant benefits for participants as reductions in bodyweight in this population is correlated with improvements in insulin sensitivity and other health parameters (Carey et al., 1996, Redinger, 2007),

In the current study BMI was reduced by 0.8 kg/m^2 (d = 0.14). Body mass index was reduced by 0.4 kg/m^2 with suspension training three times per week for 8 weeks (Dolati et al., 2017), while aerobic training four times per week for 8 weeks resulted in a 2.61 kg/m^2 decrease in BMI (Suman, 2016) and combined aerobic and resistance training resulted in a 1.3 kg/m^2 decrease in BMI as a result of training 4 times per week for 8 weeks (Sheikholeslami-Vatani et al., 2015). The reduction in BMI in the current study was due to a reduction in body fat. Since percentage muscle mass was significantly increased in the current study post intervention, it was the significant reduction in percentage body fat that caused the decrease in BMI. Reductions in BMI post exercise training intervention, particularly when the weight loss is due to reductions in fat mass, is associated with significant improvements in metabolic and cardiovascular health, as well as mortality in this population (Borrell and Samuel, 2014; De Koning et al., 2010)

5.2.2 Body composition

Following the 8-week intervention in the current study, significant improvements were observed in percentage body fat (-2.8 kg, d = 0.31), and percentage muscle mass (1.3 kg, d = 0.55), with small and moderate effect sizes for percentage body fat and percentage muscle mass. Decreasing percentage body fat has substantial positive implications for health in overweight and obese individuals including decreased risk of developing; hypertension (Hall et al., 2015), dyslipidaemia (Feingold and Grunfeld, 2015), CVD (De Koning et al., 2007), T2DM (Nguyen et al., 2011) and consequentially, mortality (Staiano et al., 2005). Likewise, the WHR and WC were significantly improved with large (d = 0.61) and moderate (d = 0.33) effect sizes respectively. De Koning et al., (2010) observed that an increase in WC significantly increased an individual's risk of developing T2DM. This is due to insulin resistance as a result of increased secretion of cytokines and macrophages from adipose tissue which initiate signalling cascades that dysregulate carbohydrate and lipid metabolism by impairing insulin signalling, and thus contribute to the development of insulin resistance, T2DM, and cardiovascular disease (Jung and Choi, 2014).

Furthermore, a correlation has been observed between obesity and ectopic lipid stores also (Kankaanpää et al., 2006), and by a reduction of WC such as in the current study, risk of developing this condition is reduced. The lowering of the WHR also has clinical significance as elevations of this metric have been associated with higher TG levels, higher 2-hour glucose levels, elevated SBP, and higher risk of T2DM (Emdin et al., 2017).

During an eccentric contraction, skeletal muscle is forcibly stretched under tension which induces damage to cellular components and activates signalling pathways that promote muscle hypertrophy (Schoenfeld, 2011). As eccentric contractions produce higher levels of mechanical tension than concentric contractions, muscular damage is more pronounced resulting in muscular tenderness or soreness known as delayed onset muscle soreness (DOMS) (MacIntyre et al., 1995). Suspension training incorporates an extensive eccentric component, which magnifies DOMS post training. Post DOMS the myocytes adapt, and increase in size (Schoenfeld et al., 2015). This can result in an increase in overall muscle mass such as was observed in the current study. Increasing muscle mass has been associated with elevated resting metabolic rate (Dolezal and Potteiger, 1998), which accounts for a large percentage of an individual's total daily energy expenditure. Therefore, a small increase in resting metabolic rate can result in a substantial increase in total energy expenditure over time. Increasing resting energy expenditure is essential for obese individuals as this can contribute to the energy deficit required for fat loss.

The magnitude of change in body composition post training intervention can vary hugely between exercise modes, due to differences in modes of exercise incorporated in the intervention in terms of frequency, intensity and time. Suspension training three times per week for 8 weeks resulted in 1.5-5% improvements in body fat percentage in both healthy, and overweight participants (Smith et al., 2016, Dolati et al., 2017), whereas a meta-analysis by Wewege et al., (2017), observed a 6% improvement in percentage body fat in overweight in obese participants as a result of aerobic training 3-5 times per week, at exercise of intensities of 60–75% of maximum heart rate or 50–65% of VO_{2max} for 4-12 weeks. Among the studies included in the meta-analysis was an 8-week aerobic training intervention consisting of 3 session per week by Sawyer et al., (2016), which

resulted in a 1% improvement in body fat percentage. Combined aerobic and resistance training interventions have resulted in 2-4.3% improvements in body fat percentage in overweight and obese participants (Maiorana et al., 2002; Ho et al., 2012; Cervantes Sanabria & Hernandez-Elizondo, 2017, Willis et al., 2012) with training interventions of 8-week to 8-month durations, and exercise frequencies of 3-5 times per week. The current exercise intervention elicited a significant 7.9% improvement with a moderate effect size (d = 0.31) in body fat percentage which suggests a combination of aerobic exercise and suspension training may be superior to other modes of exercise, though direct comparisons are difficult to make given that each study differed in frequency and intensity of exercise.

5.3 Performance Characteristics
In the current study, the intervention resulted in a significant improvement in all physical performance characteristics investigated, indicating the effectiveness of the training intervention at improving muscular strength and speed, muscular endurance, aerobic fitness, and dynamic balance.

5.3.1 Muscular strength
The 3RM chest and leg press improved significantly post intervention with a large effect size for the leg press (d = 0.90). Force produced in isokinetic knee extension and flexion was significantly increased at both $60°.s^{-1}$ and $300°.s^{-1}$ speeds with medium to large effect sizes observed (d = 0.63 (medium), d = 0.87 (large), d = 1.09 (large), d = 1.16 (large), respectively). In addition, total work performed during isokinetic knee flexion and extension was significantly improved at both $60°.s^{-1}$ and $300°.s^{-1}$ speeds with effect sizes of 0.50 (medium), 0.50 (medium), 0.53 (medium), and 0.79 (medium), respectively. Increasing muscular strength is linked to a myriad of health benefits for all individuals including decreased risk of; sarcopenia (Mayer et al., 2011), muscular injury (Zouita et al., 2016), osteoporosis (Nikander et al., 2010), and all-cause mortality (Stamatakis et al, 2017), and an increased health related quality of life (Samuel at al., 2011). Specifically, for the obese population, increased muscular strength is associated with decreased risk of; hypertension (Artero et al., 2011), metabolic syndrome (Wijndaele et al., 2001; Jurca et al., 2004), and CVD (Silventoinen et al., 2008). Increases in strength could also allow

obese individuals to do more work in subsequent training sessions, therefore expending more energy, potentially augmenting weight loss (Abboud et al., 2013).

Improvements in muscular strength can result from a combination of muscular hypertrophy, and/or neural adaptations. Neural adaptations to resistance training typically occur in the early stages of a resistance training programme and can lead to improvements in muscular strength in the absence of muscular hypertrophy (Moritani and DeVries, 1979). Such neural adaptations can include increased excitability of motor neurons, changes in motor unit firing rate, and enhanced muscle fibre conduction velocity and coordination, that ultimately result in an increase in the rate of force production and muscular strength post-resistance training (Hedayatpour and Falla, 2015). While the current study involved suspension training and not traditional resistance training, it is likely, based on the research that has been conducted to date (Smith et al., 2016; Dolati et al., 2017; Janot et al., 2013) that similar or augmented adaptations would occur as suspension training exercises have shown to increase motor unit recruitment to a greater extent than similar traditional resistance training exercises (Snarr et al., 2013). Balshaw et al., (2017) observed increases in muscular strength after a 12 week lower body resistance training programme where participants trained three times per week. The strength gains were attributed to improvements in agonist neural activation, quadriceps cross sectional area, and absolute strength levels prior to training. Improvements in agonist neural activation was the single most important predictor of muscular strength resulting in approximately 30.6% of the total variance, with quadriceps muscle cross sectional area and absolute strength pre intervention resulting in 18.7% and 10.6% of the variance respectively (Balshaw et al., 2017). It is possible that the same factors provided the basis for strength improvement in the current study.

Adaptations to the architecture of the muscle are the primary factors responsible for longer term strength improvements. Such adaptations include muscular hypertrophy, with the eccentric component of resistance training playing a vital role in the increase of muscle size post training (Schoenfeld et al., 2015). During exercise incorporating a large eccentric component, such as observed with suspension training, the contracting muscle is forcibly lengthened under tension, resulting in mechanically induced muscular

damage, which is an import precursor for muscular hypertrophy, and increasing muscular strength (Schoenfeld, 2010). Increases in muscle cross-section can explain some of the variance in strength levels reported post training interventions (Balshaw et al., 2017) and recent literature supports the efficacy of suspension training at increasing strength levels (Smith et al., 2013; Dolati et al., 2017). The current study resulted in significant improvements in the leg press (24% increase) and chest press (14% increase). When compared with Dolati et al., (2017) who noted a 6 kg increase in leg press and 1.5 kg increase in chest press the current exercise protocol elicited superior results with a 30 kg and 5 kg increase, respectively. Though the study by Dolati et al., (2017) performed suspension training three times per week on females only, the intervention length (8 weeks), participant weight status (overweight), the total amount of exercise sets performed per week (90 sets), was similar to the current study (80 sets). Male participants generally present with higher absolute strength values following a training intervention (Dias et al., 2005; Gentil et al., 2016), and since the participants in the current study were both male and female, this could explain some of the differences in strength in the current study compared with Dolati et al., (2017).

5.3.3 Isokinetic strength and endurance

To the best of the authors' knowledge there is no published work on the effect of a combined aerobic and suspension training protocol on isokinetic strength and work, but there are however studies based on traditional resistance training. Isokinetic testing can provide information on peak force produced (peak torque) and the ability to sustain that force over a number of repetitions can be measured also (total work). Resistance training has previously been observed to increase $60°.s^{-1}$ KEPT significantly (15.6%) (Rabelo et al., 2011), while combined aerobic and resistance training significantly increased both $60°.s^{-1}$ KEPT (13.1%), and $60°.s^{-1}$ KFPT (12.2%) following an 8-week intervention consisting of 3 session per week (Lee et al., 2015). Aerobic training three times per week for 8 weeks in isolation resulted in no improvement in the same parameters (Lee et al., 2015). The current study appears more effective than the aforementioned literature with 29.1% and 37.3% improvements in peak torque for the same speeds, however older female adults participated in the studies by Rabelo et al., (2011) (67.4 ± 5.9 years) and Lee et al., (2015) (68.3 ± 2.9 years), compared with the

current study (37.1 ± 11.2 years) which may have affected the magnitude of change due to muscle loss known as sarcopenia occurring as we get older. In addition to this, male participants were utilised in the current study, and males display higher absolute strength values following a training intervention (Dias et al., 2005; Gentil et al., 2016). Although only two males participated in the current study this could still have had an effect on the results. Lastly, differences in training frequencies and durations between studies were noted. Training frequency and duration varied from three times per week for 24 weeks (Rabelo et al., 2011) to three times per week for 8 weeks (Lee et al., 2015) compared to four sessions per week for 8 weeks in the current study.

Similar to the current study, Lee et al., (2015) also investigated isokinetic total work performed, which is a measure of an individual's ability to sustain force output. Lee et al., (2015) noted a 16% and a 1% improvement for $240°.s^{-1}$ KETW and KFTW following combined aerobic and resistance training. Interestingly, aerobic training in isolation lead to an 8% increase in $240°.s^{-1}$ KETW but no improvement in $240°.s^{-1}$ KFTW (Lee et al., 2015). Improvements in total work ranged from 17% to 40% in the current study for $60°.s^{-1}$ and $300°.s^{-1}$ speeds. While a comparison of total work completed between the current study and that of Lee et al., (2015) is difficult, due to differences in participant age (37.1 ± 11.2 years vs. 68.3 ± 2.9 years), and protocol speeds ($60°.s^{-1}$ and $300°.s^{-1}$ vs. $240°.s^{-1}$) the results of the current study still indicate the effectiveness of a combined training intervention at improving the amount of force produced in the lower limbs, and the ability to maintain that force over a period of time. The increase in strength occurred alongside a significant increase in percentage muscle mass in the current study (4.5%, $P < 0.05$) post training intervention. This indicates that the intervention resulted in muscular hypertrophy in addition to the neuromuscular adaptations. The increases in muscular strength noted in the current study are associated with numerous health benefits in the overweight population such as with decreased risk of; hypertension (Artero et al., 2011), metabolic syndrome (Wijndaele et al., 2001; Jurca et al., 2004), and CVD (Silventoinen et al., 2008). Abboud et al., (2013) previously observed a positive relationship between resistance training volume and post exercise energy expenditure, therefore it is possible that the strength gains made in the current study would lead to

an increased volume load in subsequent sessions, therefore elevating energy expenditure and thus contributing toward improvements in body composition.

5.3.4 Aerobic endurance

The combined intervention in the current study elicited an improvement in aerobic endurance, with a significant increase observed in VO_{2max} with a medium effect size ($d = 0.58$). Numerous health benefits are associated with improvements in aerobic endurance including augmented blood glucose utilisation and insulin sensitivity (Solomon et al., 2015), decreased blood pressure (Hall et al., 2015), improved blood lipid markers (Schjerve et al., 2008), increased fat oxidation (Acheten and Jeukendrop, 2004), and reduced production and secretion of pro-inflammatory biomarkers (Church et al., 2002). Improvements in VO_{2max} are an expected consequence of training interventions in untrained and sedentary populations due to many physiological adaptations that occur in response to training including increases in; cardiac output (Lee and Oh, 2016), aerobic enzyme activity (Maltais et al., 1996), $A-VO_2$ difference (Prior et al., 2014), blood plasma and erythrocyte levels (Sawka et al., 2000), and mitochondrial size, number, and function (Miller et al., 2011). While aerobic training is the most effective way to increase VO_{2max}, significant increases in VO_{2max} have also been observed with suspension training interventions. Dolati et al., (2017) noted a 9.8% improvement following three suspension training sessions per week for 8 weeks. In studies examining combined aerobic and resistance training, 12-15% improvements in VO_{2max} have been reported previously (Ho et al., 2012; Willis et al., 2012), comparable to the 15% improvement observed in the current study. Though the previously mentioned combined aerobic and resistance training studies on overweight and obese participants by Ho et al., (2012) and Willis et al., (2012) involved 5 and 3 days of exercise per week for 3 and 8 months respectively, the improvements are still in line with the current study that consisted of 4 days exercise per week.

5.3.5 Sprint speed

Five, ten and twenty metre sprint times were all significantly improved from baseline with effect sizes of 0.51 (medium), 0.48 (small), and 0.56 (medium) respectively. While there is little evidence examining exercise training and sprint performance of overweight participants in the literature, there is substantial evidence supporting the

efficacy of exercise at improving sprinting performance in the athletic population, with suspension training reported to improve 20m sprint time (Nalbant, 2018), while combined aerobic and resistance training resulted in improved sprint performance in trained cyclists (Levin et al., 2009). Though the study by Nalbant, (2018) involved the addition of suspension training to regular basketball training, reductions in sprint time were observed following six weeks of suspension training. Reductions in sprint time in the current study could potentially be a result of the decrease in body weight, which has previously been linked to significant improvements in 20m sprint time ($P < 0.05$) (Huovinen et al., 2015). Likewise, strong correlations have previously been reported in trained populations between maximum strength levels and sprint speed ($r = 0.94$, $P < 0.01$) (Wisløff et al., 2004). The strength gained in the 3RM tests in the current study, combined with the improvement in body composition could have facilitated the increased speed observed over the 20m. However as the population in the aforementioned study consisted of trained athletes, it is difficult to make comparisons to the current intervention in terms of the magnitude of change. The improvements in 20m speed in the current study will enhance the physical functioning of the participants involved and could potentially have a positive impact on their quality of life. Physical functioning, incorporating movement speed, is suggested to play a large role in health related quality of life of overweight and obese individuals with Wang et al., (20103) reporting that up to 15% of the variance in quality of life was explained by physical functioning.

5.3.6 Muscular endurance

Muscular endurance is a vital component of everyday life with strong implications for quality of life, especially in clinical populations (Keogh and MacLeod, 2012; Latimer-Cheung et al., 2013). Plank time trial and TUG significantly increased from baseline in the current study with large effect sizes ($d = 1.33$ and $d = 1.52$, respectively). Suspension training has been shown to increase muscular endurance for push ups and curl ups (Smith et al., 2016), and abdominal side bridge exercises (Janot et al., 2013), though no data pertains to the plank time trial. Likewise, combined resistance and aerobic training has been reported to increase muscular endurance in a loaded carry (Hendrickson et al., 2010, McRae et al., 2012). Due to exercise selection being different in all the muscular

endurance measurements in the aforementioned studies, it is difficult to compare intervention effectiveness, however the large effect size of the current study indicates an effective intervention. Similar to this, strength training has been reported to improve TUG in older adults (P < 0.05) (Sousa and Sampaio, 2005). The large magnitude of change in both muscular endurance and TUG in the current study could be as a result of the aforementioned improvements in muscular strength. Muscular strength has been strongly correlated with muscular endurance measurements (r = 0.83, P < 0.05), (Naclerio et al., 2009). In addition, weight loss has been reported to improve TUG independent of exercise training (Vargas et al., 2013). This is possibly due to the decrease in bodyweight allowing an increased speed of movement. Research has noted that muscular endurance is inversely correlated with fat mass (P < 0.05) (Pauwels and Wilmaerts, 2017), and as mentioned previously plays a vital role in physical functioning and quality of life in overweight and obese populations (Wang et al., 2013).

5.4 Cardiovascular Characteristics

The cardiovascular health of the participants was assessed by measuring SBP, DBP, TC, HDL cholesterol, LDL cholesterol, TG and TC:HDL ratio. Significant improvements were observed in these cardiovascular characteristics post intervention.

5.4.1 Blood pressure

DBP was significantly lowered post intervention in the current study from 86 to 83 mmHg with a small effect size (d = 0.42). SBP was not significantly different post-intervention though a reduction with a small effect size was observed (131 to 127 mmHg, d = 0.33). The WHO and International Society of Hypertension guidelines note optimal resting blood pressure as lower than 120/80 mmHg, with prehypertension classified as 120–139/80–89 mmHg. Though the blood pressure values observed in the current study are in the pre-hypertensive stage, it is very possible that this was an acute elevation of blood pressure prior to exercise due to an anticipatory response to the initial physical testing (Wright et al., 2014). Nevertheless, Bundy et al., (2017) noted a linear positive correlation between mean SBP and risk of CVD and mortality, with 120 to 124 mmHg providing the lowest risk when compared to the highest risk group of 150-154 mmHg.

Several physiological adaptations occur in response to exercise training that lead to chronic reductions in SBP and DBP post intervention including decreases in; systemic vascular resistance, plasma norepinephrine, and plasma renin activity (Fagard and Cornelissen, 2007). Suspension training 3 times per week for 8 weeks has resulted in 8 mmHg decrease in SBP and 9 mmHg decrease in DBP in overweight participants (Smith et al., 2016). Aerobic exercise is associated with a 3.84 mmHg and 2.58 mmHg decrease in SBP and DBP, respectively (Whelton et al., 2002). Combined aerobic and resistance training leads to a 5.8 mmHg decrease in SBP and 3.5 mmHg decrease in DBP (Herrod et al., 2018). The meta-analysis by Whelton et al., (2002) involved 54 aerobic training interventions including mostly sedentary populations (51 of the 54 studies) of at least 2 weeks in duration, while the study by Herrod et al., (2018) consisted of 12 trials comprising 615 individuals with no information provided regarding training status of the participants. No information was provided regarding frequency, duration or intensity of exercise in either meta-analysis making it impossible to compare exercise parameters. An average decrease of 4 mmHg in SBP, and 3 mmHg in DBP was observed in the current study. Potential reasons behind the non-significant change in SBP in the current study could be as a result of an acute elevation of blood pressure due to the anticipatory response prior to the initial physical testing. Acute stress has previously been linked to increases in both SBP and DBP (Wright et al., 2014). This would have been more evident during the pre-intervention testing time point and not post testing as the participants were more familiar with the testing post-intervention and so nervousness combined with the anticipatory response to exercise was reduced. The improvement in SBP and DBP observed post intervention in the current study is a positive adaptation and is correlated with significant improvements in cardiovascular health in this population (Bundy et al., 2017).

5.4.2 Fasting blood lipids

Total cholesterol, HDL cholesterol, LDL cholesterol, TG and TC:HDL ratio were all significantly improved post intervention with effect sizes of 0.50 (medium), 0.95 (large) and 0.18 (small), 0.52 (medium) and 0.63 (medium), respectively. Though the participants were within the recommended optimal for each of the lipid variables at baseline (4.64, 2.47, 1.09, and 1.75 mmol/L, for TC, LDL cholesterol, HDL cholesterol,

and TG, respectively) they further lowered their risk of developing dyslipidemia which refers to elevated levels of TC, TG, LDL cholesterol, and low levels of HDL cholesterol (Manjunath et al., 2005). The Irish Heart Foundation recommend limits of <5 mmol/L for TC, LDL cholesterol of <3 mmol/L, HDL cholesterol of >1 mmol/L and TG levels of <2 mmol/L for optimal health (Irish Heart Foundation, 2019). Lowering TC has numerous cardiovascular benefits including reductions in CHD risk. Reductions of 0.6 mmol/L in TC have been shown to lower the incidence of ischaemic heart disease by 54% at the age of 40 years and 19% at 80 years (Law et al., 1994), and individuals with TC levels >5.172 mmol/L have a twofold higher risk of developing CHD than those with levels of <4.66 mmol/L) (Lemieux et al., 2001). Reductions in LDL cholesterol, and TC:HDL ratio also decrease the incidence of myocardial infarction and ischaemic strokes (Baigebt et al., 2010). High-density-lipoprotein cholesterol protects against atherosclerosis by removing excess cholesterol from macrophages (Rosenson et al., 2016) which is important for an overweight population as increased levels of inflammation are reported in this population.

Elevated TG can lead to the development of atherogenesis, the formation of fatty deposits in the arteries as a result of mechanisms involving binding and lipolysis at the artery wall (Ginsberg, 2000). This is particularly accelerated in the post prandial phase. Acutely increased TG levels occurring in this phase are linked with reduced vasodilation (Zheng and Liu, 2017), upregulated pro-inflammatory cytokine production (Giannattasio et al., 2005) and increased inflammatory response and monocyte activation which may contribute to endothelial dysfunction (Alipour et al., 2008). Again, although participants in the current study were within the recommended range for TG at baseline, their risk of developing atherogenesis was reduced post intervention with an 8% decrease in TG observed.

While the mechanisms behind the effect of exercise on the lipid profile are not fully understood, aerobic exercise appears to increase the ability of skeletal muscles to preferentially use lipids as a fuel source over glycogen, and lower blood lipid levels (Achten et al., 2004). The mechanisms underlying this may include adaptations to enzymes such as lecithin-cholesterol acyltrans, which regulates ester transfer to HDL

cholesterol (Calabresi, and Franceschini, 2010). Expression of enzymes such as lecithin-cholesterol acyltrans and lipoprotein lipase have been reported to increase post exercise training (Riedl et al., 2010; Harrison et al., 2012), although this may depend upon the energy expenditure during exercise. Cholesterol removal from circulation for disposal is positively correlated with increases in lecithin-cholesterol acyltrans and negatively correlated to cholesteryl ester transfer protein, which is an enzyme that facilitates transfer of cholesteryl esters to other lipoproteins following acute and chronic exercise (Lira et al., 2010). The ability of muscle fibres to oxidize fatty acids originating from plasma, TG or TC is enhanced by this increase in enzyme expression. The relationship between exercise and improvement in blood lipids was observed in the current study where 25.3% of the improvement in TC was explained by $60°.s^{-1}$ KFPT ($P < 0.05$). Meanwhile, 70.1% of the variance in HDL was explained by changes in VO_{2max}, bodyweight, and $300°.s^{-1}$ KFPT. 24.6% of the variance in LDL cholesterol was explained by $60°.s^{-1}$ KFPT ($P < 0.05$), while the percentage change in VO_{2max} explained 30.3% of the variance in TG post intervention. This indicates a relationship between both physical performance, and body composition variables with blood lipids.

Aerobic exercise can positively impact an individual's lipid profile with Nybo et al., (2010) reporting an improved TC:HDL cholesterol ratio, LeMura et al., (2000) noting a reduction in TG, and increase in HDL, while Dunn et al., (1997) noted a decrease in TC. Similar improvements across the same variables have been observed as a response to resistance training (Sheikholeslami et al., 2011) and combined aerobic and resistance training (Shaw et al., 2009; and Yang et al., 2011). Frequencies of exercise for the aforementioned studies were three times per week, with the exception of Yang et al., (2011) which consisted of five times per week. Dolati et al., (2017) noted no significant difference to any lipid variable following the performance of three suspension training sessions per week over 8 weeks. Reductions in plasma TG of 0.2, 0.3 and 0.5 mmol/L, have been observed following aerobic training (LeMura et al., 2000), resistance training (Sheikholeslami et al., 2011), and combined aerobic and resistance training (Yang et al., 2011), respectively. In addition, aerobic (LeMura et al., 2000) and resistance training (Yang et al., 2014) have elicited increases in HDL cholesterol of 0.4 and 0.5 mmol/L respectively, while reductions in TC of 0.52 and 1.0 mmol/L have been noted following

aerobic (Dunn et al., 1997) and resistance training (Yang et al., 2014), respectively. LDL cholesterol reductions of 0.48, 0.6, and 1.1 mmol/L have been observed following aerobic (Dunn et al., 1997), combined aerobic and resistance training (Shaw et al., 2009), and resistance training (Yang et al., 2014), respectively. Though training frequencies in the aforementioned studies were three and five times per week, the improvement in blood lipids in the current study is similar to the literature mentioned above for other exercise modes, with improvements in TC of 0.30 mmol/L, HDL of 0.23 mmol/L, LDL of 0.11 mmol/L and TG of 0.29 mmol/L elicited in response to the combined intervention implemented.

5.5 Metabolic Characteristics
Fasted blood glucose and fasting blood insulin measurements were taken to observe the effect of a combined training intervention on measures of glycaemic control, with improvements noted in FBG but not fasting insulin.

5.5.1 Fasting blood glucose and fasting insulin
Fasted blood glucose was significantly reduced from 5.27 (5-5.6) to 4.74 (4.2-5.1) mmol/L post intervention with a large effect size (d = 1.40). A FBG reading of 5.6 to 6.9 mmol/L is considered pre-diabetes, and although the initial baseline measurement of the participants in the current study was in the normal range, the participants reduced their FBG concentrations further post intervention, which in turn further reduced their risk of developing T2DM post-intervention. Acute benefits of resistance training include elevated utilisation of circulating blood glucose, as well as greater glycogen synthesis and storage capacity, which can occur independently of changes in insulin action (Black et al., 2010). Glucose transporter type 4 is a transporter protein that regulates glucose uptake into the cell, and an insulin like effect in GLUT4 transport has been reported as a consequence of muscular contraction (Yaspelkis III, 2006). During exercise, muscle utilises glucose made available by intramuscular glycogenolysis and by increased glucose uptake. Both aerobic and resistance exercise increase GLUT4 abundance and translocation, and hence blood glucose uptake by a pathway that is not dependent on insulin (Adams, 2013). This is a possible mechanism responsible for the reductions in FBG observed in this study.

Fasting insulin did not change significantly post intervention in the current study. This suggests that the improvement observed in FBG in this group may not have occurred via the insulin independent pathway/AMPK pathway. Smith et al., (2016) noted no changes in FBG following 8 weeks of suspension training three times per week and did not monitor insulin, while aerobic training lead to significant improvements in fasting blood insulin with no change in FBG following three sessions a week for 8 weeks (Motahari-Tabari et al., 2016). In addition to this, Maiorana et al., (2002) observed significant improvements in FBG following 8 weeks of combined aerobic and resistance exercise three times per week, while Jorge et al., (2011) observed significant improvements in both FBG and fasting insulin following 3 sessions per week for 12 weeks of both aerobic training and combined aerobic and resistance training also. Unlike that of Jorge et al., (2011), fasting insulin in the current study was not significantly different post intervention. Maiorana et al., (2002) noted improvements in FBG and though insulin was not monitored, HbA1c was also significantly improved post 8-week combined training intervention. This suggests that combining exercise modes may be more effective to improve glycaemic markers than training modes in isolation. Improvements in body weight and body fat mass result in improvements in fasting insulin as excessive adipose tissue accumulation impairs insulin signalling (Carey et al., 1996, Redinger, 2007), and although body fat and body weight were significantly reduced post intervention in the current study, no change was observed in fasting insulin.

To support the relationship between bodyweight and glycaemic measures, percentage change in bodyweight in the current study was significantly correlated with that of FBG ($r = 0.806$, $P < 0.001$). Likewise, exercise training typically improves insulin sensitivity even in the absence of weight loss (Holloszy, 2005), however no such response was noted with fasting insulin in the current study. Twenty metre sprint did however account for 70.2% of the variance in FBG post intervention ($P < 0.001$), again supporting the relationship between exercise and glycaemic measures. There are several theories as to why fasting insulin secretion would not change in response to exercise training. Inter-individual differences may play a factor with some individuals requiring greater intensities, frequencies or durations of exercise than others in order to improve insulin sensitivity (Short et al., 2003). This individual variation was evident in the current study,

with fasting insulin decreasing in some participants, but not in others in response to the same exercise intervention. The effects of exercise on insulin sensitivity may also be very acute in some individuals and so timing of insulin testing post intervention may also be a factor (Short et al., 2003). This highlights the importance of continued adherence to an exercise programme in order to maintain optimum health benefits as they are quickly reversed. Fasting insulin testing in the current study was mostly performed 72 hours post cessation of the final training session, but the testing of some individual's occurred later than this, which possibly resulted in a non-significant result. Tester error could also be a potential reason behind the non-significant change in fasting insulin, however precautions were taken to eliminate this possibility.

5.6 Novel biomarkers of insulin resistance

Biomarkers are produced in various tissues of metabolic importance across the body such as adipose tissue, liver and skeletal muscle. These biomarkers play an important role in carbohydrate and lipid metabolism which in turn influences metabolic and cardiovascular health. The circulating concentrations of biomarkers are influenced by changes in factors such as body weight, fitness levels, dietary habits and body fat levels. Favourable changes in these variables lead to increased production and secretion of anti-inflammatory biomarkers and decreased production and secretion of pro-inflammatory biomarkers. Novel biomarkers are continually emerging and their potential roles in obesity and metabolic health are currently under investigation.

5.6.1 Myostatin

Muscular tissue is an important endocrine organ that synthesises and releases various myokines involved in several cellular processes of metabolic importance throughout the body (Lee and Jun, 2019). Some of these myokines are pro-inflammatory which contribute to insulin resistance and metabolic ill health, whereas other myokines are anti-inflammatory and enhance insulin sensitivity and metabolic health.

Myostatin is a pro-inflammatory cytokine that inhibits skeletal muscle growth. The primary function of myostatin is to inhibit skeletal muscle size, and mutations of the myostatin gene downregulate myostatin production, which in turn causes increases in skeletal muscle size (Lee, 2007). Increases in skeletal muscle mass have been noted in

mice with the myostatin gene removed, with individual muscles weighing twice as much as mice with the gene (Lee and McPherron, 2001). Both aerobic and resistance training have resulted in lower circulating levels of myostatin (Allen et al., 2013; Kim et al., 2005), while an increased production of myostatin has been observed in the skeletal muscle of untrained, overweight and obese women (Hittel et al., 2009). Furthermore, Hittel et al., (2010) reported that myostatin levels have a strong negative correlation to insulin sensitivity (r = -0.82). One theory behind the effect of myostatin reducing insulin sensitivity involves the activation of the serine-threonine kinase Akt signalling pathway, which mediates the signalling of insulin, insulin-like growth factor 1 (IGF1), and other growth factors (McPherron, 2010). The activation of this pathway is greater in skeletal muscle of mice with the myostatin gene removed (McPherron, 2010).

In the current study myostatin was significantly reduced with a moderate effect size (d = 0.67) following the 8-week combined training intervention. Though reductions post exercise have been cited in the literature, these studies have examined interventions involving a single exercise mode (Allen et al., 2013; Kim et al., 2005; Hittel et al., 2010). A 37% decrease in muscle myostatin was observed following 6 months aerobic training (Hittel et al., 2010), while a 44% decrease in myostatin expression was observed 24 hours post resistance training (Kim et al., 2005). Meanwhile, the current study noted a 24% decrease in circulating concentrations following a combined suspension and aerobic training programme. While the current study noted a 24% decrease, which is lower than previously mentioned literature there are substantial differences in exercise interventions. The aerobic intervention consisted of 6 months of a 1,200 kcal energy expenditure per week at 40–55% VO_{2max}, at a self-selected frequency (Hittel et al., 2010), while resistance training consisted of a single exercise session (Kim et al., 2005). The literature that is available to date suggests a potential time course relationship between circulating myostatin levels and exercise. Harber et al., (2009) noted a significant decrease in circulating concentrations of myostatin 4, 8 and 12 hours post aerobic training intervention, but not 24 hours post. The aforementioned studies by Hittel et al., (2010) and Kim et al., (2005) both collected blood samples for myostatin analysis 24 hours post exercise while the current study conducted this analysis 72 hours post final exercise session which may have impacted the results.

The improvements in the chest press 3RM, WHR, and the plank time trial explained 82.3% of the variance in myostatin post intervention, which suggests that increases in muscle strength, and decreases in abdominal fat play a role in myostatin regulation. The decrease that was observed in the circulating concentrations of myostatin post intervention occurred alongside a significant increase in muscle mass post intervention, which suggests that myostatin may have had a role to play in regulating muscle size in this study. Given the role of myostatin as a negative regulator of both muscle mass and insulin signalling, reductions in the circulating concentration of this biomarker likely has important implications for the overweight and obese population as observed in this study.

5.7 Conclusion

The results of the current study indicate a highly effective training intervention. All physical, performance, metabolic, and cardiovascular health characteristics improved significantly post intervention with the exception of fasting insulin. Likewise, physical measures including percentage body fat, percentage muscle mass, BMI and bodyweight were significantly improved. Improvements in body fat, muscular strength, speed, muscular endurance, and aerobic endurance appeared to be responsible for the changes that occurred to the metabolic status of the participants, as identified by regression analysis. Such findings indicate the interactive relationship between several fitness parameters and metabolic health, and not any one physiological quality. This is an important factor to note when considering exercise intervention design. Several advantages of suspension training were observed during the current study including ease of adjustment, with the ability to alter resistance mid exercise quickly and safely with minimal interruptions. Another practical advantage of suspension training includes ease of access, with minimal equipment required. This could be particularly useful for individuals with limited time to travel to and from a commercial gym, and could exercise at home. Combining exercise modes could also potentially lead to greater variety and exercise enjoyment also. Anecdotally, participants enjoyed the suspension training immensely compared with the aerobic exercise.

Chapter 6: Conclusion, Limitations and Future Recommendations

6.1 Main findings

The results of this study indicate that combining suspension training and aerobic training into one intervention is an effective method to improve physical, performance, cardiovascular, and metabolic health markers in overweight and obese participants. Improvements were noted across all physical variables with the exception of calf and arm circumferences. In addition, improvements were noted across all performance variables, all cardiovascular variables with the exception of SBP, and all metabolic health markers with the exception of fasting insulin. In addition, the novel biomarker, myostatin was improved post intervention. Improvements in metabolic health markers were influenced by a wide variety of improvements in physical, and performance markers. FBG correlated with improvements in percentage body fat and sprint speed, while regression analysis indicated that improvements across several physical and performance parameters influenced the observed changes in metabolic and cardiovascular characteristics including muscular endurance, aerobic fitness, body weight, isokinetic leg strength and body fat.

6.2 Recommendations for reducing obesity and improving cardiovascular metabolic health

The results of the current study suggest that partaking in an exercise intervention that targets muscular strength, aerobic fitness, muscular endurance and body composition will improve cardiovascular and metabolic health markers. It appears that weight loss alone should not be the sole focus of exercise programmes as many other factors, such as those listed above influence cardiovascular and metabolic health. Therefore, a combined exercise intervention such as the one performed in the current study, has the potential to elicit adaptations which can enhance metabolic and cardiovascular health and reduce the incidence of overweight and obesity.

6.3 Conclusion

Obesity is currently a global epidemic and with this comes the associated comorbidities and negative health implications including insulin resistance, T2DM, and CVD. With these negative health implications comes the need to develop new and more effective exercise interventions to combat this problem. This study has identified that a combination of suspension and aerobic training is an effective method at improving

physical and metabolic health variables. In addition, myostatin, a novel biomarker of insulin resistance and an inhibitor of muscle growth was positive affected by this combined intervention. Combining exercise modalities may provide a greater chance to elicit improvements across a wider spectrum of physical, performance, cardiovascular, and metabolic factors when compared with training interventions involving a single mode. The results of the current suggest that improving these physical and performance variables will consequentially increase metabolic and cardiovascular health parameters also.

6.4 Thesis Limitations

- While participants were instructed not to deviate from normal dietary habits, a food diary was not kept. Participants could have potentially offset increases in PA with increases in energy consumption. Food diaries could potentially have ruled out possible increases/decreases in energy consumption that may have affected results.

- A control group was not utilised in this study due to an insufficient number of volunteering participants. In an attempt to account for this the exercise trial was performed over two phases in order to increase participant numbers.

- Overall PA levels were not monitored during the study. It is possible that individuals compensated for the increased activity levels induced by the exercise intervention by reducing non-exercise activity thermogenesis (energy expended for activities that are not sleeping, eating or structured exercise) as has previously reported by McLaughlin et al., (2006). Inclusion of a PA monitor such as an accelerometer could have ascertained whether or not this occurred.

- Due to scheduling issues for gym facilities there was no set time or order of completion allocated between exercises classes for participants. Though all participants completed two aerobic and two suspension training sessions over the course of the week, the order of these varied weekly depending on the participant schedule. Some weeks participants completed two consecutive aerobic sessions, followed by two consecutive suspension training sessions. This potentially negatively impacted participants' recovery as DOMS peaks approximately 48 hours post training session (MacIntyre et al., 1995), which

could have resulted in participants performing some training sessions in a fatigued state. The desired training structure of alternating the exercise modes would have allowed for adequate recovery between sessions.

- Blood testing for one participant occurred one week later than all other participants, which could have influenced the results of the tests.

- The trial was heavily weighted to female (16) vs. male participants (2). This makes comparison of the results of the current study to other studies more difficult as there is generally a more even gender split. Of the 5 participants that dropped out due to scheduling commitments, 4 of these participants were male so although the trial did start with more male volunteers it finished with just two.

6.5 Future recommendations

- Future research should include a method of monitoring total PA levels in order to ascertain the effect of exercise interventions on non-exercise activity thermogenesis. Previous research has been inconclusive regarding this topic when comparing aerobic and resistance training in isolation (Stubbs et al.., 2002; McGuire and Ross, 2012; Di Blasio et al., 2012). Given that the combination of aerobic and suspension training is novel, this is an area that should be addressed.

- Time efficient exercise interventions should be developed and investigated. Exercise modes such as high intensity interval training (HIIT) should be combined with suspension training, as research has shown HIIT potentially induces similar physiological and metabolic adaptations as aerobic exercise training, but with a reduced time commitment. This is especially important as lack of time was anecdotally reported as the main barrier to exercise by the population of the current study, and is often cited as a barrier in other studies (Hunter et a., 2019; Cerin et al., 2010)

- Interventions aimed at reducing sedentary time over the course of a day should be examined also given the time commitment barrier. Research by Ganesan et al., (2016) aimed at increasing daily step counts over the course of a working day has proved effective at increasing PA and reducing body weight. Such interventions should be developed further in order to optimise effectiveness.

- A randomised controlled trial study protocol comparing an experimental intervention such as the current experimental protocol compared to a control group, and a more traditional aerobic and resistance training group should be examined also. This would determine the effectiveness of the experiment intervention compared to other protocols.

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Appendices

Appendix 1 – Recruitment Email

To all potential participants,

I would like to invite you to take part in an exercise and health related research project run by the Department of Sport & Health Sciences at Athlone Institute of Technology.

This project will look at the effect of an 8-week suspension training and aerobic training on your body weight, physical performance, cardiovascular and metabolic health.

If you would like to take part in this study you must be currently inactive and overweight. Physically inactive means that you do not perform more than 150minutes of moderate to vigorous exercise over the course of a week. Overweight means that you have a body mass index of over 25 kg/m^2. For this project I am seeking individuals of body mass index between 25-35kg/m^2 and between ages 18 to 35 years old. Body mass index is your weight (in kilograms) divided by your height squared (in centimetres). There are various calculators online that can calculate this for you. Below is a link to a BMI calculator.

https://www.nhlbi.nih.gov/health/educational/lose_wt/BMI/bmicalc.htm

If you are interested hearing more about this project, contact me for a more detailed information sheet regarding all the specific details of the project.

I can be contacted at the following address:

Mr David Cogley: (masters research student)   email: d.cogley@research.ait.ie

I look forward to hearing from you with the possibility of working with you in the near future.

Yours sincerely,

David Cogley.

Department of Sport & Health Sciences.

Athlone Institute of Technology.

d.cogley@research.ait.ie

Appendix 2 – Plain language statement

Plain Language Statement

Principal Investigator: David Cogley   **Supervisors:** Dr. Diane Cooper

Dr. Niamh Ní
Chéilleachair

Purpose of this research:

To investigate how effective a combined aerobic and suspension training intervention is at improving body composition, fitness, cardiovascular and metabolic health. The information collected from you is the minimum required to successfully achieve this purpose.

Overview of the research project:
This study involves an 8-week supervised training intervention, and a series of fitness and health tests to be conducted before and after the 8-week period. Screening, testing days and the exercise programme will all take place in Athlone Institute of Technology.

What is required of you?

Introductory Session

Pre-participation Screening

1. You will be required to fill out a physical activity readiness questionnaire related to the study, and sign the informed consent form.
2. All details of the study will be explained to you
3. After this your height and weight will be measured.

You will be required to return on an agreed date for the testing session that suits you and the investigator.

Pre-Intervention Tests

Testing Day 1 (approximately ten minutes)

Intravenous Blood Sample Testing

- An intravenous line will be inserted in your arm by a trained phlebotomist.

- Blood samples will be taken while fasted.
- This will then be analysed for the blood insulin.

- The purpose of the test is to see how well your body processes sugar.

Fasting Blood Glucose and fasting lipid profile

- This involves pricking your finger with a single use sterile lancet to collect a drop of blood and placing the blood on a special test strip, which is inserted into the blood glucose meter
- This particular test will be used to measure fasting glucose.
- Another drop of blood will be placed on to the blood lipid meter.
- This will give you the result for fasting total cholesterol, fasting HDL cholesterol, fasting LDL cholesterol and fasting triglycerides from this sample.

Testing Day 2 (approximately 1 hour)

Heart Rate

- You will be asked to sit still in a chair while resting.
- A heart rate monitor consisting of a chest strap and a watch will be attached to you, and your resting heart rate will be recorded.

Timed up and go.

- You will start in a seated position.

- You will stand up upon testers command walk 3 meters, turn around a cone, walk back to the chair and sit down.

- The time stops when you are seated again.

Blood Pressure

- You will be asked to sit still in a chair while resting.
- A blood pressure monitor will be attached to you and your blood pressure will be recorded.
- This consists of a cuff that will be attached to your upper arm and then inflated in order to read blood pressure.

Sit and reach testing

- This test involves you sitting on the floor with legs stretched out straight ahead.

- Shoes should be removed and the soles of your feet are placed flat against the sit and reach box.

- Both knees should be locked and pressed flat to the floor.

- With the palms facing downwards, and the hands on top of each other or side by side, you will reach forward along the measuring line as far as possible.

- This test measures the flexibility in the back and leg muscles.

Height

- You will be required to remove footwear, and step onto a stadiometer, in order for your height to be recorded.

Body Mass

- You will be required to step onto a weighing scales, in order for your body mass to be measured.

Bio-Electrical Impedance Analysis (BIA)

- You will be required to remove any footwear and step onto the BIA platform in your bare feet.
- A low level, imperceptible electrical current will be sent through your body in order to determine body fat levels.

Body Circumferences

- You will have the circumference of each of the following measured: thigh, abdomen, hip, waist, upper arm, calf. The measurement will be taken using a tape measure for the widest point of the thigh, abdomen, hip, upper arm and calf. The waist measurement is taken at the narrowest point.
- All procedures during the introductory/screening and testing sessions will be carried out behind a curtained off area. While data is being collected it will not be verbalised.

Plank time trial

- You will place your forearms on the ground with the elbows aligned below your shoulders, and arms parallel to the body at about shoulder-width distance.

- You will raise your body off the ground on the go command, for the longest time period possible.

- You continue until you cannot hold the position any longer.

- This tests measures the muscular endurance of the core muscles.

Leg press strength testing.

- The purpose of this test is to measure the strength of the leg muscles.
- Your tester will estimate a light warm-up weight that you can press for the leg press exercise easily for 6-8 reps. You will then rest for 1 minute.
- Your tester will then estimate a warm-up weight that you can complete 5-7 reps, by adding 4-9 kg or 5-10% to your light warm-up weight.
- You will press the weight for 5-7 reps.
- You will then rest for 2 minutes.
- Your tester will then estimate a conservative, near-max weight with which you can complete 4-6 reps, by adding 4-9 kg or 5-10% to your warm-up weight.
- You will press the weight for 4-6 reps.
- You will then rest 2 to 4 minutes.
- The weight will then be increased by adding 4-9 kg or 5-10%.

- You will press the weight for 3 reps then rest for 2 to 4 minutes
- You will continue steps 9 & 10 until you reach a weight that you can complete for 3 reps with proper technique.

Chest Press strength testing

- The purpose of this test is to measure the strength of the upper body.
- The protocol will be the same as the leg press protocol described above except using the chest press instead of the leg press exercise.

20m speed test.

- You will get ready on the start-line (positioned behind the first timing gates used to measure speed) in a standing split-stance start position.
- You will be counted down '3 – 2 – 1 – GO '.
- On the 'go' signal you must accelerate maximally to the finish line as quickly as possible.
- You must complete 20m three sprints, each separated by a 3 minute rest.

Testing Day 3 (approximately 60 minutes)

Biodex strength testing
- You will be tested in a seated position with straps placed over your waist, both shoulders, and untested thigh for stabilisation.
- Knee flexion and extension will be tested.
- Peak torque of the knee extensors and flexors will be recorded at 60 and 300°.s^{-1}
- Two sets of five maximal attempts will be made.
- This is a machine based test that measures the strength and power produced by the thigh muscles.

VO$_{2max}$ Testing

- This test measures your general endurance and fitness levels.
- You will perform a warm up protocol consisting of 10 min at a voluntary speed (3-6 km/h) and incline (0-5 percent).
- You will then apply the face mask and the test will begin.
- The face mask is used to measure the oxygen you breathe in and the carbon dioxide you breathe out.
- The tests consists of an incremental protocol.
- After warm up, treadmill incline and speed will be set on zero and 5.3 km/h (3.3 mi/h), respectively.
- Thereafter, the grade will be increased by 2% every minute until the test is terminated due to exhaustion.

Exercise Intervention

This will be led by a trained instructor. All exercises will be adapted to suit your ability. There will be a range of session times available during the week, but the only need to attend two aerobic training sessions, and two suspension training sessions.

Aerobic Exercise

- The exercise intervention will consist of 2 hours per week.
- This be divided into two, 1 hour sessions.
- Each session will consist of a warm up of 10 minutes in duration.
- The aerobic exercise session warm up will consist of dynamic exercises designed to reduce injury risk during the session.
- This will be followed by the main body of the exercise session which will last 40 minutes.
- The main body of the session will consist of walking/jogging at a pre-determined heart rate zone for that session.
- Each session will then end with a cool down period of 10 minutes.
- The cool down period will consist of slow walking, and static stretches.
-

Suspension Training

- This is an 8-week intervention.
- The exercise intervention will consist of 2 hours per week.
- This be divided into two, 1 hour sessions.
- Each session will consist of a warm up of 10 minutes in duration.
- The suspension training protocol warm up will consist of dynamic exercises utilizing the suspension training system designed to reduce injury risk during the main session also.
- This will be followed by the main body of the exercise session which will last 40 minutes.
- The main body of the session will consist of 40 sets of various exercises incorporating upper body, lower body and core musculature.
- Each set will involve performing a set amount of repetitions for a period of 45 seconds, followed by a period of 15 seconds rest.
- The suspension training session will be conducted at a 15 (hard) to 17 (very hard) rate of perceived exertion.
- Each session will then end with a cool down period of 10 minutes.
- The cool down protocol will consist of slow walking, and static stretches designed to lower the heart rate.

Post-Intervention Tests:

This will be a repeat of all tests performed beforehand.

Potential Risks and Benefits

Risks

During the exercise tests and exercise classes there is a small chance of musculoskeletal injury. However there are measures in place to stop this happening. All tests and classes will be proceeded by a warm up protocol designed to minimize injury risk, and all tests and classes will be conducted by a trained sport scientist.

There is a chance of bleeding and/or bruising occurring during and after blood capillary sampling test. To minimise discomfort these procedures will be conducted by a trained

phlebotomist with clinical experience in that area. There is a risk of infection occurring, however gloves will be worn by the tester, and there will be a designated sterile area to reduce this risk.

<u>Benefits</u>

As part of the screening and testing process you will be have gone through a number of tests used to assess your physical performance capabilities, metabolic health and body composition. You will then participate in an 8-week exercise protocol, designed to improve your performance capabilities, metabolic health and body composition, followed by the same tests as performed before the exercise programme. You will receive a detailed physiological report regarding any progress made over the duration of the experiment after all data has been analysed.

Confidentiality

The results and information received from this study are regarded as confidential and will be used by the investigating team only. It will be stored in a secure filing cabinet and password protected computer which will only be accessible to the investigating team. Your data will be kept pseudononymous through your personal ID code. Your ID code will be kept in a password protected excel file separate to any other data. Personal data will be destroyed on completion of this study and exercise data will be destroyed 5 years after publication of this study. Hard copies of data will be destroyed by shredding and soft copies will be deleted.

Freedom of Withdrawal

Participation in the study is voluntary. Therefore you are free to withdraw from the study at any time without prejudice or reason. If you have any queries prior to consenting participation or during the study please ask any of the investigating team. Should you withdraw from the study before its completion, all hard copies of data will be destroyed and all soft copies of data will be deleted at that point.

You can contact the researcher at any time should you have any questions.

Contact Information:
David Cogley.
Department of Sport & Health Sciences.
Athlone Institute of Technology.
Email: d.cogley@research.ait.ie

Informed Consent Form

An Investigation into the Effect of Combined Aerobic and Suspension Training on Physical and Metabolic Characteristics of Overweight and Obese Individuals

- I have read and understand all the information in the plain language statement.
- I understand what the project is about and what the results will be used for.
- I am fully aware of all testing procedures and they have been verbally explained to me in detail.
- I am aware of the potential risks and benefits associated with this study.
- I understand that any information about me will be kept confidential and my information will be coded with a participant ID.
- I understand that the results of the research study may be published but that my identity will not be revealed.
- I know that my participation in this study is voluntary and that I can withdraw from the study at any time without giving a reason and that all data concerning me will be destroyed at this point.
- I understand that if I have any questions regarding any aspect of this research study I can contact any of the investigators involved with this study.

Volunteer's name: _____
Volunteer's signature: _____
Date: _____

Witness' Name: _____
Witness' Signature: _____
Date: _____

Investigator's signature: _____
Date: _____

Appendix 4 - Physical Activity Readiness Questionnaire

Physical Activity and Readiness Questionnaire

Name: Phone No:

Date of Birth: Email:

Are you aware of, through your own experience or doctors' advice, any reason that should prevent you exercising with prior medical approval? YES/NO. If yes please explain.

………

Are you taking any prescribed medicine/medication? YES/NO. If yes, please explain.

……..

Have you had any major injuries/surgeries in the last 3 years? YES/NO. If yes, please explain.

………

Are you now, or have you been pregnant in the last three months? YES/NO

………

Have you ever suffered from any lower back, hip joint, or knee joint injuries? YES/NO. If yes, please explain

………

Do you have any current injuries? YES/NO. If yes, please explain.

………

Are you currently or have you ever suffered from the following:

a) Asthma or breathing difficulties……..YES/NO   j) Chronic cough…………………..YES/NO

b) Pain/Tightness in the chest……………YES/NO   k) Stomach/duodenal ulcer….YES/NO

c) High blood pressure……………………….YES/NO   l) Liver/kidney condition………YES/NO

d) High cholesterol/triglycerides……….YES/NO   m) Arthritis/joint pain…………….YES/NO

e) Rheumatic Fever……………………………YES/NO   n) Muscular pain…………………..YES/NO

f) Any heart condition……………………….YES/NO   o) Lower back pain………………YES/NO

g) Gout……………………………………………YES/NO   p) Hernia……………………………..YES/NO

h) Dizziness…………………………………….YES/NO   q) Cramps…………………………….YES/NO

i) Diabetes……………………………………….YES/NO   r) Circulation problems………..YES/NO

Suspension Training

Warm Up:

- High knees
- High heels
- Squats
- Lunges
- Hamstring kicks
- Arm swings
- Push ups
- Tip toe walking

60 seconds of each

Main Session:

Weeks 1-4	Weeks 5-8
1. Squat	1. Squat
2. Wide Grip Row	2. Close Grip Row
3. Lunge Left Leg	3. Single Leg Squat Left Leg
4. Chest Press	4. Close Grip Chest Press
5. Lunge Right Leg	5. Single Leg Squat Right Leg
6. Bicep Curl	6. Bicep Curl
7. Hamstring Curl	7. Hamstring Curl
8. Plank	8. Knee Tuck
9. Double Leg Raise	9. Russian Twist
10. Leg hold (heels off ground)	10. Leg Scissors

45 seconds exercise: 15 seconds rest.

Repeat 4 times

Cool down:

Stretch all muscle groups for 20seconds each