The effect body composition has on adaptations to high-intensity interval exercise

Thomas Patrick Steer

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School of Social and Health Sciences

Abertay University

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Author:	Thomas Patrick Steer
Title:	The effect body composition has on adaptations to high-intensity interval exercise.
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Abstract

Introduction:

Obesity rates have increased worldwide and recently a sedentary lifestyle have been associated with obesity incidence. Being obese and/or overweight is associated with adverse effects on health. Given that time is consistently reported as the biggest barrier to exercise, particularly amongst young adults females, traditional training methods are deemed time consuming methods of training. High-intensity interval exercise (HIIE) is a time-efficient method of training that induces positive health effects despite having a substantially lower training volume and time commitment. The current study investigated the effects 6 weeks of HIIE has on specific health markers in the normal weighted and overweight female populations.

Methods:

Nineteen females were recruited and split into 3 training groups; Normal (n=7, 21 ± 0.5years; BMI, 20 ± 0.7 $kg.m^{-2}$), Overweight (n=6, 23 ± 3.7years; BMI, 30 ± 6$kg.m^{-2}$) and Control (n=6, 22 ± 1years; BMI, 22 ± 1$kg.m^{-2}$). The normal and overweight group performed three weekly HIIE sessions for 6 weeks with the control group serving as a non-training group. HIIE involved 4x15 second (s) sprints for week 1 & 2, 5x15 s sprints for week 3 & 4 and 6x15 s sprints for week 5 & 6, all alternated with a 2 minute recovery against a resistance of 7% body weight. Health markers measured before and after training were; immediate word recall, verbal fluency, segmental analysis and body composition, perception of happiness, subjective happiness, waist to hip ratio blood pressure, triglycerides, delayed memory recall and $\dot{V}O_{2\ peak.}$ Heart rate recovery between sprints was also measured during the first and last sprinting session.

Results:

In response to 6 weeks of HIIE, $\dot{V}O_{2\ peak}$ significantly increased by 25% in the normal weighted group (N: 33.7±6.3 to 41.8±8.8 $ml.kg^{-1}.min^{-1}$; p = 0.008), but no change in the overweight group. Time to exhaustion significantly increased by 18% in the normal weighted group (N: 424 ± 69 to 498 ± 84s; p = 0.005) and

12% in the overweight group (O: 497 ± 140 to 561 ± 177s; p = 0.051). Significant reductions of 3.6% were found in the normal weighted group (N; 116.7±8 to 112.4±7 mm Hg; p = 0.037) and 4.5% reduction in the overweight group (O; 125.4 ± 8.2 to 119.3 ± 7.4 mm Hg; p = 0.001) in left arm systolic pressure. A 21% enhancement in memory recall was observed in the overweight group (O; 7.8 ± 1.2 to 9.5 ± 1.8words; p = 0.019) and a 44% increase in verbal fluency letter 1 score was found in the overweight group (O; 9.8 ± 2.2 to 16 ± 4.7words; p = 0.071). A significant reduction in waist to hip ratio was found in the normal weighted group (N; 0.82 ± 0.03 to 0.81 ± 0.03; p = 0.033). Results suggest adaptations to HIIE are partially regulated by body composition in females.

Conclusion:

Given the most commonly cited barrier to exercise amongst young adult females is time. HIIE is a time-efficient method of training that improves specific health markers in both normal and overweight female's populations; the degree of adaptations however is partially regulated by body composition. Therefore, a new strategy or percentage of body weight applied to the cradle needs to be explored.

Abbreviations and Symbols

Moderate to vigorous physical activity	(MVPA)
Metabolic equivalent	(MET)
Non-communicable diseases	(NCD)
Endurance Training	(ET)
Resistance Training	(ET)
High-intensity intermittent exercise	(HIIE)
Head down bed rest	(HDBR)
Countermeasure Exercises	(EX)
Diacylglycerol	(DAG)
Carnitine palmitoyltransferase 1	(CPT-1)
Lipoprotein lipase	(LPL)
High-density lipoprotein	(HDL)
Maximal oxygen consumption	($\dot{V}O_{2max}$)
Cardiac output	(Q_{max})
Flow mediated dilation	(FMD)
Submaximal graded exercise test	(GXT)
Heart rate reserve	(HRR)
Brain-derived neurotrophic factor	(BDNF)
One-repetition max	(1RM)
Intra-abdominal adipose tissue	(IAAT)
Phosphocreatine	(PCr)
Peroxisome proliferator-activate receptor gamma coactivator 1-alpha	(PGC-1α)
AMP-activated protein kinase	(AMPK)
Sirtuin 1	(SIRT 1)

Adenosine triphosphate	(ATP)
B-hydroxyacyl-CoA dehydrogenase	(β-HAD)
Citrate synthase	(CS)
Pyruvate dehydrogenase	(PDH)
Pulse wave velocity	(PWV)
Arterial baroreflex sensitivity	(BRS)
Very low density lipoproteins triglyceride	(VLDL-TG)
Low-density lipoprotein	(LDL)
Sprint interval training	(SIT)
Central nervous system	(CNS)
Event-related potentials	(ERP)
Profile of mood states	(POMS)
Growth hormone	(GH)
Body mass index	(BMI)
Normal Group	(N)
Overweight Group	(O)
Control Group	(C)
Physical activity readiness questionnaire	(PAR-Q
Seconds	(S)
Oxford Happiness Questionnaire	(OHQ)
Analysis of variance	(ANOVA)
Time to exhaustion	(TTE)
Waist to hip ratio	(WTHR)
Verbal fluency letter 1	(VF L1)
Verbal fluency letter 2	(VF L2)
Verbal fluency letter 3	(VF L3)
Delayed memory recall	(DMR)

Revolutions per Minute	(rpm)

Symbols

Centimetres	(cm)
Significant Value	(p)
Kilograms	(kg)
Plus or Minus	(±)
Less than	(<)
Greater than	(>)
Hours	(h)

1 Introduction

Obesity rates have risen to an estimated 7% of the world's population (Trapp et al. 2008) with Scotland facing one the highest European obesity prevalence rates, with obesity rates increasing by 15.1% in men and 21.6% in women from 1995 to 2003 (Stamatakis, Hirani and Rennie 2009). Extended periods of sedentary behaviour increases the risk of becoming obese (Salmon et al. 2003) and recently obesity incidence increases have been associated with children and adolescents living a sedentary lifestyle (Rennie et al. 2005). A sedentary lifestyle type has no or irregular moderate to vigorous physical activity (MVPA) and sedentary behaviour refers to prolonged waking activities with a low energy expenditure of ≤1.5 metabolic equivalent (METs) (Tremblay et al. 2010). Being overweight and/or obese is associated with an increased risk of developing metabolic syndromes such as diabetes; cardiovascular disease related disorders and reduced quality of life (Trapp et al. 2008). The most commonly cited barrier to exercise is time (Booth et al. 1997); therefore, high-intensity interval exercise (HIIE) may provide positive health adaptations due to the potent time-efficient training approach.

Gender differences

During sedentary behaviour (TV viewing) females are more prone to non-communicable diseases (NCD); each hour women watch TV, the risk of an irregular glucose metabolism increases by 18%, however this trend was not as robust in men (Dunstan et al. 2004). It has been shown watching ≥20 hours of TV a week increases the risk of metabolic syndrome by 1.5 fold in men and 1.93 fold in women (Chang et al. 2008). Physical activity rates drop through adolescence with females having a higher drop out frequency due to femininity (Sallis and Patrick 1996) and lower physical activity levels in comparison to males (Gorely et al. 2007). Young, untrained adult females carry on average 10% more body fat than males (Sparling et al. 1998) and females have a 20% lower aerobic capacity when maximal oxygen consumption ($\dot{V}O_{2max}$) is conveyed

relative to body mass ($ml,kg^{-1}.min^{-1}$) (Sparling et al. 1998). Increases in testosterone during puberty allow males to have a 10-14% greater haemoglobin concentration (Woodson 1984), suggesting reasons why males have a higher aerobic capacity potential during training. Increases in testosterone allow males to grow larger bones and more muscle mass than females, and increases in estrogen secretion increases fat content in females compared to males (Akalan, Kravitz and Robergs 2004).

Traditional Training Methods

Endurance training (ET) is the most common method of training and for health promotion, with public health guidelines currently recommending individuals undertake a minimum of 150 minutes of moderate-to-vigorous exercise per week (Gibala et al. 2012). It has been documented that ET induces improvements to health-related markers such as skeletal muscle oxidative capacity increases (Carter et al. 2001; Jones and Carter 2000; Hurley et al. 1986), increased stroke volume (Stratton et al. 1994), improved insulin sensitivity (Kang et al. 1996) and improved blood pressure (Guimaraes et al. 2010). Reports also show ET helps control body composition and increases intramuscular glycogen stores (Elina et al. 2008).

Resistance training (RT) is another method of training and is recommended by the American College of Sports Medicine (ACSM) to complete 8-12 repetitions for 8-10 exercise with a minimum of 1 major muscle group and for older individuals 10-15 repetitions in order for muscular, flexibility and cardiorespiratory fitness retention (Kraemer et al. 2002). The ACSM and the American Heart Association both suggest incorporating RT into an aerobic training program to help control, treat and prevent hypertension (Pollock et al. 2000) and type 2 diabetes (Albright et al. 2000). RT programs incorporated into aerobic training have been shown to reduce the risk of NCD, promote psychological well-being (Stewart, Mason and Keleman 1988) and aid weight loss and management (Wilmore 1974; Katch and Drum 1986).

Barriers

The most commonly cited barrier for physical exercise participation is time (Booth et al. 1997), particularly amongst female adults of the ages 20-27 years (Ansari and Lovell 2009) and young female adolescents (Tergerson and King 2002). Despite time being a major barrier for partaking in physical exercise among young females, males do not elicit time as a major barrier for participation (Tergerson and King 2002). Furthermore, time is also considered the biggest barrier to exercise-constraints by inactive individuals who want to become active, especially amongst female college students (Harne and Bixby 2005).

High-intensity intermittent exercise

HIIE is a training method characterized by short 'all-out' vigorous sprints, alternated with a form of recovery (Gibala and McGee 2008) that induces a number of positive health-related indicators and performance adaptations among healthy and diseased populations (Whyte et al. 2013; Trapp et al. 2008; Burgomaster et al. 2008). Improvements to respiratory fitness following HIIE has shown to be superior over ET in individuals suffering NCD such as heart failure and coronary heart disease among obese, middle aged and adolescent populations (De Araujo et al. 2012; Moholdt et al. 2009; Wisloff et al. 2007; Gibala et al. 2012). In response to HIIE, improvements to endothelial function were considerably greater than those elicited by ET (Moholdt et al. 2009; Wisloff et al. 2007). Studies have documented improvements to glucose tolerance (Babraj et al. 2009), $\dot{V}O_{2max}$ (Rakobowchuk et al. 2008), blood pressure (Whyte Gill and Cathcart 2010); positive body composition alterations (Heydari et al. 2012) enhanced insulin sensitivity (Richards et al. 2010; Whyte et al. 2010) and increased fat oxidation (Whyte et al. 2010) following HIIE, despite a 90% lower training volume and 67% lower time commitment than ET (Burgomaster et al. 2005; Gibala and McGee 2008). However most of these studies have used predominately male populations or mixed. Given the most common cited barrier to exercise constraints among young female adolescents is time constraints (Tergerson and King 2002), HIIE is a time-efficient method of training with a

substantially lower training volume and time commitment to traditional training methods that induces positive health markers among all populations.

Therefore, the present study aims to compare the effects a 6 week HIIE has on specific health markers including cardiorespiratory fitness, cognitive function, body composition, blood lipids and mood in the normal and overweight female populations'. It is hypothesised that 6 weeks of HIIE would result in alterations to health markers in both groups however greater alterations may be seen in the overweight population due to the cardiovascular, mitochondrial, parasympathetic and sympathetic nervous system adaptations attained through HIIE.

2 Literature Review

2.1 Sedentary Behaviour

A sedentary lifestyle is characterized by prolonged periods of low energy expenditure activities and with absent or irregular MVPA (Tremblay et al. 2010). Energy expenditure can be quantified as METs, with resting equating to 1 MET and sedentary behaviour characterized by an energy expenditure of ≤1.5 METs (Tremblay et al. 2010). A number of health risks are associated with sedentary behaviour;

- Body composition; Obesity assessed through body mass index, waist and hip circumferences (Rey-Lopez et al. 2008).
- Cardiovascular disease; (Wannamethee and Shaper 2001) blood pressure (Heffernan et al. 2013).
- Metabolic syndrome; Type 2 diabetes (Wilmot et al. 2012), poor blood lipids (Yanagibori et al. 1997).
- Mental Health; Cognitive function (Lipnicki et al. 2009), negative mood (Ryback, Trimble and Lewis 1971), poor self-esteem (Tremblay et al. 2010).

2.2 Adaptations to Sedentary Behaviour

2.2.1 Cardiovascular Adaptations

Bed rest has been suggested to be a model for the short term effects of sedentary behaviour; however it does not fully mimic sedentary behaviour (Convertino, Bloomfield and Greenleaf 1997). Prolonged bed rest has been shown to decrease $\dot{V}O_{2max}$; the magnitude of which is dependent on initial fitness levels and the duration of the bed rest (Convertino, Bloomfield and Greenleaf 1997). Following 21 days of bed rest among young males, a 26% decrease in $\dot{V}O_{2max}$ was observed, along with a 26% reduction in maximal cardiac output. The author also reported a 2.6% increase in maximal heart rate and a 29% reduction in stroke volume (Saltin et al. 1968). Reductions in $\dot{V}O_{2max}$ were attributed to changes in stroke volume, due to the unchanged arteriovenous oxygen difference, meaning

unchanged blood supply to capillary beds and working skeletal muscles (Saltin et al. 1968). In response to 60 days of head down bed rest (HDBR) (CON) and 60 days of bed rest with countermeasure exercises (EX) among women, the CON group had a 9% decrease in blood volume, whereas the blood volume in EX group's was maintained with a 4% decrease (Guinet et al. 2009). Furthermore, exercisers had a 19% lower heart rate during supine rest following HDBR and a decreased rate in stroke volume and heart rate during the tilt/lower body negative pressure test, however this change was not elicited in the CON group. Similarly, a sedentary lifestyle is expected to result in a greater resting heart rate due to inhibition of parasympathetic activity which stimulates vagal nerves of the heart (Milgliaro et al. 2001). With sedentary individuals, arterial compliance was reduced and fasting insulin was reported to be twice as high compared to trained individuals (McGavock et al. 2006).

2.2.2 Fat Metabolism Adaptations

Sedentary behaviour has been shown to result in an elevated rate of adipose tissue lipolysis (Alibegovic et al. 2010) and 9 days of bed rest leads to a reduction in whole body insulin sensitivity (Alibegovic et al. 2010). A loss of sensitivity to insulin results in elevated rates of lipolysis with evidence suggesting this inhibition occurs in intraabdominal adipose tissue (Hickner et al. 1999). Furthermore, there is inhibition of skeletal muscle fat oxidation, insulin resistance in the skeletal muscle and potential lipotoxic effect of diacylglycerol (DAG) build up in skeletal muscle (Hickner et al. 1999). During bed rest protocols, fat oxidation can be altered due to a fuel metabolism shift favouring carbohydrate metabolism, the magnitude of this shift however is dependent on the duration of bed rest (Bergouignan et al. 2011). In response to 3 months of bed rest, carbohydrate oxidation increased by 21% and a 37% decrease was found in lipid oxidation among healthy men (Bergouignan et al. 2006). Seven days of bed rest resulted in skeletal muscle insulin resistance in men, skeletal muscle and liver insulin resistance among women, basal lipid oxidation decreases in both men and women by ~90%, higher carbohydrate oxidation rates in men of 25%, a 570% increase in net lipogenesis in women and reduced sympathetic activity in both men and women (Blanc et al. 2000). Although the exact mechanisms for a

shift in fuel metabolism are unknown, rodent studies found this may be due to a skeletal muscle fiber shift; decreased oxidative skeletal muscle fibers and increased glycolytic skeletal muscle fibers (Bergouignan et al. 2011). In response to 28 days of bed rest in humans, a greater type II fibre loss was found compared to type I (Fitts et al. 2007). This loss of type II fibres may be due to a greater atrophy of oxidative fibres found in the rodent study. Decreased insulin rates and reduced fat oxidation rates occur in glycolytic skeletal muscle fibres, meaning a decrease in skeletal muscle glucose uptake (Bergouignan et al. 2011). Although the substrate use shift occurred prior to the shift in skeletal muscle fiber following a study on rats (Grichko et al. 2000). Physical inactivity has led to reductions in carnitine palmitoyltransferase 1 (CPT-1); responsible for long-chain fatty acid transportation to the mitochondria, is linked to decreases in fat uptake within the skeletal muscle (Stein et al. 2002). Following several weeks of hindlimb unloading among rodents, glycolytic capacity in atrophied skeletal muscle was increased (Stein et al. 2002; Wittwer et al. 2002) through the increased expression of the enzymes involved with glycolysis; pyruvatekinase, hexokinase and phosphofructokinase, with a metabolic shift from fat to glucose occurring (Stein et al. 2002). Furthermore, reductions in the rate of lipid oxidation were indicated by reductions in the enzymes used in beta-oxidation (Stein et al. 2002). Significant reductions have been reported during sedentary activities in skeletal muscle lipoprotein lipase (LPL) activity, a fat metabolism enzyme regulator (Bey and Hamilton 2003; Zderic and Hamilton 2006). Rodent based studies have found in response to acute leg immobilisation, reductions in skeletal muscle LPL activity (Bey and Hamilton 2003; Zderic and Hamilton 2006). Reductions in LPL activity have been linked with decreases in high-density lipoprotein (HDL) cholesterol, mild decreases in blood sugar following food ingestion and decreased skeletal muscle plasma triglyceride (TG) uptake (Reymer et al. 1995). Serum TG levels were increased by 40% in the sedentary individuals (McGavock et al. 2006) and in animal studies; there is a reduction in lipoprotein lipase activity which is associated with high circulating TG levels in response to sedentary behaviour (Trembley et al. 2010). Prolonged bed rest causes insulin-mediated glucose disposal resistance, which partly explains why inactive older individuals have an increased risk of type 2 diabetes mellitus (Manson et al. 1992). This supports the finding of a sedentary lifestyle

accelerates the progression of chronic diseases and is related to insulin resistance (Seals et al. 1984) and arterial stiffness (Tanaka et al. 2000).

2.2.3 Cognitive Function and Mood Adaptations

Following 60 days of HDBR, a detrimental effect on executive functioning and reaction time among healthy males was found and the decline in executive functioning and reaction is suggested to be due to the lack of physical exercise, with evidence suggesting alterations in prefrontal cortex function may also be an underlying mechanism due to its relation with cardiac vagal tone (Lipnicki et al. 2009). A comparison of sedentary and endurance trained middle-aged individuals found neuropsychological performance and occipitoparietal perfusion to be greater and central stiffness to be lower in the endurance individuals compared to the sedentary individuals (Tarumi et al. 2013). Improved cognitive function scores such as memory and attention-executive function were associated with greater cardiopulmonary fitness. Improved neuropsychological outcome independent of factors such as education, age, and sex were correlated with lower central arterial stiffness and greater occipitoparietal blood flow (Tarumi et al. 2013). This suggests taking part in aerobic exercise at midlife may prevent cognitive decline in further years. Edwards et al. (1982) and Loseliani, Narinskaya and Khisambeyev (1985) both found a decline in cognitive performance following bed rest. Contrary to this, bed rest protocols have been found to have no effect on cognitive measures; for example 60 or 90 days of HDBR did not affect cognitive functioning, with high individual cognitive ability and motivation levels proposed for effect on cognitive function (Seaton et al. 2009). Impairment of mood states and enhanced neurotic and depressive levels in healthy males has been found in response to 20 days of bed rest (head-down tilting - microgravity), with exercise sessions not affecting the alterations (Ishizaki et al. 2002). Suggested reasons are due to immobilization caused by the bed rest, the cardiovascular changes caused by microgravity and the social isolation caused by the prolonged periods of bed rest (Ishizaki et al. 2002). Furthermore, 3 days of bed rest appears to increase depression scores assessed through the Beck Depression Inventory, increase back and abdominal pain, headaches and leg pain, although participants had lower scores on the Bond-

Lader mood questionnaire among healthy males (Jorma et al. 2001). Suggested reasons for increases in depression may be due to increase in pain caused by the bed rest, particularly to the spinal column and lumbar nerve roots (Jorma et al. 2001).

2.4 Traditional Exercise Training

Traditional exercise involves both endurance and resistance training. Endurance describes an individual's velocity-time curve, relating to a sequence of power-outputs an individuals is able to sustain over a time period (Whipp et al. 1982). The term endurance or aerobic training indicates numerous training practices and can be used to maintain or improve the neuromuscular, pulmonary and cardiovascular systems (Jones and Carter 2000). Partaking in ET increases intramuscular glycogen stores, decreases total and abdominal fat (Elina et al. 2008) and reduces resting systolic and diastolic blood pressure (Guimaraes et al. 2010). The design an ET session is a continuous event lasting approximately 40-120 minutes (Gibala et al. 2006), completed at 65-to-100% $\dot{V}O_{2\ max}$ (Jones and Carter 2000). The majority of RT programs are progressive, meaning individuals undertake the program until their specific training goal has been reached, in this case improving muscular size or strength (Kraemer et al. 2002). RT programs can also be used if the training goal is to maintain muscular fitness levels (Kraemer et al. 2002). The extent of these adaptations however is dependent on individual goals, genetic predisposition, intensity, volume, rest and selection of exercise (Fry 2004; Spreuwenberg et al. 2006). For individuals suffering from hypertension (Pollock et al. 2000) and type 2 diabetes (Albright et al. 2000), both the ACSM and the American Heart Association suggests for treatment, control and prevention of these diseases, moderately intense RT should be incorporated into aerobic training programs (Braith and Steward 2006). RT that is incorporated into comprehensive fitness programs, prevents osteoporosis (Layne and Nelson 1999; Gutin and Kasper 1992), reduces the risk factors associated with coronary heart disease (Hurley et al. 1988; Goldberg 1989), helps weight management and loss (Wilmore 1974; Katch and Drum 1986), and promotes psychological well-being (Stewart, Mason and Keleman 1988). Both ET and RT modalities have been found to be beneficial for health (Guimaraes et al. 2010; Albright et al. 2000).

2.4.1 Cardiovascular Adaptations

2.4.1.1 Endurance Training

ET induces increases in $\dot{V}O_{2max}$, determined by alterations to cardiac output (Q_{max}), the oxygen carrying capacity (haemoglobin content) and the skeletal muscles ability to utilize oxygen to the mitochondria (Jones and Carter 2002). The magnitude of these adaptations is dependent on a number of factors; baseline fitness, intensity, frequency and duration of the training program (Wenger and Bell 1986). Q_{max} and $\dot{V}O_{2max}$ are both related to stroke volume and heart rate (Spina et al. 1992) and stroke volume is a mediator of $\dot{V}O_{2max}$ improvements, mediated by improvements in O_2 pulse (Schjerve et al. 2008). Following 12 weeks high-intensity (85-95% maximal heart rate) and moderate-intense aerobic training (60-70% maximal heart rate), peak O_2 pulse improved in conjunction with improvements to $\dot{V}O_{2max}$ and flow mediated dilation (FMD) (Schjerve et al. 2008). Interestingly, the high-intensity aerobic training group made higher $\dot{V}O_{2max}$ and O_2 pulse improvements (Schjerve et al. 2008) and working at higher intensities during aerobic training elicits greater $\dot{V}O_{2max}$ improvements due to increased cardiac output and oxygen extraction by the working skeletal muscles (Shinkai et al. 1992). Changes in end-diastolic volume, left ventricular size, myocardial contractility and catecholamine sensitivity influence stroke volume and resting heart rate during submaximal exercise (not exceeding 85% HR reserve) (Spina 1999). ET has been seen to increase peak cardiac output following 6 months of 4 to 5 45 minute sessions a week, due to increases in stroke volume (Stratton et al. 1994). Stroke volume increased due to end-diastolic volume increases and ejection fraction increases (Stratton et al. 1994).

2.4.1.2 Resistance Training

RT has also been shown to improve $\dot{V}O_{2max}$. Twelve weeks of RT improved $\dot{V}O_{2max}$ by 10% however this was due to the low-intensity 15 minute warm-up prior at the start of RT, and/or improved antioxidant status and decreased levels of oxidized LDLs (Schjerve et al. 2008). Despite this, 10 weeks of maximal RT did not improve $\dot{V}O_{2max}$, although short-term endurance of 4-8 minutes time to exhaustion improved by 13% and 11% (Hickson et al. 1988). Increases in strength have a positive effect on heart, specifically work economy (Schjerve et

al. 2008), and with interventions showing a direct association between maximal RT, neural adaptations and improved work economy (Hoff et al. 1999; Hoff et al. 2002), suggesting work economy may be improved due to the neural adaptations associated with RT. Following 26 weeks of RT, no enhancement to cardiovascular function was observed (Hagberg et al. 1989). Wood et al. (2001) found cardiovascular improvements measured in the length of time on the GXT following 12 weeks of combining resistance and endurance as a training program. This is supported by Ferketich et al. (1998) who also found improvements in the time taken to complete the submaximal graded exercise test (GXT). Despite this, both the aforementioned studies also suggest that when RT was not included in the training program, the GXT times were enhanced (Wood et al. 2001). Despite the evidence suggesting RT improves $\dot{V}O_{2max,}$ RT should not solely be used if improvements in cardiovascular function are an end goal, and ET should be incorporated or solely used to improve cardiovascular function.

2.4.1.3 Endurance and Resistance Training

In response to 16 weeks of two weekly 40 minute treadmill training sessions, daytime blood pressure decreased by 1 mm Hg systolic and 2 mm Hg diastolic in hypertensive individuals (Guimaraes et al. 2010). Likewise with RT following a meta-analysis by Kelley and Kelley (2000) decreases of 3 mm Hg average in both systolic (2%) and diastolic (4%) blood pressure from 11 studies in response to RT was found. It is suggested that a 3 mm Hg reduction in systolic blood pressure among the general population reduces all-cause mortality by 4% (Lewington et al. 2002; Stamler 1991; Whelton et al. 2002). Blood pressure decreases are mediated by reductions in sympathetic nervous system, which effects the kidneys; a key factor for long-term blood pressure control (Cornelissen and Fargard 2005), the baroreflex control (Somers et al. 1991), improvements to peripheral vascular resistance and improvements in endothelial function (production and action of nitric oxide) are suggested to be involved with the mediated reductions in blood pressure (Guimaraes et al. 2010). Dynamic endurance exercise training programs at 65% heart rate reserve (HRR), consisting of three 40 minute weekly session for 16 weeks significantly increased $\dot{V}O_{2\ max}$ (Cornelissen and Fargard 2005). The same authors also found following

a meta-analysis report, degree of blood pressure alterations is significantly associated with $\dot{V}O_{2\ max}$ increases (Cornelissen and Fargard 2005; Cornelissen et al. 2011). In response to 12 weeks of aerobic exercise training at 70-75% heart rate maximum, increases in the carotid compliance were found and the same for both the endurance-trained men and healthy participants (Tanaka et al. 2000). To support this, Guimaraes et al. (2010) found 16 weeks of aerobic interval training decreased arterial stiffness. Despite this, systolic hypertensive individuals working at 65-70% HRR for 8-12 weeks did not show any improvements in arterial stiffness. This suggests for improvements to arterial stiffness in hypertensive individuals, increased manipulation of intensity or duration is needed, however in normotensive individuals not suffering from arterial stiffness, to improve arterial functionality, a low intensity and shorter duration may be required (Tanaka et al. 2000; Cameron and Dart 1994). Miyachi et al. (2004) found carotid arterial compliance; an indicator of arterial stiffness decreased by 19% following 3 days a week for 4 months of RT in young healthy male novice weight trainers. However, after 2 month follow-up, the carotid arterial compliance returned to baseline (Miyachi et al. 2004), suggesting RT to be an inadequate training method for controlling carotid arterial compliance. Despite this, high-intensity RT, consisting of 4 day a week for 3 months, reported increases in carotid-femoral pulse-wave velocity and carotid augmentation index in healthy female novice weight trainers (Cortex-Cooper et al. 2005). This study however used high-intensity super-sets, a method of training that is only recommended for competitive athletes, not the general population (Pollock et al. 2000; Pescatello et al. 2004). Combining aerobic exercise and RT for 10 weeks among middle-aged hypertensive men decreased both diastolic and systolic blood pressure of 13 mm Hg (Keleman and Efron 1990) and using a similar training program with a 6 month duration, systolic pressure reduced by 5.3 mm Hg and diastolic pressure reduced by 3.7 mm Hg among hypertensive older adults (Stewart et al. 2005). This suggests combining aerobic and RT is superior to solely RT for reductions in systolic and diastolic blood pressure.

2.4.2 Lipid Metabolism Adaptations

Following 12 weeks of ET, a 25% increase in the rate of fat oxidation has been demonstrated due to increased TG production and improved muscular mitochondrial activity (Horowitz et al. 2000). Likewise, 2 years (3 set of 10 repetitions at 85-100% 10 RM with a 90 seconds(s) rest) of RT among physically active young men resulted in greater whole body fat oxidation (Ormsbee et al. 2007). Reasons for elevated lipolysis oxidation rates may be due to increased catecholamines and lower skeletal muscle intramuscular lipids may have also contributed (Ormsbee et al. 2007). With both resistance and ET there is an improvement in insulin sensitivity independent to changes in body composition (Shaibi et al. 2006; Duncan et al. 2003). For ET a higher volume of the exercise session leads to higher improvements in insulin sensitivity (Houmard et al. 2004), both using the same weekly, exercising for ~170 minutes compared to exercising for ~115 minutes, insulin sensitivity was significantly greater in the group with the higher volume (Houmard et al. 2004). Skeletal muscle insulin resistance can be altered by fat oxidation and fat oxidation can be supressed by insulin (Kelley et al. 1990). During fasting conditions in individuals with normal insulin sensitivity, skeletal muscles rely on fat oxidation (Andres and Zierler 1956), however among obese and type 2 diabetic individuals; fat oxidation in the skeletal muscle is reduced (Kelley and Simoneau 1994). Aerobic training above 50% $\dot{V}O_{2\ peak}$ results in improvements in whole body insulin sensitivity (Seals et al. 1984; Kang et al. 1996); suggesting working at high-intensity during endurance based activities elicits enhanced improvements to insulin sensitivity. For increasing insulin action, it has been suggested moderate-to-heavy RT consisting of 1-4 set of 4-15 repetitions at 75-90% RM, 3 to 4 times a week elicits increases in insulin action by 22-24% (Zachwieja et al. 1996; Millar et al. 1994), whereas light-to-moderate RT of 2-3 sets of 8-20 repetitions at 40-60% RM 3 to 5 times a week elicits increases in insulin action of 23-48% (Ishii et al. 1998; Eriksson et al. 1998). Improvements in insulin sensitivity following RT are due to the up-regulation of key protein concentrations involved with insulin-signalling; glycogen synthase, GLUT-4 and protein kinase (Shaibi et al. 2006), partly explaining improvements to insulin sensitivity and insulin action. Regulation of insulin sensitivity will improve fat processing and regulated adipose tissue lipolysis leading to lower circulating blood lipids. Indeed both resistance and endurance exercise have been shown to lower circulating blood

lipids (Tambalis et al. 2008). However, RT does not appear to alter mitochondrial function (Tanaka and Swensen 1998) whereas ET has been shown to up-regulate mitochondrial enzyme activity and content (Horowitz et al. 2000).

2.4.3 Cognitive Function and Mood Adaptations

Nagamatsu et al. (2012) found 12 months; twice a week of RT significantly improves cognitive function assessed through the Stroop Test in the elderly. Brain-derived neurotrophic factor (BDNF) levels directly affect the brain health and cognitive function (Tsai 2003) and BDNF induces excitatory postsynaptic currents and modulates neurotransmitter release, as well as directly inducing neuronal depolarization (Kafitz et al. 1999). This suggests cognitive function improvements were induced by the increases in BDNF levels and the extent of the BDNF release is dependent the intensity of the exercise (Ferris, Williams and Shen 2007). Working at high intensities during RT lasting 10 and 20 weeks improves quality of life and decreases depressive like symptoms (Singh, Clements and Fiatarone 1997; Singh, Clements and Singh 2001). Indeed, ET consisting of 12 months, 3 to 5 sessions a week among older sedentary individuals has also found improved self-reported morale (Hill, Storandt and Malley 1993). This increase in self-reported morale was in conjunction with a 23% increase in $\dot{V}O_{2max}$ (Hill, Storandt and Malley 1993). This is supported by Belardinelli et al. (1999) who found improvements in quality of life were in conjunction with $\dot{V}O_{2peak}$ increases, supporting the notion of increases to psychological well-being may be partly attributed to exercise capacity increases. Suggested reasons for improvements made to depressive individuals were due to biological alterations such as reducing somatic symptoms and improving health and physical functions (Singh, Clements and Singh 2001) which were in conjunction with the significant correlation found between intensity of RT and reducing the percentage of depressive scores. This suggests working at a higher intensity will improve depression symptoms due to the physical and mental improvements along with the cognitive mechanisms involved (Singh, Clements and Fiatarone 1997). Improvements in quality of life are due to the role exercise may play in facilitating social interaction and enhanced health, positively affecting quality of life and leading to social life improvements (Resnick and

Spellbring 2000). Exercise trials among non-depressive individuals showed less depressive symptoms accompanied by decreases in plasma ß-endorphin (Lobstein and Rasmussen 1991) and following exercise, increases in mood were associated with basal ß-endorphin levels (Janal et al. 1984), suggesting decreases in depression scores may be attributed to role exercise plays in releasing ß-endorphins.

2.4.4 Body Composition Adaptations

Following 4 months of ET (5 days/week for 1 hour of running or cycling) with intensity progressively increasing and heart rate corresponding to 50-85% $\dot{V}O_{2max}$ in overweight men, increases in lipid oxidation rates were found and seen as a key marker for fat mass regulation and obesity prevention (De Glisezinski et al. 2003). Improvements in body composition have been found following 2 years of RT consisting of a 40-45 minute session (3 set of 10 repetitions at 85-100% 10 RM with a 90 s rest) among physically active men (21-27 years), improved body composition measured through skinfolds were observed and due to increases whole body fat oxidation and expenditure, along with greater abdominal subcutaneous adipose tissue lipolysis (Ormsbee et al. 2007). Increased amounts of catecholamines and lower intramuscular lipids within the skeletal muscle may have contributed to the increases in lipolysis oxidation rates (Ormsbee et al. 2007) and during heavy RT in comparison to ET, plasma catecholamines concentrations are significantly greater when energy expenditure is the absolute same (Hurley et al. 1984). In response to 8 months of either high volume vigorous intensity exercise (20 miles of light jogging at 65-80% peak oxygen consumption), low volume vigorous intensity exercise (12 miles jogging per week at 65-80% peak oxygen consumption), and low volume moderate intensity (12 miles walking at 40-55% peak oxygen consumption) among sedentary, overweight men and women (40-65 years) found the most significant body mass and fat mass alterations were in the high volume group, however all groups significantly decreased waist to hip and abdominal measurements (Slentz et al. 2004). The high volume exercise group elicited the largest weight and fat reductions due to the positive imbalance of calories in the highest energy used, the study suggests to prevent weight gain, 6-7 miles of exercise per week is

adequate enough (Slentz et al. 2004). Binder et al. (2005) found using a 3 month, 3 times a week progressive RT exercise (progressing from 1-3 sets of 6-12 repetitions at 65%-100% one-repetition max (1RM)) in men and women >78 years and found increases in total and fat free mass, along with isokinetic skeletal muscle strength, although no changes were elicited in fat mass. It is suggested in order to reduced abdominal adiposity, combined aerobic and RT is necessary (Binder et al. 2005). Furthermore, improvements in body composition have been seen following 2 years of resistance exercise, suggesting based on research if Binder et al. (2005) study was longer, further improvements in body composition may have been achieved. Following 24 weeks of RT, consisting of 3 45 minute session a week of two sets of 10 repetitions of 8-10 different exercises and intensity increased to 80% 1-RM if two sets of 10 repetitions were completed (Hunter et al. 2002). Both men and women increased fat free mass and decreased fat mass, however women significantly reduced intra-abdominal adipose tissue (IAAT) but men did not (Hunter et al. 2002). The losses found by women and not men may be due to initial visceral distribution and fat distribution may be affected by the hormones such as sex hormone binding globulin, growth hormone, estradiol and cortisol associated with RT and exercise (Hunter et al. 2002).

2.5 Problems with traditional training methods

2.5.1 Injury

RT related injuries are most common amongst adolescents due to the unsafe lifting behaviour and techniques, lack of qualified supervision and unsafe lifting loads (Myer et al. 2009; Jones, Christensen and Young 2000). To support this, a 13 year-old boy attempted to overhead press 30kg at his home gymnasium and had bilateral fracture separations of the distal radial epiphyses as a results (Jenkins and Mintowt-Czyz 1986). Endurance exercise provokes overuse injuries due to a disproportion of overtraining and a lack of recovery time (Cosca and Navazio 2007). Eventual tissue breakdown and cellular tissue damage occur prior to pain experiences and are results of over training and insufficient recovery time (Wilder and Sethi 2004). Overuse injuries are degenerative and characterized by the disorganization and degeneration of collagen fiber, enhanced tenocyte count and prolonged healing response (Kahn et al. 1999). An athlete's ET program can be overwhelming for their body and cause overtraining, which is considered an overuse injury (Halson and Jeukendrup 2004). Overtraining can cause decreases in the athlete's performance and high fatigue rates, as well as increasing the athlete's risk of injury and illness (Cosca and Navazio 2007).

2.5.2 Time

A lack of time has been consistently found for reasons to a lack of participation in traditional exercise constraints (Schutzer and Graves 2004; Ansari and Lovell 2009; Harne and Bixby 2005). A survey using 471 college students found the most common barriers to participation in exercise were; lack of willpower, lack of time and lack of motivation, with the survey also findings no difference between genders for the barriers (Silliman, Rodas-Fortier and Neyman 2004). In comparison to younger women, older women showed more barriers to physical exercising due to family commitments and lack of time (Ansari and Lovell 2009). College women already taking part in RT have less barriers for

participation compared to college women who do not take part in RT, and one of the highest barrier to participation for non-strength training college women was time-effort (Harne and Bixby 2005). To support this, time-effort is perceived higher by inactive individuals as the biggest barrier to becoming more active compared to those who are already taking part in physical exertion (Myers and Roth 1997).

2.6 High-Intensity Intermittent Exercise (HIIE)

Given that most frequently cited barrier for exercise is a lack of time (Nybo et al. 2010; Gibala et al. 2012), numerous studies have demonstrated HIIE induces a number of key health-related adaptations such as enhanced insulin sensitivity (Richards et al. 2010; Whyte et al. 2010), improved glucose tolerance (Babraj et al. 2009), positive body composition alterations (Heydari et al. 2012), vascular function improvements (Rakobowchuk et al. 2008) and reductions in blood pressure (Whyte et al. 2010) despite HIIE expressing a substantially lower training volume than traditional training methods (Little et al. 2011; Heydari et al. 2012). HIIE broadly refers to very short intense repeated bursts of vigorous exercise interspersed with bouts of active recovery or rest (Little et al. 2011; Gibala et al. 2012). The most utilized HIIE model used is the Wingate Test; this consists of 'all-out' 30 s sprints against a high resistance depending on the individuals' weight, on a specialised cycle ergometer (Gibala and McGee 2008). The typical HIIE exercise model employed is 4-6 exercise sprints alternated with a 4 minute recovery or low-intense cycling, with each session being performed 3 times week over a 2-6 week period, resulting in training sessions lasting approximately 15-30 minutes (Boutcher 2011; Gibala et al. 2012). Due to the vastly growing body of literature suggesting HIIE improves health-related markers and the Wingate model not being suitable among populations such as overweight, sedentary and elderly, studies have manipulated the duration, volume, intensity and recovery. For example studies have used 8s sprints alternated with a 12 s recovery for a 20 minute period for 15 weeks (Heydari, Boutcher and Boutcher 2013; Boutcher et al. 2011), with one using inactive, overweight men (Heydari, Freund and Boutcher 2012).

It is demonstrated the majority of phosphocreatine (PCr) stores are utilized during the first 10s of the sprint (Bogdanis et al. 1996) and 44% of the total work done occurs in the first 10s of a 30s sprint (Bogdanis et al. 1998). It is suggested that PCr resynthesis is similar to those who are sprinting for 10s to 20s when alternated with a 2 minute recovery due to the contribution to the aerobic metabolism (Bogdanis et al. 1998). When sprinting for 30s following either a 20s

or 10s, peak power output was significantly lower in the 20s sprint due to the higher production of skeletal muscle acidosis, although the mean power output was the same in each sprint (Bogdanis et al. 1998). In the present study, a 15 second sprint duration was used and has been shown reducing the Wingate sprint duration by 50% (<15s) does not impede the demand imposed on the metabolism, for example Zelt et al. (2014) recently compared 4-6 30s sprints alternated with a 4.5 minute recovery to 4-6 15s sprints alternated with a 4.75 minute recovery and found similar improvements in $\dot{V}O_{2peak,}$ lactate threshold and critical power (Zelt et al. 2014). Using smaller work-to-rest ratios during cycle sprinting induces greater adaptations to the aerobic capacity due to the shorter rest periods, placing a greater demand on the body, for example Hazell et al. (2010) reported larger $\dot{V}O_{2peak}$ adaptations when using a 1:8 work-to-rest ratio compared to a l:12 work-to-rest ratio. Furthermore, a work-to-rest ratio of 1:8 during 2 weeks of HIIE cycling induces mitochondrial activity (Burgomaster et al. 2008) a rightward shift in blood lactate curve (Jakeman et al. 2012). Sprint interval training sessions of 4-6 and progressively increasing the sprint number induces increases to cardiorespirtatory fitness, positively effecting fatigability as well as inducing positive adaptations to the metabolic and skeletal muscle oxidative capacity; for example Astorino et al. (2012) found increasing HIIE sprints by 4-to-6 over 2 weeks enhanced $\dot{V}O_{2peak,}$ O_2 pulse and power output improvements in both sexes. Furthermore, Burgomaster et al. (2008) progressively increased the sprint number from 4-to-6 over 6 weeks of sprint interval training and found increases to skeletal muscle oxidative capacity and induced specific metabolic adaptations.

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2.7 Adaptations to HIIE

2.7.1 Cardiovascular Adaptations

2.7.1.1 Comparing duration, times per week, gender use to $\dot{V}O_2$ and work to rest ratio

When HIIE is carried out over 2 weeks, there is a mixed adaptation to $\dot{V}O_{2\ peak.}$ With Whyte et al. (2010) reporting a 9% increase in obese individuals following 3 sessions a week but Burgomaster et al. (2005) found no change among healthy men and women when using the same frequency of session. When duration of the intervention is increased, there is a more robust increase in $\dot{V}O_{2\ peak}$ reported (15-24%) using 3 sessions a week (Heydari et al. 2013; Heydari et al. 2012; Trapp et al. 2008).When recovery of the intervention is manipulated; decreased, there are higher increases in $\dot{V}O_{2\ max}$ of 19% among recreationally active females (Talanian et al. (2007), although this was not found among highly trained cyclists (Laursen, Blanchard and Jenkins 2002). When the recovery is increased over a long training intervention, there are small increases in $\dot{V}O_{2\ peak}$ when using 3 sessions a week (Rakobowchuk et al. 2008).

Work to rest ratio appears to be important for $\dot{V}O_{2\ max}$ adaptations, with evidence suggesting small work to rest ratios are more beneficial for both short and long programs, for example Talanian et al. (2007) used a 2:1 work to rest ratio and found a 19% increase in $\dot{V}O_{2\ max}$ in active females over 2 weeks. Furthermore, Both Heydari et al. (2012; 2013) and Trapp et al. (2008) used a work to rest ratio of 1:1.5 and found increases of 15% (Heydari et al. 2012; 2013) over 12 weeks and a 23.8% increase (Trapp et al. 2008) over 15 weeks. In contrast larger work to rest ratios result in a smaller adaptation, for example; Whyte et al. (2010) used a 1:9 ratio and found a 9% increase in $\dot{V}O_{2\ max}$ over 2 weeks and Rakobowchuk et al. (2008) also used a 1:9 work to rest ratio and elicited a 7% increase in $\dot{V}O_{2\ max}$ over 6 weeks. The use of higher work to rest ratios has also been found to not increase $\dot{V}O_{2\ max,}$ for example Burgomaster et al. (2005) used a 1:8 work to rest ratio and found no improvements in $\dot{V}O_{2\ max.}$

2.7.1.2 Oxidative Metabolism

HIIE produces changes in mitochondrial quantity and activity as shown by the upregulation of a number of mitochondrial proteins and 24 hours post HIIE has led to increases in mitochondrial protein content (Little et al. 2011). This is thought to be regulated by an increase in peroxisome proliferator-activate receptor gamma coactivator 1-alpha (PGC-1α) transcription regulated via up-regulated of AMPK following HIIE (Di Donato et al. 2014). The activation of AMP-activated protein kinase (AMPK) following HIIE is comparable to an acute bout of endurance exercise (Nikolai, Bangsbo and Pilegaard 2003). PGC-1a is a key regulator of mitochondrial biogenesis, particularly through the transcriptional coordinating of mitochondrial genes (Wright et al. 2007). In response to 6 weeks of HIIE, increases in silent mating type information regulation 2 homolog 1 sirtuin 1 (SIRT 1) protein among men and women were found (Gurd et al. 2010). SIRT 1 and PGC-1a stimulates mitochondrial biogenesis through interaction and increases in peak oxygen consumption along with blunting the occurrence of fatigue in response by enabling replication, transcription and translation (Davis et al. 2008). The expression of PGC-1a is coupled with the mitochondrial demand for production of adenosine triphosphate (ATP), which increases when the demand for energy becomes increasingly difficult (Davis et al. 2008). Trump et al. (1996) found ATP production was predominantly aerobic during 5 x 30 s HIIE sprints. During HIIE, oxidative ATP production gradually increases and by the third out of five sprints ATP was primarily generated oxidatively (Putman et al. 1995). This demand on the aerobic energy system has been suggested as a trigger for mitochondrial adaptation (Trump et al. 1996).

2.7.1.3 Mitochondrial Adaptations

HIIE elicits improvements to the skeletal muscle oxidative capacity due to enhanced mitochondrial capacity through increased activities of mitochondrial enzymes; oxidative enzyme marker B-hydroxyacyl-CoA dehydrogenase (β-HAD) (Talanian et al. 2007; Burgomaster et al. 2008), citrate synthase (CS)

(Burgomaster et al. 2008; Gibala et al. 2008 and 2009) and pyruvate dehydrogenase (PDH) (Burgomaster et al. 2008). β-HAD and CS activities reflect cell and mitochondrial volume and the capacity for fatty acid β-oxidation (Reisch and Elpeleg 2007; Holloszy and Coyle 1984). CS has been suggested to enhance respiratory control sensitivity during exercise, which is one of the underlying mechanisms for improvements in endurance capacity (Burgomaster et al. 2005; Holoszy and Coyle 1984). PDH controls the number of carbohydrate-derived acetyl units entering the tricarboxylic acid cycle, regulating oxidative carbohydrate metabolism (LeBlanc et al. 2004).

2.7.1.4 Central Adaptations

Intensity is more important than training volume of a HIIE program to elicit $\dot{V}O_{2\ max}$ adaptations with one possible reason of increased vagal tone (Mehrdad, Boutcher and Boutcher 2013). Vagal tone is the degree of activity within the parasympathetic nervous system and following 12 weeks of HIIE there is an increase in vagal tone which influenced bradycardia (Mehrdad, Boutcher and Boutcher 2013). Increased vagal tone results in a lower heart rate which has been linked to improved gas exchange (Hayano et al. 1996). Furthermore, heart rate recovery following exercise of maximal effort is also predominantly caused by a parasympathetic reactivation, with the sympathetic activation playing a role in the initial phase of recovery (Lamberts et al. 2009). A study by Nybo et al. (2010) demonstrated that intense interval running and moderate continuous activity, but not strength training, 3 times per week for 12 weeks resulted in an increase in $\dot{V}O_{2max}$. The size of the improvement in $\dot{V}O_{2max}$ was greatest in the intense interval running group. This suggests that to elicit cardiovascular adaptations, manipulation of training intensity is more effective than manipulating the training volume (Nybo et al. 2010). Another possible reason for increases in $\dot{V}O_{2\ max}$ is improved oxygen delivery to the working skeletal muscles. It has been suggested by Oliveira et al. (2009) that improvements in $\dot{V}O_{2\ max}$ are linked to increased maximal peak $O_{2\ pulse}$ both at high and low volume HIIE, with underlying central mechanisms for increased $\dot{V}O_{2\ max}$ are partly due to increases in stroke volume (Oliveira et al. 2009). Furthermore evidence suggests a high $\dot{V}O_{2\ max}$ is partly attributed to an increase in maximal

stroke volume, increased myocardial contractility and enhanced end-diastolic volume (Schairer et al. 1992).

2.7.2 Blood Pressure Adaptations

Pulse wave velocity (PWV) is a measure of arterial stiffness and cardiovascular risk (Blacher et al. 1999; Amar et al. 2001) however 2 weeks of HIIE did not alter PWV in obese men (Whyte et al. 2010), suggesting changes to PWV following HIIE is a long term adaptation. Following 12 weeks of HIIE, arterial baroreflex sensitivity (BRS) increased by 12% (Heydari et al. 2012) and this partially defines vascular stiffness (Lipman et al. 2002). The BRS adaptation is due to the engagement of stretch-sensitive baroreceptors and decreasing baroreceptors location (Heydari et al. 2012). Whyte et al. (2010) found 2 weeks of HIIE significantly reduced systolic blood pressure 24 hours post intervention, but not 72 hours after among sedentary, overweight males who had a blood pressure lower than 160/90 mm Hg (Whyte et al. 2010). Rakobowchuk et al. (2008) found increases in popliteral artery distensibility following 6 weeks of an HIIE training program among healthy individuals however blood pressure was not statistically different (Rakobowchuk et al. 2008), suggesting 2 weeks of HIIE is not enough time to reduce blood pressure. Subsequent to 12 weeks of HIIE Nybo et al. (2010) reported 8 mm HG reductions in systolic blood pressure in conjunction with mean arterial pressure reductions (Nybo et al. 2010). Reductions in systolic blood pressure have been suggested to be due to changes in sympathetic nervous activity (Halliwill et al. 1996) and enhanced nitric oxide-mediated vasodilation (Halliwill 2001). Sixteen weeks of HIIE found reductions in norepinephrine levels, a sympathetic activity marker, among young females (Ciolac 2010). Sixteen weeks of HIIE reduced systolic and diastolic blood pressure by 10 and 6 mm Hg and improvements to endothelial function were made in conjunction to the blood pressure improvements (Tjonna et al. 2008), suggesting improved endothelial function to be linked to reductions in blood pressure.

2.7.3 Lipid and Lipoprotein Adaptations

HIIE reduces fasting plasma very low density lipoproteins triglyceride (VLDL-TG) by 30% following three sessions a week for 8 weeks of HIIE running at 60 and 90% peak oxygen consumption (Tsekouras et al. 2008). However, the same reduction in VLDL-TG was found following moderately-intensity endurance exercise (Magkos et al. 2006; Tsekouras et al. 2007), with the total energy expenditure of the high-intensity training 450-750 kcal lower compared to those in the moderately-intense endurance training (Magkos et al. 2006; Tsekouras et al. 2007). For the hypotriglyceridaemia effect to occur, there is a reliance on the total energy expenditure, duration and magnitude of the exercise (Tsetsonis and Hardman 1996) and if the energy expenditure is at a constant, the intensity and time of the exercise are interchangeable for lowering plasma TG (Tsekouras et al. 2008). HIIE is more effective for lowering circulating TG levels compared to moderately-intense endurance training due to the metabolic adaptations seen with HIIE (Tsekouras et al. 2008). Contradictory to this, Nybo et al. (2010) found prolonged training to be more effective than short-term HIIE for improving plasma lipoprotein-lipid profiles among untrained individuals, suggesting training volume is more superior than training intensity for improvements in plasma-lipoprotein lipid profiles (Durstine et al. 2002). HIIE has found no improvements to circulating TG (Wislof et al. 2007; Tjonna et al. 2008; Wallman et al. 2009). Crouse et al. (1997) found 24 weeks of cycle ergometer training at high intensity or moderate intensity among hypercholesterolemic men, no alteration in lipid concentrations (TG, total cholesterol and low-density lipoprotein (LDL)) for both training intensities occurred. Furthermore, the same author suggests if lipid alteration is the goal of training, then taking part in HIIE has no added benefit (Crouse et al. 1997). Lipid changes have been seen among normocholesterolemic participants following low and moderately intense exercise, backing up the notion of if lipid alterations are the training goal, then high intensity training is not required (Leon et al. 1979; Rauramaa et al. 1984). Crouse et al. (1997) suggests the energy expenditure threshold is higher in hypercholesterolemic men as the energy expended was above 1,050 kcal/wk, suggesting this was not enough to elicit alterations to body lipids (Crouse et al. 1997).

2.7.4 Body Composition Adaptations

Between 8 to 16 weeks of HIIE has found reductions in body fat among males and females (Heydari et al. 2012; Boudou et al. 2003; Mourier et al. 1997; Trapp et al. 2008; MacPherson et al. 2010). Reductions of 2.5 kg in subcutaneous fat, increases of 0.4 kg in the trunk fat free mass and 0.1 kg in the leg fat free mass in response to 15 weeks, three times a week of HIIE (8-s sprint followed by a 12-s low intensity cycling) among young women (Trapp et al. 2008). Heydari et al. (2012) used a similar HIIE training protocol although was 3 weeks shorter and found significant decreases of 1.2 kg in total fat free mass, 0.4 kg in the leg fat free mass and 0.7 kg for the trunk fat free mass among inactive, overweight men (Heydari et al. 2012). Increases to resting fat oxidation are a key predictor of exercise-induced fat loss (Barwell et al. 2009), suggesting long term HIIE may have implications on body composition control. Trapp et al. (2008) examined the effects of a 15 week, three times a week HIIE program among overweight women and found a 14.7% decrease in total fat mass and 4.3% reductions in body mass, suggesting 15 weeks of HIIE is an effective training method and length to control body composition (Trapp et al. 2008). MacPherson et al. (2010) found 6 weeks, three times a week of sprint interval training (SIT) and found a significant decrease of fat mass by 12.4% (1.7 kg) among men but not women and also lean mass increased by 1% (0.6kg) in both men and women (MacPherson et al. 2010). Possible factors determining fat loss may be due to gradually increasing catecholamines levels and increasing levels of lipid release during exercise (Harmer et al. 2000). Catecholamines are a key indicator to lipolysis, which is responsible for visceral fat loss (Issekutz 1978); suggesting high levels of catecholamines enhance lipolysis which increases visceral fat loss using a HIIE training program. Heydari, Freund and Boutcher (2012) demonstrated a 17% decrease in visceral fat following 12 weeks of HIIE, which is consistent with Mourier et al. (1997) who found a significant decrease in visceral fat among untrained males (Mourier et al. 1997). Heydari et al. (2012) found significant reductions in waist circumference which was correlated with reductions in visceral fat, which occurred in the first 6 weeks as waist circumference did not decrease past 6 weeks (Heydari, Freund and Boutcher 2012). Visceral fat is highly associated with developing the risk of cardiovascular disease (Heydari, Freund and Boutcher 2012), suggesting HIIE has the ability to

reduce visceral fat and waist circumference has a positive impact on cardiovascular health. Reductions in visceral fat have also been associated with post HIIE lipid and glucose metabolism enhancements (Ross et al. 2000) and reductions in visceral fat were seen in conjunction with reducing the risk of atherosclerotic cardiovascular disease (Okauchi et al. 2007). Furthermore, Tjonna et al. (2008) found in response to three times a week for 16 weeks of aerobic interval training reductions in waist circumference was parallel with circulating adiponectin levels increases during exercising (Tjonna et al. 2008). Furthermore, the same author but different study also found that aerobic interval training for 12 months significantly reduces waist circumference measurements (Tjonna et al. 2009), suggesting reductions in waist circumference are long term adaptations.

2.7.5 Cognitive Function Adaptations

Central nervous system (CNS) processing is dependent on exercise intensity, duration, type of exercise and exercise experience (Polich and Lardon 1997) and the intensity of the exercise is associated with arousal alterations of the CNS (Brisswalter et al. 2002). Working at 40 to 60% $\dot{V}O_{2\ max}$ during incremental protocols is optimal for cognitive performance (Reilly and Smith 1986), although aerobically fit individuals have made significant cognitive improvements when working at higher intensities (Brisswalter et al. 1997). Improvements to cognitive function are due to increased cerebral blood flow and increased catecholamine concentrations (Winter et al. 2007; McMorris 2009). Winter et al. (2007) found improved learning in response to 2 HIIE sessions in young males and suggested elevated catecholamine levels elicit long term learning and BDNF for short term learning (Winter et al. 2007). This is supported by Trapp, Chisholm and Boutcher (2007) and Yoshioka et al. (2001) who both found increased catecholamine levels and cerebral blood flow in response to HIIE. HIIE elicits increases in both BDNF and cortisol; however BDNF rapidly declines post-exercise whereas cortisol was found to be increased 10-15 minutes post exercise (Vega et al. 2006), indicating why BDNF is attributed to short term learning and cortisol attributed towards long term learning. Furthermore, increased neurotrophic entry to the CNS is induced by BDNF when crossing over the

blood-brain barrier (Pan et al. 1998; Kastinet al. 1999). Event-related potentials (ERP) such as P300 provide information of brain activity for the working memory (Donchin and Coles 1988). HIIE decreased P300 amplitude, indicating reduced cognitive function and decreased stimulus-evaluation process (Nishihira et al. 1999). HIIE decreased P300 amplitude among 22-23 year olds) as assessed through a go/no-go reaction time task, however P300 amplitude increased after medium-intensity exercise and no change after low-intensity exercise (Kamijo et al. 2004). Attention impairment occurred in the HIIE group and may be a mechanism for the decreased P300 amplitude (Kamijo et al. 2004).

2.7.6 Mood Adaptations

Mertesdorf (1994) suggests HIIE can be distressing for individuals however others enjoy the feelings associated with HIIE. Six weeks of HIIE had a positive effect on sedentary aging men's motivation levels and had a positive effect on health-related quality of life in the older population (Knowles et al. 2015). Furthermore, this is supported by Wisloff et al. (2007) and Nilsson, Westheim and Risberg (2008) who both found HIIE have positive psychological effects on older populations, especially those with clinical conditions. Six weeks of HIIE in collegiate cyclists found profile of mood states (POMS) did not significantly increase during and after the one week taper, however a large effect size was found (Martin et al. 2000). Based on the large effect size, HIIE and POMS scores were negatively associated and alterations in mood were due to the vigorous and fatigue associated with HIIE (Martin, Andersen and Gates 2000). Interestingly, resting cortisol increased 150% above resting levels in some individuals 36 hours following training (Martin, Andersen and Gates 2000). Undertaking acute bouts of HIIE running is perceived to have greater enjoyment compared to moderate-intensity continuous running along with higher feelings of physical exertion among recreation active men (Barlett et al. 2011), which has also been found in recreationally active individuals (Raedeke, 2007). Wisloff et al. (2007) and Tjonna et al. (2008) both found HIIE elicits greater feelings of enjoyments compared to continuous exercise and this is due to the continuous exercise being reportedly boring. Individuals working at higher intensities already feel comfortable; however research suggests no mood differences between less active

and more active individuals (Steptoe, Kearsley and Walters 1993; Felts and Vaccaro 1988; Kraemer et al. 1990). Negative mood states can be increased further when the cadence during HIIE is over 100W (Steptoe and Cox 1988; Steptoe and Bolton 1988).

2.7.7 Limitations to HIIE

As previously mentioned the most utilized HIIE model used is the Wingate Test; consisting of 'all-out' 30 s sprints against a high resistance (Gibala and McGee 2008). Performing this type of exercise requires the use of a specialized ergometer bike, high levels of motivation and is suggested not to be adaptable to the general population (Gibala and McGee 2008). Furthermore, HIIE also requires positive individual supervision throughout each session (Jong et al. 2003). Providing a standard Monark Ergomedic peak bike has a cost of around £3900 (HaB Direct, 2016) and barriers such as income (Burton et al. 2003) and socioeconomically deprived (Masse and Anderson 2003) are reasons why males and females participate less in physical activity. This demonstrates the barriers HIIE imposes as a training modality and expresses the difficulties the general population face to the exposure of HIIE. Furthermore, overweight youths reported a lack of resources (Zabinski et al. 2003) as a barrier to physical activity and given the cost of equipment for HIIE, HIIE seems an unreasonable training modality for this particular population also. Providing the biggest drop-out rates of physical activity occur at adolescence and some female individuals may only partake in physical activity within school (Coackley and White 1992) and HIIE is deemed as distressing (Mertesdorf, 1994), this highlights the limitations HIIE imposes on these particular populations at the given time periods. Furthermore, due to HIIE has been reported to be distressing (Mertesdorf, 1994), this also highlights why HIIE may not be used for overweight individual's who do not enjoy the physical symptoms such as sweating associated with exercising (Vandelanotte et al. 2008). Given overweight individuals have fat (Bell et al. 200) and the physical symptoms associated with exercise (Vandelanotte et al. 2008) as extra motivators for participation in physical activity, and HIIE requires high levels of individual motivation (Gibala and McGee 2008), this highlights

the issues HIIE has as a training method for increasing physical activity rates among overweight populations.

2.8 The Use of Females in Studies

The menstrual cycle is a 28 day cycle with a number of different phases; beginning with menstrual phase also known as menstruation; the shedding of the endometrium typically lasts between 4-6 days (Farage et al. 2009). This leads into the follicular phase (proliferative phase) which lasts from day 7 to 14, with the menstrual phase and early follicular phase characterised by low progesterone and estrogen levels (Farage et al. 2009). The follicular phase peaks at the ovulation phase, which is day 14 and circulating estradiol increases the week prior to ovulation and peaks the day before ovulation (Farage et al. 2009). Day 15 to 28 is known as the luteal phase (secretory), with estradiol increasing during the early stages of the luteal phase and late in the luteal phase, estrogen, progesterone and estradiol decrease, initiating the cycle and menstruation again (Farage et al. 2009). In terms of health irregular or loss of menstruations through adolescence may be a vital sign for health conditions suffered during adulthood such as atypical blood pressure, respiratory control and heart rate (Diaz et al. 2006). It has been demonstrated elite athletes feel better during the follicular phase compared to the luteal phase (Kishali et al. 2006). Despite this, evidence suggests the luteal and follicular phase have little or no effect on endocrine and substrate exercise-induced responses (Bonen et al. 1983). However, in a 24h fasted state growth hormone (GH) response were increased, with reduced luteinizing hormone and augmentation of progesterone (not exercise-induced) and pre-exercise nutrition and menstrual cycle phase affecting these alterations to hormones (Bonen et al. 1983). Furthermore, if food ingestion increases during the menstrual cycle by 12.5% (Pliner and Fleming 1983) to 38% (Dalvit 1981), a two phase alteration occurs in energy balance, with a negative balance occurring during the follicular phase and a positive during the luteal phase (Bisdee et al. 1989). Early follicular and mid-luteal phase have no impact on performance tests and cardiorespiratory parameters, although the aerobic capacity can be somewhat affected by steroid hormone increases during the ovulatory phase (Lebrun et al. 1995). During the luteal phase, females have an increased core temperature

which has adverse effects on exercise efficiency and increases the demand for oxygen consumption during exercise (Lebrun et al. 1995).

2.9 Barriers to Exercise

Despite time and injury previously being discussed as barriers to traditional training methods, there are a wider range of barriers to exercise modalities which seems to be more robust in females; with reports demonstrating only 59% of high school females adhering to the recommended physical activity guidelines a week, with males showing a greater percentage (73%) meeting these recommendations (Grunbaum et al. 2003). Ethnicity and income have both been associated with engagements in physical activity in female adults (Masse and Anderson, 2003) and both sexes are expected to participate less in physical activity if socioeconomically deprived (Burton et al. 2003). Furthermore, Taylor et al. (1999) found females were less likely to participate in physical activity due to appearance maintenance, and it was also found that African American high school females disliked the physical symptoms associated with physical activity such as perspiration and sweating (Perry and Kelder, 1992). However white females were more likely to partake in physical activity due to the physical attractive maintenance exercise prescribes (Mariane et al. 2006). Higher BMI levels have been associated with higher levels of physical inactivity, which were more robust African American females compared to their white equivalents; with race ascribed as reasons for the differences found (Felton et al. 2002). A cited barrier for physical activity in overweight female youths was there concern for body consciousness during physical activity (Zabinski et al. 2003). Furthermore, overweight youths also had higher rates of a lack of resources compared to non-overweight children (Zabinski et al. 2003). A perceived barrier for overweight individuals is that fat is an extra motivator (Bell et al. 200) and exercising was found to be uncomfortable and causes discomforts such as sweating, also found as not motivating for participating in physical activity (Vandelanotte et al. 2008). Furthermore, it has been reported that females living a sedentary lifestyle experience more barriers in comparison to individuals who regularly participate in physical activity (Kowal and Fortier, 2007). Providing the biggest decline in female physical activity drop-out rates occurs at adolescence, partaking in

physical education during school may be the only opportunity females have to take part in physical activity (Coakley and White, 1992), therefore addressing and overcoming the barriers at this time period seems to be imperative for female physical activity drop-out rates, health promotion and future physical activity participation.

3 Methodology and Data Analysis

3.1 Importance of maximal $\dot{V}O_2$

$\dot{V}O_{2\ max}$ is the most widely used parameter that indirectly assesses the cardiovascular and respiratory systems' ability to transport oxygen to the working skeletal muscles (Day et al. 2003). Improvements in $\dot{V}O_{2\ max}$ are due to muscular oxidative metabolism adaptations (Harmer et al. 2008), enhanced mitochondrial oxidative capacity (Slordalh et al. 2005), improved oxygen transport (Helgerud et al. 2007), cardiac adaptations to exercise (Abe et al. 2010), increased capillarization of the muscle (Jensen et al. 2004) and reduced arterial stiffness (Ferreira et al. 2003). $\dot{V}O_{2\ max}$ as a health outcome is the strongest acute predictor of mortality, especially when low (Tjonna et al. 2013; Mancini et al. 1991). A $\dot{V}O_{2\ max}$ test assesses the individuals' ability to attain the highest value of $\dot{V}O_2$, a $\dot{V}O_{2\ peak}$ test assesses an individuals' capacity to attain the highest value of $\dot{V}O_2$ in the exercise test used (Whipp 2010). $\dot{V}O_{2\ peak}$ is used for individuals suffering from chronic diseases, not $\dot{V}O_{2\ max}$ due to the health risks associated (Day et al. 2003). Deterioration in performance measured through exercise testing, particularly $\dot{V}O_{2max}$ can indirectly indicate organ failure, especially liver and heart (Mancini et al. 1991), provide a chronic prognostic tool for coronary artery disease patients and low ejection fraction (Pilote et al. 1989) and allow cardiovascular reserve assessments in patients with heart failure (Weber and Janicki 1985).

3.2 Participants

Young healthy female participants were recruited through the use of poster advertisement (Appendix 1); individuals who had suffered from a lower limb injury within the last 6 months or had a chronic health condition were excluded from the study. Participants standing height and weight were determined before the intervention began, from which their body mass index (BMI) was calculated by dividing their weight (kg) by height (m). Participants were then split into three

groups, control and two training groups. Training groups were then allocated according to the participants BMI, the normal BMI group (n=7, age, 21 ± 0.5years; height, 168 ± 6cm; pre weight, 58 ± 1.8kg; post weight, 60 ± 3.1kg; pre BMI, 20 ± 0.7 $kg.m^{-2}$; post BMI, 21 ± 0.9 $kg.m^{-2}$) or the overweight BMI group (n=6, age, 23 ± 3.7years; height, 175 ± 12cm; pre weight, 93.8 ± 28kg; post weight, 94 ± 31kg; pre BMI, 30 ± 6$kg.m^{-2}$; post BMI, 30 ± 7$kg.m^{-2}$). The control group was used (n=6, age, 22 ± 1years; height, 169 ± 5cm; pre weight, 63 ± 5kg; post weight; pre BMI, 22 ± 1$kg.m^{-2}$; post BMI, 22 ± 1$kg.m^{-2}$) to determine natural change in outcome measures. Participants had no experience prior to the start of the intervention of cycle based HIIE training. The experimental protocol and risks associated with the protocol were fully explained verbally and in writing (Appendix 2) to participants and informed consent was attained prior to the start of the experiment (Appendix 3). Participants completed a physical activity readiness questionnaire (PAR-Q) before the start of experiment to screen for health implication that may place the participants at risk to partake in the study (Appendix 4) (McKay et al. 2001; American College of Sports Medicine 2006). This study was approved by the Social and Health Sciences ethics committee at Abertay University and was performed according the declaration of Helsinki (Nybo et al. 2010; Goodyear et al. 2007).

3.3 Baseline Testing

Prior to the start of the training period, participants had their height measured using Seca 264 Standiometer (Seca, Seca, United States). Participants attended the subsequent baseline tests following an overnight fast (Fisher et al. 2015). Participants refrained from alcohol and caffeine consumption and from taking part in physical activity 24 prior to testing.

Immediate Word Recall (Episodic Memory)

Participants were given a unique set of fifteen words (Appendix 5). Participants then had 60 s to record as many words as they could recall. The fifteen words

were different for pre and post-tests but were matched for word length, frequency and familiarity (Durga et al. 2007).

Verbal Fluency (Executive Function/Semantic Memory)

Participants were presented with three unique letters and given 60 s to write down as many words beginning with that letter (Appendix 6) (Tombaugh et al. 1999).

Segmental Analysis and Body composition

Body composition and segmental analysis were determined by using a Tanita, Body Composition Analyzer (Maeno-cho, Habashi-Ku, Tokyo, Japan). Participants removed shoes and socks and stood barefooted onto the 4 plated electrodes and placed 2 hands on the hand plated electrodes (Shafer et al. 2009). Participants were asked to place 2 hands on the bipolar handgrips for no longer than 2 minutes; the segmental compositions were obtained and recorded. Estimations of body fat mass (kg), trunk fat (%), left leg fat (%), right leg fat (%) and BMI were given and recorded.

Perception of Happiness

Participants were invited to complete and return the Oxford Happiness Questionnaire (OHQ); a single answered questionnaire with a six-point Likert scale; ranging from ‘strongly agree’ to ‘strongly disagree’ (Appendix 7). Prior to administration, answers marked R were reversed and changed; ‘1’ to a ‘6’, ‘2’ to a ‘5’, ‘3’ to a ‘4’, ‘4’ to a ‘3’, ‘5’ to a ‘2’, ‘6’ to a ‘1’. Scores were then calculated, with 1-2; meaning not happy, 2-3; somewhat happy, 3-4; not particularly happy or unhappy, 4; somewhat happy and moderately happy, 4-5; rather happy, pretty happy, 5-6; very happy and 6; too happy (Hills and Argyle 2002).

Subjective Happiness

Participants were invited to complete and return a Subjective Happiness Questionnaire; a 4-item questionnaire with a seven-point Likert scale; ranging from 1 to 7 and lower scores indicating smaller happiness and higher score indicating greater happiness (Appendix 8) (Lyubomirsky and Lepper 1999).

Waist to Hip Ratio

Waist/hip ratio was measured using a Rollflix tape measure (HaB direct, Germany). Participants were measured at the umbilicus level and measured at hip and maximum buttocks circumference. Waist to hip ratio was calculated using a standard formula:

waist measurement (cm) / hip measurement (cm) (Taylor et al. 2000).

Blood Pressure

Once arrived into the laboratory, participants were seated and rested for 15 minutes. Blood pressure for both arms was measured by using an automatic sphygmomanometer (Microlife, Twin200ABI, Switzerland). Participants were asked to place both arms flat on the table surface whilst sat down, cuffs were placed on both arms and measurements were taken 3 times and averages from both arms were given and recorded. Blood pressure (mean arterial pressure) was calculated using a standard formula; ((systolic pressure – diastolic pressure)/3 + diastolic pressure) (Fisher et al. 2015).

Triglycerides

Blood was drawn using an Accu-Chek to prick the participant's forefinger; the first drop discarded and then the second drop was extracted using a Capillary Tube (Heparinized Plastic Clad 15uL, Polymer Technology Systems, Inc. Indianapolis) and plunger (Polymer Technology Systems, Inc. Indianapolis).

Blood was plunged onto a PTS test strip (CardioChek, Polymer Technology Systems, Inc.) attached into a CardioChek circulating triglyceride count (Polymer Technology Systems, Inc.). One minute later circulating triglycerides measurement were given and recorded. (Luzi et al. 2003).

Delayed Word Recall (Episodic Memory)

Fifteen minutes after the initial memory test, participants were given 60 s to write down as many words presented previously as possible (Durge et al. 2007).

$\dot{V}O_{2\ peak}$

On separate occasions, participants' performed an exhaustive continuous incremental cycle test on a cycle ergometer (Monark Ergomedic 835, Monark Exercise AB, Sweden) to determine pulmonary $\dot{V}O_{2\ peak}$ using an automated gas analysis system (Metalyzer 3B gas analyser, Cortex, Leipzig, Germany). Participants had their weight measured using Seca Weigh Scales (Seca 875 (III) Floor scale, USA) and each participant completed a 5 minute warm up, cycling at and maintaining a speed of 60 rpm with only the cradle applied to the wheel's resistance. Once started, participants were required to maintain a cadence of 70 rpm or above throughout, every 2 minutes 0.5 kg was added to the cradle. Participants continued until 70 rpm could no longer be sustained or they chose to stop. Verbal encouragement was given throughout and time was recorded using a Quantu, 5500 stop clock (EA Combs Ltd, UK) to measure time to exhaustion. $\dot{V}O_{2peak}$ was taken as the highest 30 second average across the test.

Heart Rate Recovery from Sprints

During the first and last training sessions, heart rate recovery was assessed. Firstly the Zephyr ™ BioHarness ™ (Zephyr Technology Ltd, Auckland, New Zealand) attached to the wetted Zephyr ™ BioHarness ™ fabric strap. Participants were asked to place strap underneath their clothing with the Zephyr BioHarness logo placed in front on the sternum. The Omni Directional Antenna

(Zephyr Technology Ltd, Auckland, New Zealand) allowed the heart rate to be read through the wireless Bluetooth signal and each participant's heart rate was monitored throughout both the first and last training session (Selig et al. 2004). Heart rate recovery was calculated by; adding each participant's heart rate at every second of the sprints and recovery, the average of every second was taken by dividing by the number of participants to give the final average group heart rate reading. This was calculated for each group at the first and last sprinting session.

3.4 High-Intensity Interval Exercise Training Protocol

The participants performed 15 s HIT sprints 3 times a week for 6 weeks, against a resistance of 7% body weight, alternated with 2 minute recovery between bouts. During week 1 and 2; participants performed 4x15 s sprints; week 3 and 4 participants performed 5x15 s sprints; week 5 and 6 participants performed 6x15 s sprints. Prior to the start of the bouts, participants warmed up, cycling for 5 minutes on the workload of the participant's choice (Trapp et al. 2008). Before the resistive load was applied to the flywheel, participants were given a 3 s countdown before cycling 'all out' for 15 s. For each bout, participants had to cycle to 110 rpm for the weight to drop and be applied to the cycle ergometer's cradle. Once each sprint had been completed, the resistive load was removed from the flywheel. Verbal feedback was given throughout each sprint and towards the latter stages of the sprints, verbal encouragement was given along with clear instructions to stop sprinting (McKay et al. 2009).

Post-training Assessment

Baseline measurements were conducted 7 days after completion of the last sprint, to determine whether the training programme had induced any potential changes.

3.5 Data Analysis

All data is reported as means ± standard deviation. Data was checked for skewness and kurtosis and these values did not exceed twice the standard error, therefore the data was deemed to be normally distributed. A 3x2 repeated measures analysis of variance (ANOVA) was used to compare the three different groups by time and group by time. Paired samples within independent t-tests were run to assess any significance between pre and post measures. An unpaired samples independent t-test was run to asses any significance between groups. Significance was accepted at $p<0.05$. Cohen's effect size was defined at: $d<0.2$, trivial effect; 0.2-0.5, small effect; 0.6-1.1, moderate effect; and 1.2-1.9, large effect (Cohen 1998).

3.6 Power calculation and participant recruitment and retention limitations

A small sample size can negatively affect the reading of the p-value and give less power to detect significant changes, whereas a larger sample size provides more power to detect significant changes (Wacholder et al. 2004). Furthermore, a larger sample size can give a larger margin of error, causing the expression of false positives within the p-value (Wacholder et al. 2004). Experimental mortality (dropout rates) can affect the internal validity of the study due to the unequal dropout and make-up of the different group numbers (Higgins et al. 2011). Individual's suffering from non-communicable diseases are at risk of injuries (Xiang et al. 2005), which may partly explain the higher drop-out rates due to injury, expressed among the 'overweight' group in the present study. This subsequently lead to an unequal make-up of participants across the three groups; affecting the internal validity of the study and giving less power to detect significant changes. There are a larger number of individual's classed within the 'normal' category, based on body mass index (BMI) compared to those classed within the 'overweight and 'obese' category (Statistics on obesity, physical activity and diet, 2016). This raises the problems the present study faced with recruiting a larger sample size for the 'overweight' group due to a lower number of individuals classed within this category and providing more power to detect

changes within the p-value. Furthermore, a larger sample size may have been recruited in the ‘normal’ group in the present study due to a higher number of individuals classed within this category, however this may of caused a larger margin of error within the p-value and affected the internal validity due to an unequal make-up of groups; expressing the importance and difficulty the present study had of finding a balance of a sufficient sample size for power to detect changes, controlling the internal validity and having a similar make-up of groups.

4 Results

$\dot{V}O_{2peak}$

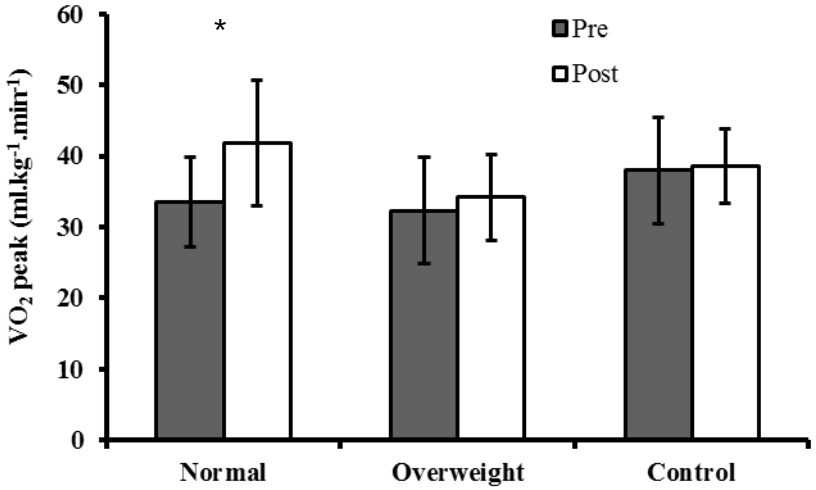


Figure 1: A comparison of the $\dot{V}O2peak$ for the normal, overweight and control groups pre-tests and post-tests (* p<0.05).

There was a main effect found for time, F (1, 16) = 12.527, p = 0.003 and a significant group x time interaction effect, F (2, 16) = 5.587, p = 0.014. Six weeks of HIIE showed a significant increase only in the normal (N) group for $\dot{V}O_{2peak}$, (Figure 1, N pre; 33.7 ± 6.3 $ml.kg^{-1}.min^{-1}$, N post; 41.8 ± 8.8 $ml.kg^{-1}.min^{-1}$; p = 0.008). No significant difference was found in the overweight (O) group (Figure 1, N pre; 32.3 ± 7.5$ml.kg^{-1}.min^{-1}$, O post; 34.2 ± 6.1$ml.kg^{-1}.min^{-1}$; p = 0.255). No significant difference was found in the control (C) group (Figure 1, C pre; 37.9 ± 7.4$ml.kg^{-1}.min^{-1}$, C post; 38.6 ± 5.2$ml.kg^{-1}.min^{-1}$; p = 0.654). After six weeks of HIIE training the N group found a 25.2 ± 17.2% increase, the O group had a 7.4 ± 11.6% increase and a 2.8 ± 9.3% increase was found in C group. A significant difference in magnitude of change was found between the N and C groups (p = 0.018). However no significant difference were found between the N and O groups (p = 0.061) or between the O and C (p = 0.466).

Time to Exhaustion

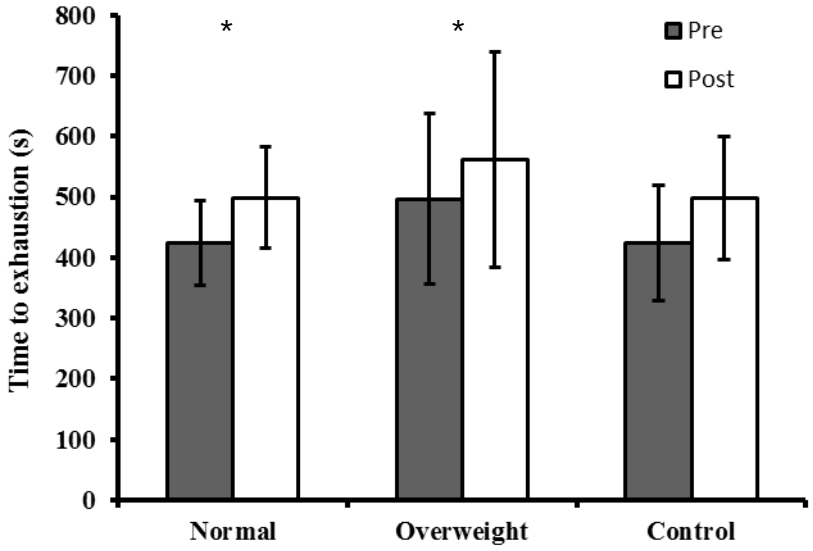


Figure 2: A comparison of the time to exhaustion (TTE) for the normal, overweight and control groups pre-tests and post-test (* p<0.05).

There was a main effect for time, F (1, 16) = 17.879, p = 0.001 and a significant group x time interaction effect, F (2, 16) = 5.442, p = 0.016. Six weeks of HIIE showed a significant increase in TTE for the N group, (Figure 2, N pre; 424 ± 69s, N post; 498 ± 84s; p = 0.005) and in the O group (Figure 2, O pre; 497 ± 140s, O post; 561 ± 177s; p = 0.051). No significant difference was observed in the C group (Figure 2, C pre; 500 ± 96s, C post; 495 ± 102s, p = 0.563). After six weeks of HIIE training, there was a 17.9 ± 11.8% increase in TTE in the N group, a 12.4 ±11.8% increase in the O group, and no difference in the C group -1.1 ± 3.4%. A significant difference in magnitude of change was found between the N and C groups (p = 0.003) and the O and C groups (p = 0.022). However no significant interaction between the N and O groups was found (p = 0.422).

Table 1: A comparison of right and left arm blood pressure for the normal, overweight and control group pre-test and post-test.

Blood Pressure (mm Hg)	Normal (n = 7)		Overweight (n = 6)		Control (n = 6)	
	Pre	Post	Pre	Post	Pre	Post
Right arm systolic	117.6 ± 5.4	112.4 ± 6.4	122.5 ± 8.8	116.5 ± 6.3	117.6 ± 5.4	116.8 ± 7.9
Right arm diastolic	70.6 ± 6.5	68.1 ± 5.1	73.3 ± 6.2	69.3 ± 5.6	73.2 ± 6.3	71.8 ± 7.2
Left arm systolic	116.7 ± 8	112.4 ± 7*****	125 ± 8.2	119.3 ± 7.4*****	118 ± 5.8	116.2 ± 8.8
Left arm diastolic	70.1 ± 3.1	68.6 ± 3.9	75.8 ± 4.4	71.8 ± 5.2	73.6 ± 6.3	71.6 ± 7.4

*Values reported as means ± SD, Significant finding = * p = < 0.05.*

There was a main effect for time, F (1, 16) = 6.48, p = 0.022, however no interaction effect was found for group x time, F (2, 16) = 1.003, p = 0.389. Six weeks of HIIE did not significantly alter right arm systolic blood pressure for the N group, (p = 0.140), although a moderate effect size was found (d = 0.87). No significant difference was found in the O group (p = 0.075), however a moderate effect size was found (d = 0.78). No significant difference was found in the C group (p = 0.718). After six weeks of HIIE, there was a 4.2 ± 6.4% decrease in right arm systolic blood pressure in the N group, a 4.7 ± 5.1% decrease in the O group and no decrease in the C group 0.7 ± 4.4%. No significant difference in magnitude of change was found between the O and N groups (p = 0.886), nor between the N and C groups (p = 0.283) and between the C and O groups (p = 0.180).

There was no main effect for time, F (1, 16) = 2.424, p = 0.139 and no interaction effect for group x time, F (2, 16) = 0.207, p = 0.815. Six weeks of

HIIE did not significantly alter right arm diastolic blood pressure for the N group, ($p = 0.140$). No significance difference was found in the O group for right arm diastolic blood pressure, ($p = 0.239$). No difference was in the C group ($p = 0.655$). Following six weeks of HIIE, there was a $2.9 \pm 10.1\%$ decrease in right arm diastolic blood pressure in the N group, a $5 \pm 9.9\%$ decrease in the O group, and no difference in the C group, $1.6 \pm 8.9\%$. No significant difference in magnitude of change was found between the N and C groups ($p = 0.814$), between the N and O groups ($p = 0.707$) and between the O and C groups ($p = 0.543$).

There was a main effect for time, $F (1, 16) = 8.281$, $p = 0.011$, although no interaction effect was found for group x time, $F (2, 16) = 0.644$, $p = 0.538$. Six weeks of HIIE significantly altered left arm systolic blood pressure for the N group, ($p = 0.037$). A significant difference was found in the O group ($p = 0.001$). No difference was found in the C group, ($p = 0.649$). After six weeks of HIIE, there was a $3.6 \pm 3.4\%$ decrease in left arm systolic blood pressure in the N group, a $4.5 \pm 1.5\%$ decrease in the O group, and no difference in the C group $1.43 \pm 7.75\%$. No significant difference in magnitude of change was found between N and O groups ($p = 0.555$), between the N and O groups ($p = 0.517$) and between the O and C groups ($p = 0.363$).

There was no main effect for time, $F (1, 16) = 3.069$, $p = 0.099$ and no interaction effect for group x time, $F (2, 16) = 0.268$, $p = 0.768$. Six weeks of HIIE did not significantly alter left arm diastolic blood pressure for the N group, ($p = 0.402$). No significant difference was found in the O group, ($p = 0.151$). No difference was found in the C group, ($p = 0.573$). In response to six weeks of HIIE, left arm diastolic blood pressure decreased by $2.1 \pm 6.6\%$ in the N group, $5.10 \pm 7.5\%$ in the O group and $2.3 \pm 10.9\%$ in the C group. No significant difference in magnitude of change was found between N and C groups ($p = 0.962$), between the N and O groups ($p = 0.458$) and between the O and C groups ($p = 0.621$).

Perception of Happiness

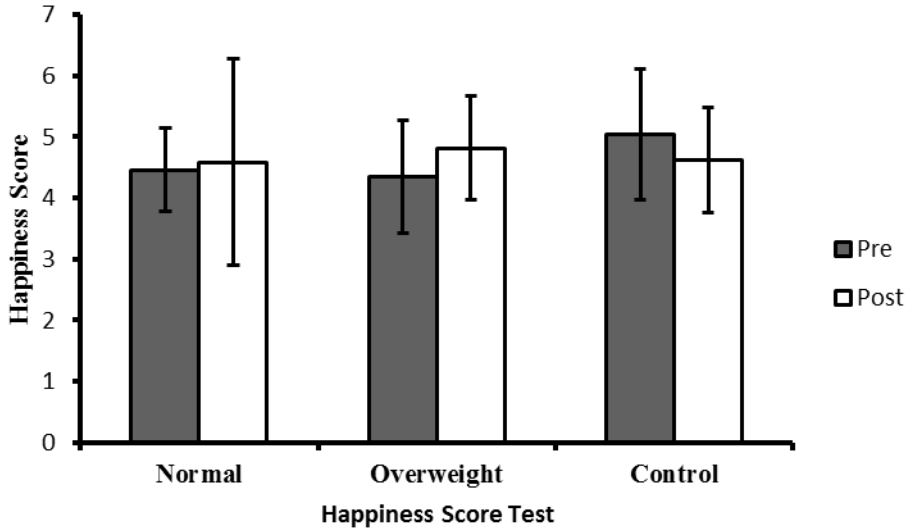


Figure 3: A comparison of perception of happiness scores for the normal, overweight and control groups pre-test and post-test (* p<0.05).

There was no main effect for time, F (1, 16) = 0.091, p = 0.767 and no group x time interaction effect, F (2, 16) = 1.566, p = 0.239. Six weeks of HIIE showed no significant difference in perception of happiness scores in the N group, (Figure 3, N pre; 4.5 ± 0.7, N post; 4.6 ± 1.7; p = 0.775). No difference was found in the O group, (Figure 3, O pre; 4.4 ± 0.92, O post; 4.8 ± 0.8; p = 0.216), although a small effect size was found (d = 0.53). No difference was found in the C group, (Figure 3, C pre; 5.03 ± 1.06, C post; 4.6 ± 0.8; p = 0.086). After six weeks of HIIE, the N group had no alteration in perception of happiness score 0.37 ± 26.1%, the O group had a 13.3 ± 21.7% increase in the happiness score and the C group had a 7.7 ± 8.5% decrease in the perception of happiness score. A significant difference in the magnitude of change was found between the O and C groups (p = 0.052), however no difference in the magnitude of change was found between the N and O groups (p = 0.358) and the N and C groups (p = 0.485).

Triglycerides

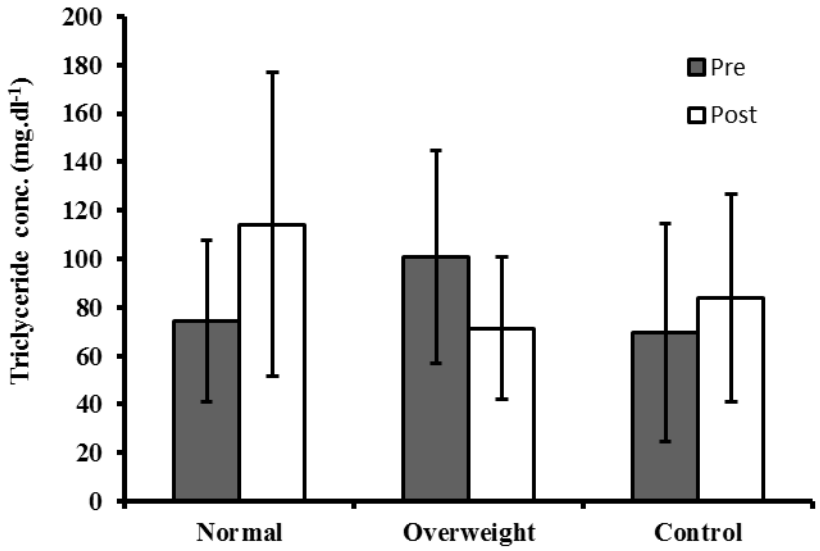


Figure 4: A comparison of triglycerides for the normal, overweight and control group pre-test and post-test (* p<0.05).

There was no main effect for time, F (1, 16), = 0.452, p = 0.511 and no group x time interaction effect, F (2, 16), = 2.828, p = 0.089. Six week of HIIE found no difference in TG for the N group, (Figure 4, N pre; 74.3 ± 33.3mg/dl, N post; 114.1 ± 62.7mg/dl; p = 0.171). No significant difference was found in the O group, (Figure 4, O pre; 101 ± 43.9mg/dl, O post; 71.3 ± 29.4mg/dl; p = 0.064), although a moderate effect size was found (d = 0.79). No difference was found in the C group, (Figure 4, C pre; 69.3 ± 44.9mg/dl, C post; 83.6 ± 42.9mg/dl; p = 0.509). Six weeks of HIIE found a 63.7 ± 87.6% increase in TG for the N group, a 21.69 ± 33.06% decrease was found in the O group, and a 38.9 ± 86.9% increase was found in the C group. No difference in the magnitude of change was found between the N and O groups (p = 0.984), between the N and C groups (p = 0.376) and between the O and C groups (p = 0.540).

Subjective Happiness

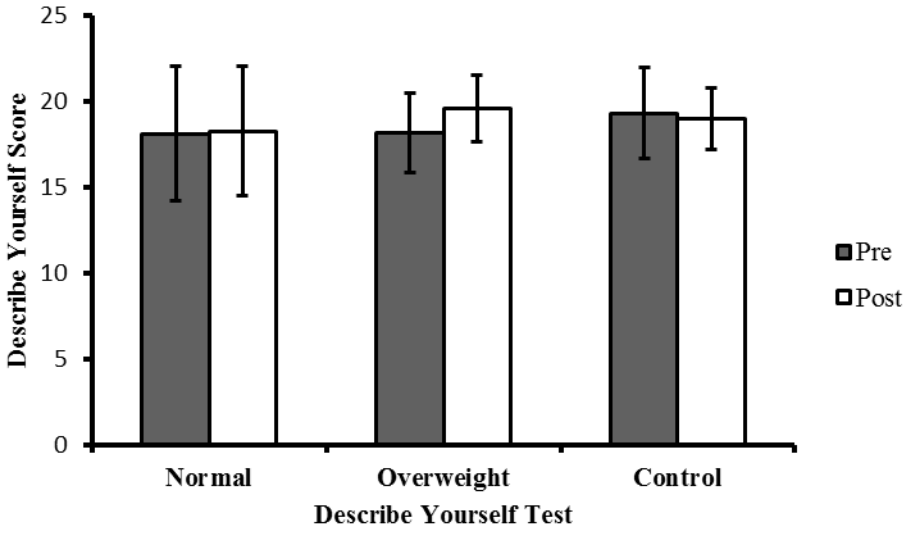


Figure 5: A comparison of subjective happiness scores for the normal, overweight and control group pre-test and post-test (* p<0.05).

There was no main effect for time, F (1, 16), = 0.353, p = 0.561 and no group x time interaction effect, F (2, 16), = 0.849, p = 0.447. Six weeks of HIIE found no difference in subjective happiness scores for the N group, (Figure 5, N pre; 18.14 ± 3.93, N post; 18.28 ± 3.77; p = 0.689). No difference was found in the O group, (Figure 5, O pre; 18.16 ± 2.31, O post; 19.6 ± 1.94; p = 0.405), although a moderate effect size was found (d = 0.67). No difference was found in the N group, (Figure 5, C pre; 19.33 ± 2.65, C post; 19 ± 1.78; p = 0.610). Six weeks of HIIE found a 12.75 ± 43.70% increase in the subjective happiness scores in the O group, no difference was found in the N group 1.05 ± 4.83% and no difference was found in the C group, 1.02 ± 7.97 decrease. No difference in the magnitude of change was found between the N and O groups (p = 0.420), between the N and C groups (p = 0.574) nor between the O and C groups (p = 0.532).

Table 2: A comparison of body composition parameters for the normal, overweight and control group pre-test and post-test.

Parameter	Normal (n = 7)		Overweight (n = 6)		Control (n = 6)	
	Pre	Post	Pre	Post	Pre	Post
Fat Mass (kg)	12.1 ± 1.4	13.7 ± 2.8	33.2 ± 17.4	33.9 ± 19.9	16 ± 1.1	15.9 ± 1.1
Trunk Fat (kg)	15.6 ± 1.7	18.6 ± 4.3	31 ± 6.5	23.6 ± 5.9	21.9 ± 2.3	21.5 ± 3
Left Leg Fat (%)	28.6 ± 5.7	28.8 ± 5.9	37.8 ± 9.7	40.2 ± 8.9*	31.4 ± 3.3	31.9 ± 3.2*
Right Leg Fat (%)	27.8 ± 6.5	28.9 ± 5.9	38.03 ± 10.2	40.06 ± 9.6	30.9 ± 3.2	31.1 ± 3.4
WTHR (cm)	0.82 ± 0.03	0.81 ± 0.03*	0.8 ± 0.05	0.8 ± 0.07	0.8 ± 0.03	0.8 ± 0.02

*Values reported as means ± (SD), Significant finding = * p = < 0.05, Abbreviations: WTHR, waist to hip ratio.*

There was no main effect for time, F (1, 16), = 0.003, p = 0.958 and no group x time interaction effect, F (2, 16), = 0.009, p = 0.991. Six weeks of HIIE did not alter fat mass in the N group, (p = 0.884). No difference was found in the O group, (p = 0.956). No difference was found in the C group, (p = 0.930). Six weeks of HIIE found a 0.86 ± 8.7% decrease in fat mass in the N group, a 1.34 ± 7.5% decrease was found in the O group, and a 0.02 ± 8.4% decrease were found in the control group. No difference in the magnitude of change was found between the N and O groups (p = 0.919), between the N and C groups (p = 0.863) and between the O and C groups (p = 0.781).

There was no main effect for time, F (1, 16), = 1.484, p = 0.241 and no group x time interaction effect, F (2, 16), = 1.486, p = 0.256. Six weeks of HIIE found no significant difference between trunk fat in the N group, (p = 0.267). No

difference was found in the N group, (p = 0.267). No difference was found in the O group, (0.291), although a large effect size was found (d = 1.2). No difference was found in the C group, (p = 0.610). Six weeks of HIIE found a 12.4 ± 24.04% decrease in trunk fat % in the O group, however no alterations were made in the C group with a 0.43 ± 9.1% decrease and no alteration were made in the N group with a 2.1 ± 9.1% increase. No difference in the magnitude of change was found between the N and O groups (p = 0.172), between the N and C groups (p = 0.442) and between the O and C groups (p = 0.351). Although no significant improvements in body composition were made, a large effect size (1.2) was observed for trunk fat (kg) in the O group,

There was no main effect for time, F (1, 16), = 1.098, p = 0.310, however a significant group x time interaction effect was found, F (2, 16), = 6.834, p = 0.007. Six weeks of HIIE did not alter left leg fat mass in the N group, (p = 0.641). A significant difference was found in the O group, (p = 0.026). A significant difference was found in the C group, (p = 0.040). Six weeks of HIIE found a 1.4 ± 4.5% decrease in left leg fat mass in the N group, a 4.3 ± 4.2% increase was found in the O group, and a 1.7 ± 1.5% increase was found in the C group. A significant difference between the magnitude of change was found for the N and O groups (p = 0.039), however no difference was found between the N and C groups (p = 0.139) and between the O and C groups (p = 0.182).

There was a main effect found for time, F (1, 16), = 6.274, p = 0.023, however no group x time interaction effect was found, F (2, 16), = 1.389, p = 0.278. Six weeks of HIIE did not significantly alter right leg fat mass in the N group, (p = 0.088). No difference was found in the O group, (p = 0.102). No difference was found in the C group, (p = 0.770). Six weeks of HIIE found a 1.7 ± 2.4% increase in right leg fat mass in the N group, a 3.1 ± 4.3% increase was found in the O group, and no alteration was made in the C group, 0.8 ± 5.8% decrease. No difference in the magnitude of change was found for the N and O groups (p = 0.498), between the N and C groups (p = 0.310) and between the O and C groups (p = 0.220).

There was no main effect for time, $F(1, 16) = 2.416$, $p = 0.140$ and no group x time interaction effect found, $F(2, 16) = 0.936$, $p = 0.413$. Six weeks of HIIE significantly altered WTHR in the N group, ($p = 0.033$). No difference was found in the O group, ($p = 0.363$). No difference was found in the C group, ($p = 0.787$). Six weeks of HIIE found a 1.2 ± 1.2% decrease in WTHR in the N group, a 1.5 ± 3.7% in the O group and no alteration was made in the C group, 0.3 ± 1.8% decrease. No difference in the magnitude of change was found between N and O groups ($p = 0.857$), between the N and C groups ($p = 0.103$), and between the O and C groups ($p = 0.315$).

Table 3: A comparison of cognitive function parameters for the normal, overweight and control group pre-test and post-test.

Parameter (words)	Normal (n = 7)		Overweight (n = 6)		Control (n = 6)	
	Pre	Post	Pre	Post	Pre	Post
Memory Recall	7.8 ± 1.7	8.5 ± 1.9	7.8 ± 1.2	9.5 ± 1.8*	7.2 ± 1.6	8.3 ± 1.4
VF L1	9.6 ± 3.1	12.7 ± 3.1	9.8 ± 2.2	16 ± 4.7*	8.3 ± 3.2	13 ± 2.7*
VF L2	11.6 ± 2.2	12.7 ± 3.8	12 ± 2.5	10.4 ± 3.3	13.5 ± 2.1	10.6 ± 3.01
VF L3	9.1 ± 3.3	11.5 ± 2.5	11.3 ± 1.6	14.2 ± 3.1	11.3 ± 3.2	11.5 ± 3.4
DMR	5.5 ± 1.6	6.8 ± 2.1	7.2 ± 1.7	7.5 ± 2.1	4.8 ± 1.8	6.5 ± 1.4

*Values reported as means ± SD, significant finding = * p = < 0.05, abbreviations: VF L1, verbal fluency letter 1; VF L2, verbal fluency letter 2; VF L3, verbal fluency letter 3; DMR, delayed memory recall.*

There was no main effect for time, $F(1, 16) = 0.091$, $p = 0.767$ and no group x time interaction effect found, $F(2, 16) = 1.566$, $p = 0.239$. Six weeks of HIIE found no significant difference in memory recall for the N group, ($p = 0.477$). A significant difference was found in the O group for the memory recall score, ($p = 0.019$). No difference was found in the C group, ($p = 0.180$). Following six weeks of HIIE, a 13.2 ± 32.3% increase was found in the N group for the memory recall, the O group had a 21.1 ± 14.7% increase and the C group had a 20.2 ± 29.3% increase. No difference in the magnitude of change was found between the N and O group ($p = 0.592$), for between the N and C groups ($p = 0.691$) and for between the O and C groups ($p = 0.948$).

There was a main effect for time, F (1, 16), = 27.855, p = 0.000, however no group x time interaction effect was found, F (2, 16), = 1.091, p = 0.361. Six weeks of HIIE found no difference for verbal fluency letter 1 scores in the N group, (p = 0.071), although a moderate effect size (d = 1) was found. A significant difference was found in the O group, (p = 0.046). A significant difference was found in the C group, (p = 0.003). Six weeks of HIIE found a 43.5 ± 51.2% increase in verbal fluency letter 1 for the N group, the O group found a 44.3 ± 87.9% increase, and the control group found a 71.7 ± 59.5% increase. No difference in the magnitude of change was found between the N and O group (p = 0.984), between the N and C groups (p = 0.376) and between the O and C groups (p = 0.540).

There was no main effect for time, F (1, 16), = 1.112, p = 0.308 and no group x time interaction effect, F (2, 16), = 2.012, p = 0.168. Six weeks of HIIE found no difference in verbal fluency letter 2 scores for the N group, (p = 0.339). No difference was found in the O group, (p = 0.674). No difference was found in the C group, (p= 0.063). Six weeks of HIIE increased verbal fluency letter 2 scores in the N group by 10.1 ± 28% increase, a 19.5 ± 55.2% decrease was found in the O group, and a 20.3 ± 22.3% decrease was found in the C group. No difference in the magnitude of change was found between the N and O groups (p = 0.237), a significant difference in the magnitude of change was found between the N and C groups (p = 0.056) and no difference was found between the O and C groups (p = 0.975).

There was a main effect for time, F (1, 16), = 5.822, p = 0.029, however no group x time interaction effect was found, F (2, 16), = 1.287, p = 0.305. Six weeks of HIIE found no significant difference between verbal fluency letter 3 scores for the N group, (p = 0.116). No significant difference was found in the O group for letter 3, (p = 0.114). No difference was found in the C group, (p = 0.903). Six weeks of HIIE found a 41.3 ± 64.7% increase in verbal fluency letter 3 scores in the N group, a 11.4 ± 63.2% increase in the O group and a 6.7 ± 36.4% increase in the C group. No difference in the magnitude of change was found between the N and O groups (p = 0.418), between the N and C groups (p = 0.272) and between the O and C groups (p = 0.879).

There was a main effect for time found, $F(1, 16) = 4.896$, $p = 0.042$, however no group x time interaction effect was found, $F(2, 16) = 0.615$, $p = 0.553$. Six weeks of HIIE found no difference in delayed memory recall in the N group, ($p = 0.222$). No difference was found in the O group for pre and post measures, ($p = 0.174$). No difference was found in the C group, ($p = 0.185$). Six weeks of HIIE found a 32.7 ± 52.6% increase in the N group, a 3.9 ± 6.1% increase in the O group and 1 ± 7.9% decrease in the C group. No difference in the magnitude of change was found between the N and C groups ($p = 0.212$), between the N and C groups ($p = 0.150$) and between the O and C groups ($p = 0.254$).

Heart Rate

Normal Group

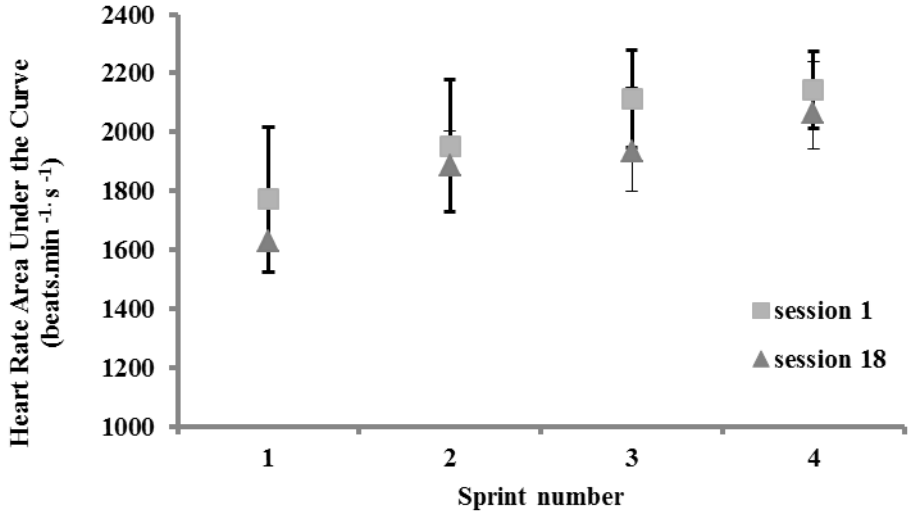


Figure 6: A comparison of heart rate area under the curve for session 1 and session 18 for the normal group (* p<0.05).

Between sprint 1 of session 1 and sprint 1 of session 18, there was no main effect for time found, F (1, 16) = 0.118, p = 0.736 and no group x time interaction effect during the first sprint, F (2, 16) = 1.164, p = 0.337. Between sprint 2 of session 1 and sprint 2 of session 18, no main effect for time was found, F (1, 16) = 0.325, p = 0.577, and no group x time interaction effect was found, F (2, 16) = 0.278, p = 0.761. Between sprint 3 of session 1 and sprint 3 of session 18, there was no main effect for time, F (1, 16) = 0.610, p = 0.446 and no group x time interaction effect, F (2, 16) = 1.946, p = 0.175. Between sprint 4 of session 1 and sprint 4 of session 18, there was no effect for time, F (1, 16), = 0.344, p = 0.566 and no group x time interaction effect was found, F (2, 16) = 1.101, p = 0.356. Six weeks of HIIE did not significantly alter the heart rate area under the curve (HRAUC) for the N group for sprint 1, (Figure 6, N pre; 1771 ± 246 beats.min$^{-1.}$ s $^{-1}$, N post; 1629 ± 134 beats.min$^{-1.}$ s $^{-1}$; p = 0.281), although a moderate effect size was found (d = 0.72). Six weeks of HIIE did not significantly alter the HRAUC for the N group for sprint 2, (Figure 6, N pre; 1953 ± 225 beats.min$^{-1.}$ s $^{-1}$, N post; 1886 ± 120 beats.min$^{-1.}$ s $^{-1}$; p = 0.561), although a small effect size was found (d = 0.37). Six weeks of HIIE did not significantly alter the HRAUC for the N group for sprint 3, (Figure 6, N pre; 2115 ± 166 beats.min$^{-1.}$ s $^{-1}$, N post;

1935 ± 218 beats.min$^{-1.}$ s $^{-1}$; p = 0.244), although a moderate effect size was found (d = 0.93). Six weeks of HIIE did not significantly alter the HRAUC for the N group for sprint 4, (Figure 6, N pre; 2144 ± 129 beats.min$^{-1.}$ s $^{-1}$, N post; 2063 ± 175 beats.min$^{-1.}$ s $^{-1}$; p = 0.503), although a small effect size was found (d = 0.53). Six weeks of HIIE found a 6 ± 17% decrease in HRAUC between sprint 1 session 1 and sprint 1 session 18, a 2 ± 15% decrease between sprint 2 session 1 and sprint 2 sessions 18, a 7 ± 17% decrease between sprint 3 session 1 and sprint 3 session 18, and a 3 ± 14% decrease between sprint 4 session 1 and sprint 4 session 18. For sprint 1, no difference in the magnitude of change was found between groups O and C group (p = 0.240) and groups N and C groups (p = 0.137). For sprint 2, no difference in the magnitude of change was found between O and C (p = 0.402), and for between the N and C groups (p = 0.553). For sprint 3, no difference in the magnitude of change was found between the O and C (p = 0.087) and for between the N and C (p = 0.111). For sprint 4, no difference in the magnitude of change was found between the O and C groups (p = 0.148) and between the N and C groups (p = 0.319).

Heart Rate Recovery

Normal Group

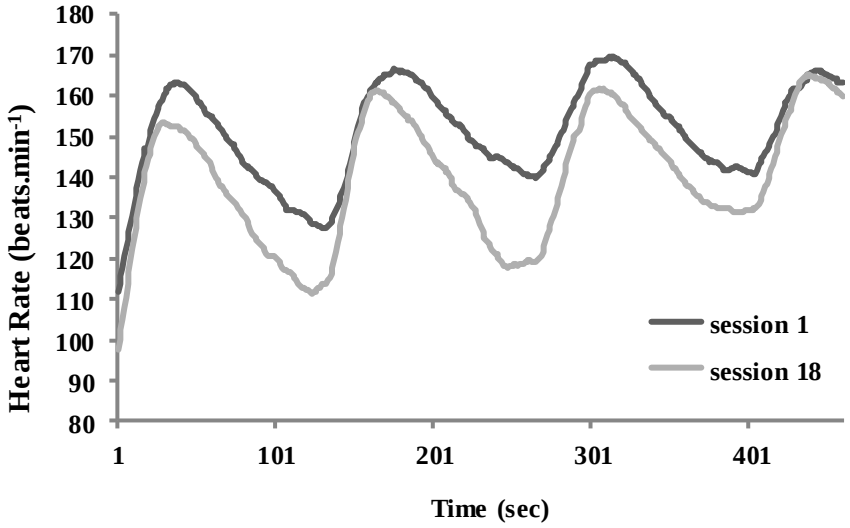


Figure 7: A comparison of heart rate recovery during for session 1 and session 18 for the normal group (* = significant findings).

Between sprint 1 session 1 recovery and sprint 1 session 18 recovery, no main effect for time was found, F (1, 16) = 2.027, p = 0.174 and no group x time interaction effect was found, F (2, 16) = 0.745, p = 0.490. Between sprint 2 session 1 recovery and sprint 2 session 18 recovery, no main effect for time was found, F (1, 16) = 3.683, p = 0.073, a significant interaction effect was found for group x time, F (2, 16) = 3.879, p = 0.042. Between sprint 3 session 1 recovery and sprint 3 session 18 recovery, no main effect for time was found, F (1, 16) = 1.798, p = 0.199 and no group x time interaction effect was found, F (2, 16) = 1.209, p = 0.324. Six weeks of HIIE did not alter recovery between sprint 1 session 1 and sprint 1 session 18 in the N group, (Figure 7, N pre; 17321 ± 1440s, N post; 15994 ± 886s; p = 0.118). Six weeks of HIIE did not alter recovery between sprint 2 session 1 and sprint 2 session 18, (Figure 7, N pre; 18268 ± 1343s, N post; 16738 ± 802s; p = 0.066), although a large effect size was observed (d = 1.4). Six weeks of HIIE did not alter recovery between sprint 3 session 1 and sprint 3 session 18, (Figure 7, N pre; 18421 ± 1302s, N post; 17381 ± 873s; p = 0.187), although a moderate effect size was observed (d = 0.9). Six weeks of HIIE found a 7 ± 11% decrease in recovery between sprint 1

session 1 and sprint 1 session 18, an 8 ± 10% decrease was found between recovery sprint 2, session 1 and sprint 2 sessions 18, a 5 ± 10% decrease was found between sprint 3 recovery, session 1 and sprint 3 session 18. For sprint 1 recovery, no difference in the magnitude of change was found between groups O and N ($p = 0.441$) and groups N and C ($p = 0.287$). For sprint 2 recovery, no difference in the magnitude of change was found between O and N groups ($p = 0.681$), however a significant difference in the magnitude of change was found between groups N and C ($p = 0.027$). For sprint 3 recovery, no difference in the magnitude of change was found between the O and N groups ($p = 0.924$) and between the N and C groups ($p = 0.227$).

Heart Rate

Overweight Group

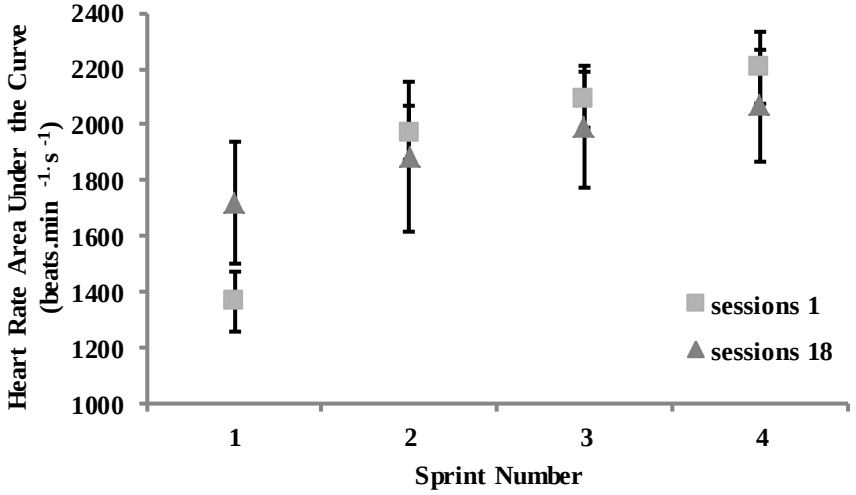


Figure 8: A comparison of heart rate area under the curve for session 1 and session 18 for the overweight group (* p<0.05).

Between sprint 1 of session 1 and sprint 1 of session 18, there was no main effect for time found, F (1, 16) = 0.118, p = 0.736 and no group x time interaction effect during the first sprint, F (2, 16) = 1.164, p = 0.337. Between sprint 2 of session 1 and sprint 2 of session 18, no main effect for time was found, F (1, 16) = 0.325, p = 0.577, and no group x time interaction effect was found, F (2, 16) = 0.278, p = 0.761. Between sprint 3 of session 1 and sprint 3 of session 18, there was no main effect for time, F (1, 16) = 0.610, p = 0.446 and no group x time interaction effect, F (2, 16) = 1.946, p = 0.175. Between sprint 4 of session 1 and sprint 4 of session 18, there was no effect for time, F (1, 16), = 0.344, p = 0.566 and no group x time interaction effect was found, F (2, 16) = 1.101, p = 0.356. Six weeks of HIIE did not significantly alter the HRAUC for the O group for sprint 1, (Figure 8, O pre; 1748 ± 105 beats.min$^{-1.}$ s $^{-1}$, O post; 1718 ± 219 beats.min$^{-1.}$ s $^{-1}$; p = 0.726), although a small effect size was found (d = 0.2). Six weeks of HIIE did not significantly alter the HRAUC for the O group for sprint 2, (Figure 8, O pre; 1970 ± 95 beats.min$^{-1.}$ s $^{-1}$, O post; 1886 ± 268 beats.min$^{-1.}$ s $^{-1}$; p = 0.407), however a small effect size was found (d = 0.42). Six weeks of HIIE did not significantly alter the HRAUC for the O group for sprint 3, (Figure 8, O pre; 2092 ± 100 beats.min$^{-1.}$ s $^{-1}$, O post; 1989 ± 219 beats.min$^{-1.}$ s $^{-1}$; p =

0.1778), although a moderate effect size was found (d = 0.6). Six weeks of HIIE did not significantly alter the HRAUC for the O group for sprint 4, (Figure 8, O pre; 2205 ± 127 beats.min$^{-1.}$ s $^{-1}$, O post; 2069 ± 202 beats.min$^{-1.}$ s $^{-1}$; p = 0.102), however a moderate effect size was found (d = 0.81). Six weeks of HIIE found a 2 ± 12% decreased in HRAUC between sprint 1 session 1 and sprint 1 session 18, a 4 ± 12% decrease between sprint 2 session 1 and sprint 2 sessions 18, a 5 ± 8% decrease between sprint 3 session 1 and sprint 3 session 18, and a 6 ± 8% decrease between sprint 4 session 1 and sprint 4 session 18. For sprint 1, no difference in the magnitude of change was found between groups O and N (p = 0.588) and for the O and C groups (p = 0.240). For sprint 2, no difference in the magnitude of change was found between O and N (p = 0.768) and for between the O and C groups (p = 0.402). For sprint 3, no difference in the magnitude of change was found between the O and N groups (p = 0.753) and between the O and C groups (p = 0.087). For sprint 4, no difference in the magnitude of change was found between the O and N groups (p = 0.637) and between the O and C groups (p = 0.148).

Heart Rate Recovery

Overweight Group

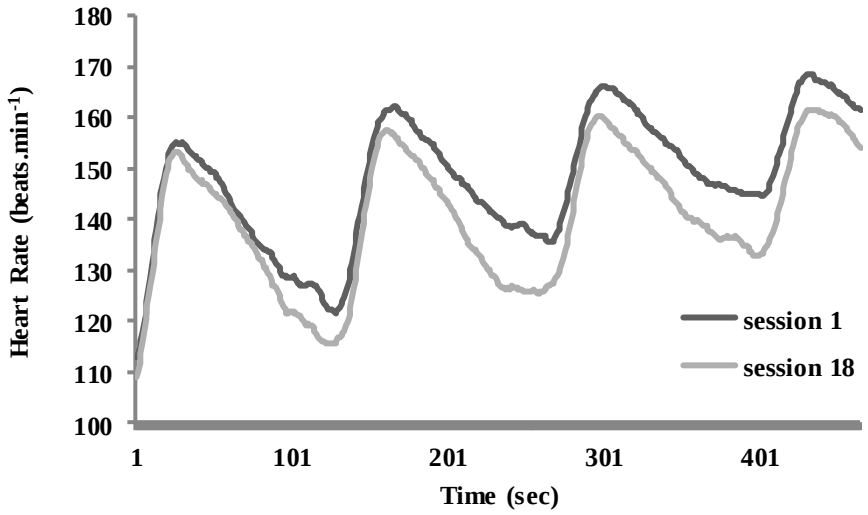


Figure 9: A comparison of heart rate recovery during for session 1 and session 18 for the overweight group (* p<0.05).

Between sprint 1 session 1 recovery and sprint 1 session 18 recovery, no main effect for time was found, F (1, 16) = 2.027, p = 0.174 and no group x time interaction effect was found, F (2, 16) = 0.745, p = 0.490. Between sprint 2 session 1 recovery and sprint 2 session 18 recovery, no main effect for time was found, F (1, 16) = 3.683, p = 0.073, a significant interaction effect was found for group x time, F (2, 16) = 3.879, p = 0.042. Between sprint 3 session 1 recovery and sprint 3 session 18 recovery, no main effect for time was found, F (1, 16) = 1.798, p = 0.199 and no group x time interaction effect was found, F (2, 16) = 1.209, p = 0.324. Six weeks of HIIE did not alter recovery between sprint 1 session 1 and sprint 1 session 18 in the O group, (Figure 9, O pre; 16459 ± 928s, O post; 15937 ± 1218s; p = 0.203), although a small effect size was observed (d = 0.48). Six weeks of HIIE did not alter recovery between sprint 2 session 1 and sprint 2 session 18, (Figure 9, O pre; 17602 ± 991s, O post; 16559 ± 1274s; p = 0.084), although a moderate effect size was observed (d = 0.91). Six weeks of HIIE did not alter recovery between sprint 3 session 1 and sprint 3 session 18, (Figure 9, O pre; 18332 ± 1027s, O post; 17273 ± 1397s; p = 0.155), although a moderate effect size was observed (d = 0.86). Six weeks of HIIE found a 3 ± 5% decrease in recovery between sprint 1 session 1 and sprint 1 session 18, a 6 ± 7%

decrease was found recovery sprint 2 session 1 and sprint 2 sessions 18, a 6 ± 8% decrease was found between recovery of sprint 3 session 1 and sprint 3 session 18. For sprint 1 recovery, no difference in the magnitude of change was found between groups O and N ($p = 0.441$) and groups O and C ($p = 0.537$). For sprint 2 recovery, no difference in the magnitude of change was found between O and N ($p = 0.681$), however a significant difference in the magnitude of change was found between groups O and C ($p = 0.028$). For sprint 3 recovery, no difference in the magnitude of change was found between the groups O and N ($p = 0.924$) and between groups O and C ($p = 0.197$).

Heart Rate

Control Group

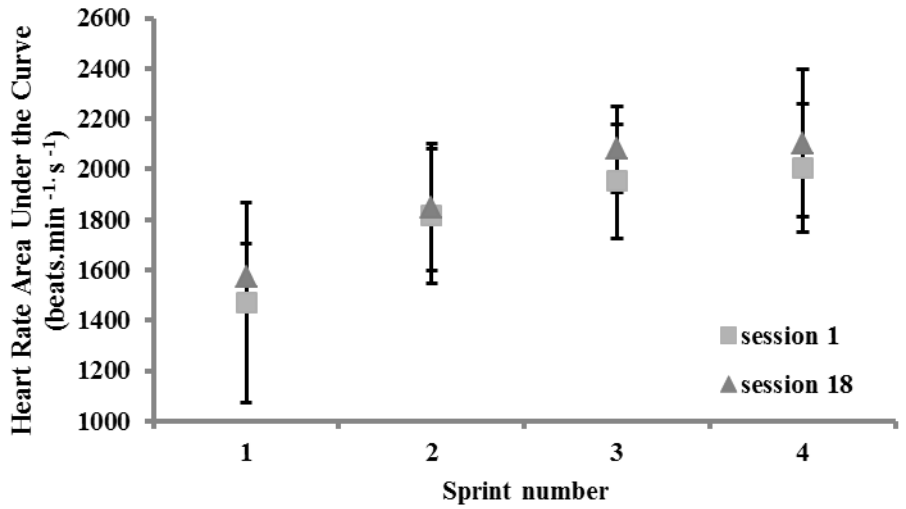


Figure 10: A comparison of heart rate area under the curve for session 1 and session 18 for the control group (* p<0.05).

Between sprint 1 of session 1 and sprint 1 of session 18, there was no main effect for time found, F (1, 16) = 0.118, p = 0.736 and no group x time interaction effect during the first sprint, F (2, 16) = 1.164, p = 0.337. Between sprint 2 of session 1 and sprint 2 of session 18, no main effect for time was found, F (1, 16) = 0.325, p = 0.577, and no group x time interaction effect was found, F (2, 16) = 0.278, p = 0.761. Between sprint 3 of session 1 and sprint 3 of session 18, there was no main effect for time, F (1, 16) = 0.610, p = 0.446 and no group x time interaction effect, F (2, 16) = 1.946, p = 0.175. Between sprint 4 of session 1 and sprint 4 of session 18, there was no effect for time, F (1, 16), = 0.344, p = 0.566 and no group x time interaction effect was found, F (2, 16) = 1.101, p = 0.356. Six weeks of HIIE did not significantly alter the HRAUC for the C group for sprint 1, (Figure 10, C pre; 1470 ± 395 beats.min$^{-1.}$ s $^{-1}$, C post; 1574 ± 130 beats.min$^{-1.}$ s $^{-1}$; p = 0.281). Six weeks of HIIE did not significantly alter the HRAUC for the C group for sprint 2, (Figure 10, C pre; 1815 ± 267 beats.min$^{-1.}$ s $^{-1}$, C post; 1849 ± 251 beats.min$^{-1.}$ s $^{-1}$; p = 0.561). Six weeks of HIIE did not significantly alter the HRAUC for the C group for sprint 3, (Figure 10, C pre; 1952 ± 225 beats.min$^{-1.}$ s $^{-1}$, C post; 2080 ± 170 beats.min$^{-1.}$ s $^{-1}$; p = 0.244). Six weeks of HIIE did not significantly alter the HRAUC for the C group for sprint

4, (Figure 10, C pre; 2005 ± 256 beats.min$^{-1.}$ s $^{-1}$, C post; 2104 ± 291 beats.min$^{-1.}$ s $^{-1}$; p = 0.503). Six weeks of HIIE found a 6 ± 17% decreased in HRAUC between sprint 1 session 1 and sprint 1 session 18, a 2 ± 15% decrease between sprint 2 session 1 and sprint 2 sessions 18, a 7 ± 17% decrease between sprint 3 session 1 and sprint 3 session 18, and a 3 ± 14% decrease between sprint 4 session 1 and sprint 4 session 18. For sprint 1, no difference in the magnitude of change was found between groups O and N group (p = 0.588) and N and C groups (p = 0.137). For sprint 2, no difference in the magnitude of change was found between O and N (p = 0.768), and between the N and C groups (p = 0.553). For sprint 3, no difference in the magnitude of change was found between the O and N (p = 0.753) and between the N and C groups (p = 0.111). For sprint 4, no difference in the magnitude of change was found between the O and N groups (p = 0.637) and between the N and C groups (p = 0.319).

Heart Rate Recovery

Control Group

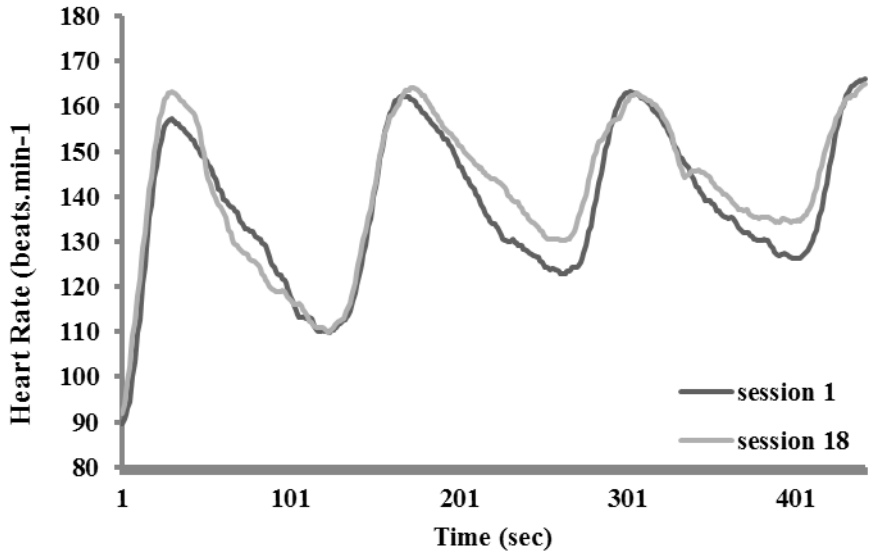
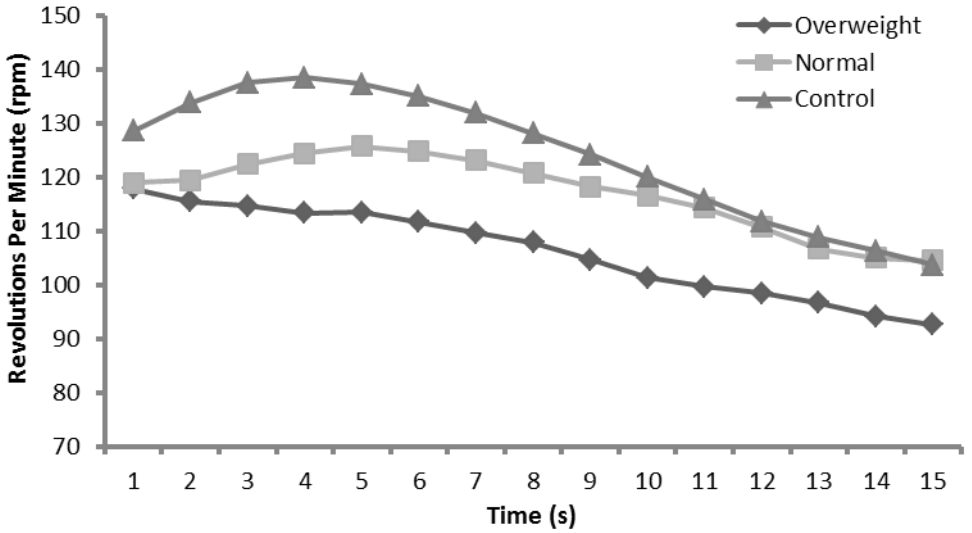


Figure 11: A comparison of heart rate recovery during for session 1 and session 18 for the control group (* p<0.05).

Between sprint 1 session 1 recovery and sprint 1 session 18 recovery, no main effect for time was found, F (1, 16) = 2.027, p = 0.174 and no group x time interaction effect was found, F (2, 16) = 0.745, p = 0.490. Between sprint 2 session 1 recovery and sprint 2 session 18 recovery, no main effect for time was found, F (1, 16) = 3.683, p = 0.073, a significant interaction effect was found for group x time, F (2, 16) = 3.879, p = 0.042. Between sprint 3 session 1 recovery and sprint 3 session 18 recovery, no main effect for time was found, F (1, 16) = 1.798, p = 0.199 and no group x time interaction effect was found, F (2, 16) = 1.209, p = 0.324. Six weeks of HIIE did not alter recovery between sprint 1 session 1 and sprint 1 session 18 in the C group, (Figure 11, C pre; 15745 ± 2252s, C post; 15703 ± 2005s; p = 0.969). Six weeks of HIIE did not alter recovery between sprint 2 session 1 and sprint 2 session 18, (Figure 11, C pre; 16794 ± 1879s, C post; 17441 ± 1911s; p = 0.240). Six weeks of HIIE did not alter recovery between sprint 3 session 1 and sprint 3 session 18, (Figure 11, C pre; 16967 ± 1835s, C post; 17345 ± 2233s; p = 0.682). Six weeks of HIIE found a 1 ± 15% increase in recovery between sprint 1 session 1 and sprint 1 session 18, a 4 ± 7% increase was found recovery sprint 2 session 1 and sprint 2 sessions

18, a 3 ± 12% increase was found between recovery of sprint 3 session 1 and sprint 3 session 18. For sprint 1 recovery, no difference in the magnitude of change was found between groups C and N ($p = 0.287$) and groups O and C ($p = 0.537$). For sprint 2 recovery, a significant difference in the magnitude of change was found between the O and C groups ($p = 0.028$) and between the N and C groups ($p = 0.027$). For sprint 3 recovery, no difference in the magnitude of change was found between the groups C and N ($p = 0.227$) and for between the O and C groups ($p = 0.197$).

A



B

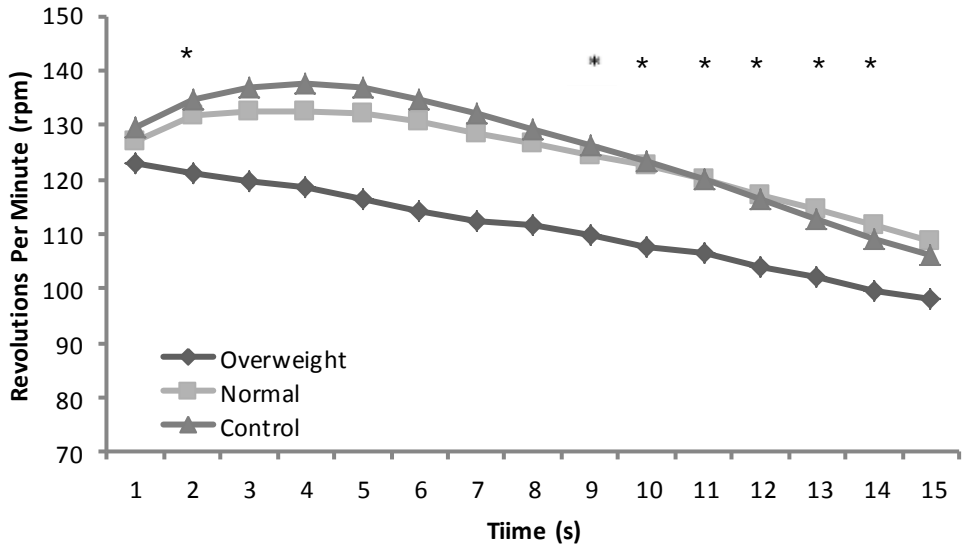


Figure 12: Change in speed over the duration of a sprint. A: session 1, B session 18 (* p<0.05 pre to post in normal weight group).

Across the 2 training sessions, there was no main interaction for time after 1s [F (1, 16) = 5.830, p = 0.028], 2s [F (1, 16) = 5.784, p = 0.029], 9s [F (1, 16) = 6.522, p = 0.021], 10s [F (1, 16) = 9.989, p = 0.006], 11s [F (1, 16) = 12.189, p = 0.003], 12s [, F (1, 16) = 14.181, p = 0.002], 13s [F (1, 16) = 15.682, p = 0.001], 14s [F (1, 16) = 15.298, p = 0.001], 15s [F (1, 16) = 8.654, p = 0.010] but no group x time interaction effect (p>0.05). There was no time main effect or group x time interaction effect at the other time points (p>0.05). Six weeks of HIIE resulted in a significant increase in speed in the N weight group at 2s [N sprint 1: 119.42 ± 13.08 rpm, N sprint 18; 131.57 ± 11.48 rpm, p = 0.040], 9s [], 10s [], 11s [], 12s [], 13s [], 14s [] but not at the other time points. There were no significant differences in speed in the C or O groups at any time point (Fig. 12A&B).

5 Discussion

The aim of the present study was to compare the adaptations to 6 weeks of HIIE in the normal and overweight female population's health markers. The hypothesis proposed was that 6 weeks of HIIE would induce alterations in both female populations, with greater adaptations elicited in the overweight population. The present study demonstrated short-term HIIE improves health markers in females but the response was different in normal weight and overweight population. Following HIIE there was a 25% increase in $\dot{V}O_{2\ peak}$ in the normal weight group but no change in the overweight group, an 18% increase in time to exhaustion in the normal weight group and a 12% increase in the overweight group. There was a similar reduction in systolic blood pressure in both groups 3.6% reduction in normal weight group and a 4.5% reduction in the overweight group. Furthermore the overweight group has a 21% enhancement in memory recall and a 44% increase in VF L1. These results suggest that the adaptation to HIIE is partially regulated by body composition in females.

5.1 $\dot{V}O_{2\ peak}$

A significant improvement in $\dot{V}O_{2\ peak}$ was found in N group but not the O group following 6 weeks of training with no alteration in the control group (Fig. 1). The N group significantly increased $\dot{V}O_{2\ peak}$ by 25% whereas there was only a 7% increase in $\dot{V}O_{2\ peak}$ in the O group. The magnitude of change in the N group is similar to that reported by Gillen et al. (2016) of a 19% increase in $\dot{V}O_{2\ peak}$ in a young female population with a BMI of 27 $kg.m^{-2}$ and a starting $\dot{V}O_{2\ peak}$ of 32 ml/kg/min after 12 weeks of HIIE. In contrast in trained females with a BMI of 22 $kg.m^{-2}$ and a $\dot{V}O_{2\ peak}$ of 41 ml/kg/min, $\dot{V}O_{2\ peak}$ has only been reported to rise by 7% after HIIE (Astorino et al 2011). In the current study there was no significant difference in the initial $\dot{V}O_{2\ peak}$ between the N and O group, therefore initial fitness does not explain the difference in response. Burke and Franks (1975) showed that training intensity at the same total workload alters the magnitude of $\dot{V}O_{2\ max}$ changes following training. Therefore it seems plausible to

suggests that body mass may have altered the training intensity in the O group compared to the N group despite working against the same relative resistance. Indeed this can be seen when looking at the speed of the sprints produced by both groups (Fig. 12) and the HRAUC (Figs. 6&8). The absolute cardiovascular demand is similar across all 4 sprints for session 1 and session 18 which suggests a similar overall workload (Achten and Jeukendrup 2003). However the speed profiles of the sprints are different with the N group accelerating past the resistance drop point before declining later in the sprint whereas the O group had no initial acceleration. A potential reason for this discrepancy could be that the use of body mass to control resistance results in an excessive weight being loaded which does not take into account the contribution of fat mass into total body mass. Indeed if we compare the increase in $\dot{V}O_{2\ peak}$ seen in the N and O groups (BMI 21 $kg.m^{-2}$ and 30 $kg.m^{-2}$ respectively) to the females in Gillen's et al. (2016) study (27 $kg.m^{-2}$) we see a greater increase in the N group compared to both overweight groups (25% after 6 weeks in N, 7% after 6 weeks in O compared to 12 & 19% after 6 & 12 weeks respectively) (Gillen et al. 2016). When we look at the correlation between change in $\dot{V}O_{2\ peak}$ and BMI at the start of the study we find it to be negatively correlated (Spearman's rho: -0.77, p = 0.007). Therefore it is important that we develop a method to overcome this issue of excessive body mass.

The 25% increase in $\dot{V}O_{2\ peak}$ in the N group is comparable to Trapp et al. (2008) who found a 23.8% increase in$\dot{V}O_{2\ peak}$ among young women following 15 weeks of HIIE and attributed the improvements to the aerobic component on the HIIE. This suggests the aerobic component; 2 minute recovery between sprints during HIIE in the present study may have contributed to the $\dot{V}O_{2\ peak}$ improvements. The present study used 1:8 work to rest ratio however Trapp et al. (2008) used 1:1.5 with a longer 15 week intervention, suggesting a higher work to rest ratio used in the present study is beneficial for increasing $\dot{V}O_{2\ peak}$ over 6 weeks for the Normal population. This is supported by Kavaliauskas, Aspe and Babraj (2015) who show work: rest ratio is important for $\dot{V}O_{2\ peak}$ adaptations.

A potential reason for the increase in $\dot{V}O_{2\ peak}$ following HIIE is adaption to mitochondrial enzyme content and activity. HIIE leads to increases in

mitochondrial protein content, regulated by increases in PGC-1 alpha transcription, a key regulator of mitochondrial biogenesis (Pilegaard et al. 2003) and the up-regulation of AMPK (Di Donato et al. 2014), which is comparable to endurance exercise bouts (Wright et al. 2007). In response to 3 weeks of HIIE, mitochondrial protein synthesis increased by ~150% (Scalzo et al. 2014). Six weeks of HIIE increases SIRT 1 protein in men and women (Gurd et al. 2009) and mitochondrial biogenesis is stimulated by SIRT 1 and PGC-1α interaction, enabling increases in peak oxygen consumption and fatigue blunting (Davies et al. 2008), which may be the underlying mechanisms for improvements to $\dot{V}O_{2\ peak}$ in the present study. However because SIRT 1, PGC-1α and AMPK were not directly assessed, this can only be speculated. With no differences in adaptations when comparing 15 s and 30 s HIIE sprints (Zelt et al. 2014) and the aerobic energy system demand required for 5 x 30 s HIIE sprints has been suggested to trigger mitochondrial adaptations (Trump et al. 1996), the aerobic energy system demand imposed by the 15 s sprints in the present study may have contributed towards the $\dot{V}O_{2\ peak}$ increases. This is supported by Bogdanis et al. (1996) who found a 29% aerobic contribution during a 30 s Wingate, however following a 4 minute passive recovery, the aerobic contribution was 44% in the second Wingate (Bogdanis et al. 1996). HIIE elicits increases to skeletal muscle oxidative capacity through increased activities of β-HAD and CS, which reflect mitochondrial volume and fatty acid β-oxidation (Relsch and Elpeleg 2007; Holloszy and Coyle 1984). CS increases respiratory control sensitivity, a mechanism for improvements in endurance capacity (Burgomaster et al. 2005; Holloszy and Coyle 1984). Furthermore, HIIE elicits increases to PDH which helps regulate oxidative carbohydrate metabolism (LeBlanc et al. 2004). Although β-HAD, CS and PDH were not directly assessed, it seems reasonable to suggests similar adaptations occurring in the N group that lead to improvements in $\dot{V}O_{2\ peak}$ within the present study.

5.2 Time to Exhaustion

Significant improvements in time to exhaustion (TTE) were observed in the N and O groups following 6 weeks of training with no alteration in the C group

(Fig. 2). The N group increased by 18% and the O group increased by 12%. The improvement in TTE is in agreement with others however the difference in change varies. Burgomaster et al. (2005) found recreationally active individuals increased TTE by 100% when cycling at 80% $\dot{V}O_{2peak}$ following six sprint interval sessions. Furthermore, Burgomaster et al. (2006) observed a 9.6% increase in response to 2 weeks of SIT. MacDougall et al. (1998) and Burgomaster et al. (2005) found improvements in TTE were due to increased CS maximal activity and mitochondrial potential, which may explain improvements in TTE in the present study. Although, CS and mitochondrial potential adaptations were not directly assessed, is seems unreasonable to suggest similar adaptations occurring in the present study. Improvements in TTE may be explained through improvements to skeletal muscle oxidative capacity through increased activities of β-HAD (Talanian et al. 2007), CS and PDH (Burgomaster et al. 2008). β-HAD and CS reflects mitochondrial volume and capacity for fatty acid β-oxidation (Relsch and Elpeleg 2007; Holloszy and Coyle 1984), CS enhances respiratory control; an underlying mechanisms for endurance capacity improvements (Burgomaster et al. 2005; Holloszy and Coyle 1984) and PDH regulates the oxidative carbohydrate metabolism (LeBlanc et al. 2004). Furthermore, increases in mitochondrial capacity allow aerobic capacity increases due increased muscle mitochondria concentrations (Hoppeler et al. 1985). Although, these enzyme activities were not directly measured, similar adaptations in the present study may have contributed towards the increase in TTE for both groups. HIIE training has been shown to increase mitochondrial protein content (Little et al. 2011), regulated by PGC-1α increases; a key regulator of mitochondrial biogenesis (Di Donato et al. 2014). HIIE also increases SIRT 1 protein (Gurd et al. 2009), SIRT 1 and PGC-1α stimulate mitochondrial biogenesis helping blunt fatigue (Davies et al. 2008), which may explain the improvements in TTE for both groups, however because PGC-1α and SIRT 1 were not measured, this can only be speculated.

5.3 Blood Pressure

A significant reduction in left arm systolic blood pressure was found in the N and O groups following 6 weeks of training, with no alteration in the C group (Table 1). The N group found a 3.6% decrease (4.3mm Hg) and the O group observed a 4.5% (5.7mm Hg) decrease. No significant alterations were made for right arm systolic, right arm diastolic and left arm diastolic blood pressure, however a moderate effect size was found for right arm systolic pressure in both the N (d = 0.87) and the O groups (d = 0.78). No significant alteration for right arm systolic, right arm diastolic and left arm diastolic blood pressure is comparable to Rakobowchuk et al. (2008) who found no alterations in blood pressure following 6 weeks of HIIE although popliteal artery distensibility increased. This suggests although no alteration for right arm systolic, right arm diastolic and left arm diastolic blood pressure, popliteal artery distensibility may have increased however because popliteal artery distensibility was not assessed, this can only be speculated. Reductions in systolic blood pressure have been suggested to be due to changes to the sympathetic nervous activity (Halliwill et al. 1996), enhanced nitric oxide-mediated vasodilation (Halliwill 2001) and improved endothelial function (Tjonna et al. 2008), which may partly explain why reductions in left arm systolic blood pressure and the moderate effect size in right arm systolic pressure were found, however because these were not measured, these reasons can only be suggested. Tjonna et al. (2008) found a reduction of 6.3% following 16 weeks of training in conjunction with improved endothelial function; therefore it seems unreasonable to suggest the same but not to the same magnitude of adaptations occurred in the present study. However, because the length of study was not the same as Tjonna et al. (2008) study, this can only be suggested.

5.4 Perception of Happiness

No significant increase in perception of happiness scores were observed in response to 6 weeks of training, with no alteration in the C group (Fig. 3). Although no significant increase was observed, a small effect size was found in the O group (d = 0.53) who had a 13% increase in happiness scores and had a happiness rating of 4.8 from 4.4 (rather happy; pretty happy) (Hills and Argyle,

2002). Results from the present study are comparable to Martin et al. (2000) who found profile of mood states (POMS) did not significantly increase following 6 weeks of HIIE, although a large effect size was found due to the vigorous and fatigue feelings associated with HIIE. The same author also found increased levels of cortisol 36 hours post training, so it seems unreasonable to suggests small effect size in the present study may be due to elevated cortisol levels, however because cortisol levels were not directly assessed, this can only be suggested. The small effect size in perception of happiness scores in the present study may also be due to the feelings associated with HIIE; vigorous and fatigue however this can only be assumed. Tjonna et al. (2008), Wisloff et al. (2007) and Raedeke (2007) found HIIE elicits feelings of enjoyment compared to continuous exercise, which may be an underlying mechanism for the small effect size in perception of happiness scores. Mertesdorf (1994) suggests HIIE can be distressing for individuals, suggesting the present study may have been too distressing for individuals to increase perception of happiness scores, however because feelings associated with HIIE were not measured, this can only be suggested.

5.5 Triglycerides

No significant reductions in TG were found in both groups following 6 weeks of training, with no alteration to the C group (Fig. 4). Although no significant reduction was found for TG, a moderate effect size (d = 0.79) was found in the O group who had a 22% decrease in TG. Reductions in lipoproteins TG have been found following 8 weeks of HIIE running at 60% and 90% peak oxygen consumption (Tsekouras et al. 2008), however the same reduction was found in the moderately-intense endurance exercise group. This is supported by Leon et al. (1979) and Rauramaa et al. (1984) who found low and moderately intense exercise reduced the lipid profile among normocholesterolemic individuals. For reductions in TG there is a reliance on total weekly energy expenditure (Crouse et al. 1997; Annuzi et al. 1987; Tsekouras et al. 2007), suggesting the 6 week HIIE intervention energy expenditure threshold was too low in the present study to elicit improvements in TG. This is supported by Crouse et al. (1997) who

found partaking in HIIE has no added benefit to lipid alterations (Crouse et al. 1997). Tsekouras et al. (2008) suggests HIIE is more effective for reducing circulating TG due to the metabolic adaptations associated with HIIE, suggesting the moderate effect size observed in the present study may be due to the metabolic adaptations occurring, however because metabolic adaptations were not directly assessed, this can only be speculated. Nybo et al. (2010) reported volume to be more important than intensity for reducing circulating TGs, suggesting a higher volume in the present study may have reduced further TG further, however this can only be speculated due to volume remaining the same.

The N group had a 63.7% increase in TG and the C group had a 38.9% increase. Following meal intake, it can take up to 12 hours for TG to return normal level (Campos, Khoo and Sacks 2005). In the current study we only asked people to refrain from eating for 10 hours prior to coming to the lab, which could mean they had an elevated TG levels due to diet. This suggests increases in TG in the present study may be due to individual's TG not being at their normal levels due to the ingestion of last meal being shorter than 12 hours before testing.

5.6 Subjective Happiness

No significant improvements were made in subjective happiness following 6 weeks of training; with no alteration in the C group (Fig. 5). The O group however observed a moderate effect size (d = 0.67) of improvement and had a 12.75% increase in subjective happiness. Six weeks of HIIE has been found not to increase profile of mood states (POMS) although a large effect size was found due to the vigorous and feelings of fatigue associated with HIIE. The study also found increases in cortisol 36 hours post training, which may be a mechanism for the moderate effect size in the present study however cortisol levels were not measured so can only be speculated. Furthermore, the moderate effect size in the present study maybe due to the vigorous and feelings of fatigue associated with HIIE, however because this was not directly measured so can only be speculated. Tjonna et al. (2008), Wisloff et al. (2007) and Raedeke (2007) found HIIE elicits feelings of enjoyment compared to continuous exercise, which may be an

underlying mechanism for the moderate effect size in subjective happiness score, however because feelings of enjoyment towards HIIE were not measured, this reason can only be suggested. HIIE has been deemed distressing for individuals, suggesting no significant improvement in subjective happiness in the present study may be due to HIIE being too distressing.

5.7 Body Composition

A significant improvement in WTHR was found in the N group, with no alteration in the O and C group (Table 2). The N group observed a 1.2% decrease in WTHR. Furthermore, a significant increase of 4.3% in left leg fat (%) in the O group and a 1.7% increase in the C group was observed (Table 2). Although no other significant improvements in body composition were made, a large effect size (d = 1.2) was observed for trunk fat (kg) in the O group, with a decrease of 12.4% (7.4kg). The large effect size observed in the present study is comparable to MacPherson et al. (2010) who found a 12.4% (1.7kg) reduction in fat mass following 6 weeks, three times a week of SIT. Factors determining fat loss are increased levels of catecholamines and increasing levels of lipid release (Harmer et al. 2000), suggesting the underlying reasons for the significant finding in WTHR and large effect size for trunk fat loss may be due to increased levels of catecholamines and increasing levels of lipid release during exercise, however because these were not directly assessed, this can only be speculated. Furthermore, increases in resting fat oxidation is a key predictor of induced fat loss (Barewell et al. 2009), however because resting fat oxidation was not assessed in the present study, this can only be suggested. Tjonna et al. (2008) found 16 weeks of aerobic interval training reduced waist circumference parallel with circulating adiponectin levels during exercise and 12 months of aerobic interval training reduced waist circumferences (Tjonna et al. 2009). Based on research and the aforementioned studies by Tjonna, waist circumferences are a long term adaptation and 6 weeks may be deemed too short to elicit improvements in waist to hip ratio among the overweight population. HIIE studies between 8 to 16 weeks have been found to reduce body fat among men and women (Heydari et al. 2012; Boudou et al. 2003; Mourier et al. 1997; Trapp

et al. 2008; Dunn 2009; MacPhersen et al. 2010); this suggests the 6 week intervention used in the present study may be too short to elicit improvements in body composition in the overweight population. This is supported by Trapp et al. (2008) who found 15 weeks of HIIE to be an effective training method and length to elicited improvements in fat mass and body mass.

The significant increase in left leg fat (%) in the O and C group may be due to a number of determining factors; hydration status of the individuals affecting the body composition machine (Wells and Fewtrell 2006), which may have affected the results. Furthermore, considering every dietary factor is independently related to alterations in weight (Mozaffarian et al. 2011) and because diet was not controlled, diet may have played a part to the increases in left leg fat.

5.8 Cognitive Function

A significant improvement in the O group for memory recall was observed following 6 weeks of training, with no alteration in the C and N groups (Table 3). The O group significantly improved by 21%. A significant improvement in L1 was found in the O group with an alteration to the C group. The O group significantly increased L1 scores by 44% and the C group significantly increased L1 scores by 72%. Although no other cognitive function improvements were significant, the N group had a moderate effect size (d = 1) for verbal fluency L1 with an increase of 44% (Table 3). Winter et al. (2007) found 2 weeks of HIIE improved learning due to elevated catecholamine levels and brain-derived neurotrophic factor. Therefore seems unreasonable to suggest these reasons may have attributed to the improvements in delayed memory recall and L1 improvements. However because cerebral blood flow and catecholamines concentrations were not directly measured, this can only be speculated. Improvements to cognitive function are due to increased cerebral blood flow and increased catecholamine concentrations (Kashikara et al. 2009; Winter et al. 2007; McMorries 2009), which may be the underlying mechanism for improvements delayed memory recall and L1 scores in the present study. HIIE training has been shown to increase BDNF and cortisol concentrations and

BDNF is attributed to short term learning due to its sharp decline following exercise and cortisol is attributed to long term learning (Vega et al. 2006). This suggests BDNF levels may partly be the reason for increases in cognitive function, however because BDNF was not directly assessed, this can only be suggested. Furthermore, no improvements in delayed memory recall and letter scores may be due to the lack of cortisol concentrations. Cortisol aids long term learning (Vega et al. 2006), suggesting the HIIE intervention used was sufficient enough to increase cortisol concentrations to help long term learning; however this can only be suggested as cortisol levels were not directly measured.

5.9 Heart Rate Recovery

No significant improvement was found for sprint 1 session 1 and sprint 1 session 18 recovery in the N group (Fig. 7) and the O group (Fig. 9); with no alteration in the C group (Fig. 11). A trend of improvement was observed when comparing recovery of sprint 1 session 1 and sprint 1 session 18, with a moderate effect size (1.1) and a 7% decrease in the N group and a small effect size (d = 0.48) and a 3% decrease in the O group. A trend of improvements was elicited when comparing sprint 2 session 1 recovery and sprint 2 session 18 recovery, with the N group eliciting a large effect size (d = 1.4) along with an 8% decrease, and the O group eliciting a moderate effect size (d = 0.91) and a 6% decrease in recovery. A trend of improvement was also found when comparing sprint 3 session 1 and sprint 3 session 18 recovery, the N group observed a moderate effect size (d = 0.9) and a 5% decrease, and the O group observed an moderate effect (d = 0.86) with a 6% decrease. No significant improvement was found for sprint 1 HRAUC in the N group (Fig. 6), the O group (Fig. 8) and C group (Fig. 10), however a moderate effect size was found in the N group (d = 0.72) with a 6% decrease and a small effect size in the O group (d = 0.2) with a 2% decrease. No significant improvement was found for sprint 2 HRAUC in the N group (Fig. 6), the O group (Fig. 8) and C group (Fig. 10). A small effect size was found in the N group (d = 0.37) with a 2% decrease and the O group (d = 0.42) with a 4% decrease. No significant improvement was found for sprint 3 HRAUC in the N group (Fig. 6), the O group (Fig. 8) in the C group (Fig. 10). However a

moderate effect size was found in the N group (d = 0.93) with a 7% decrease and the O group (d = 0.6) with a 5% decrease. No significant improvement was found for sprint 4 HRAUC in the N group (Fig. 6), the O group (Fig. 8) and in the C group (Fig. 10), however a small effect size was found in the N group (d = 0.53) with a 4% decrease and a moderate effect size was found in the O group (d = 0.81) with a 6% decrease. Following maximal effort exercise, heart rate recovery is caused by a parasympathetic reactivation with the initial phase of recovery being influenced by the sympathetic activation (Lambert et al. 2009), therefore seems unreasonable to suggest these small adaptations occurred in the present study. However because parasympathetic and sympathetic activity were not directly assessed, this can only be speculated. Heart rate recovery is slower in response to maximal exercise due to the sympathetic nervous system being stimulated and following HIIE, decreases in heart rate are due to sympathetic stimulation withdrawal (Kannankeril et al. 2004). Among trained athletes, Brown and Brown (2007) assessed heart rate variability during recovery and following HIIE. Decreases in heart rate variability were found and attributed to increases parasympathetic withdrawal (Brown and Brown 2007).

The larger trend of improvement in the N group in heart rate recovery may be due the discrepancies in body mass and excessive weight on the cradle in the O group, however further research is required to fully understand the small differences.

6 Limitations

The limitations of the present study consist of:

Testing methods:

The initial limitation of the study is the lack testing methods to determine whether adaptations occurring in the body were due to suggested reasons. The first testing method includes; microneurography method, assessing the sympathetic, parasympathetic nervous system activity and vagal tone (Seravalle et al. 2013), muscle biopsies to assess mitochondrial enzyme activity within

skeletal muscle (Burgomaster et al. 2005), an echocardiogram to assess the alterations within the heart and determine the cardiovascular adaptations (Christie et al. 1987).

Furthermore, the study ran 2x3 repeated measures ANOVA to identify a time and/or group x time interaction, then an independent t-test was run to identify any significant findings between pre- and post- testing. A Cohen's D effect size was run if significance wasn't found to identify a trend of improvement. Due to no significance found following the repeated measures ANOVA and independent t-test, this may be due to a small sample size used in the present study (Hojat and Xu 2004).

Bioelectrical impedance was used as a measure of body composition however has several limitations, these include; under or over-hydration levels and the physical effects associated due to tissue conductivity, body geometry abnormalities for example individuals with abnormal body builds such as amputation and due to the trunk having a large cross-sectional area and being relatively short, the trunk contributes to a small proportion of the impedance (Kyle et al. 2004). Furthermore, the bioelectrical impedance also has validating complications due to various ethnic groups having varying body fluids (Hannan et al. 1995), frame size (Borghi et al. 1996) and leg lengths (McCullough et al. 1991) and those with clinical conditions with abnormal fluid distribution and individuals at different ages (Kyle et al. 2004). This suggests potential reasons for the unlikely 7.4kg decrease in trunk fat in the overweight group in the present study may be due to a combination of fluctuated hydration status at pre- and post- measures and the limitations associated with trunk measurements during the impedance, large cross-sectional area and being relatively short.

Revolutions per minute (RPM) is a measure of pedal rotation frequency around a fixed axis and can be used to calculate the work done (Medbo and Tabata 1989). As a measure of exercise in the present study, particularly in the 'overweight' group, RPM shows a steady decline with no acceleration period, expressing a low and misleading sprint intensity and work done. Power is a measure of the force exerted and applied to the flywheel in Watts/Kg and power output is

dependent on the exercise intensity (Bogdanis et al. 1995). Using peak and average power ouput as measures of exercise could indicate sprint intensity and express the fatigue induced by the sprint. The total work of a high-intensity sprint has previsouly been measured by multiplying the frequency of revolutions of the flywheel, by work done per revolution with power calculated between the work and duration ratio (Medbo and Tabata 1989). This expresses the importance of using a variety of exercise measures such as power, RPM and work done to evaluate the sprinting session.

Intervention:

Diet and hydration may have influenced participant's results and performance throughout the intervention. Post-testing results such as body composition (Layman et al. 2003), blood pressure (Svetkey et al. 1999), TGs (Layman et al. 2003), and mood (Benton and Donohoe 1999) may have all been affected along with hydration having an influential role during the intervention and during post-testing (Judelson et al. 2007).

The menstrual phase the females in the present study was not controlled through the 6 week protocol. Food ingestion increases during the menstrual cycle (Bonen et al. 1983) which may have played a role in performance and post-testing results. Aerobic capacity may have been influenced depending on what phase the participants were in (Lebrun et al. 1995) and energy balance may have been affected (Bisdee et al. 1989). Females elicit higher core temperature during the luteal phase which can alter exercise efficiency and impose a higher demand for oxygen consumption (Lebrun et al. 1995), which may have affected the performance results of the study.

6.1 Future Research

Grounded on the results found in the present study, the following recommendations are for future research:

Based on the discrepancies in $\dot{V}O_{2\ peak}$ and sharp decline of the rpm data in the O group in the present study, it is suggested the same protocol should be used among overweight females, however the percentage of body weight used should be 5, 6, and 7% to assess the best weight percentage for cardiovascular and respiratory adaptations.

Considering the ACSM recommend RT in order to retain flexibility, muscular and cardiovascular fitness (Kraemer et al. 2002), as well as helping to treat control and prevent hypertension (Pollock et al. 2000) and type 2 diabetes (Albright et al. 2000) and HIIE is a potent training method for improving skeletal muscle oxidative capacity (Gibala et al. 2012), glucose tolerance (Babraj et al. 2009), insulin sensitivity and fat oxidation (Whyte et al. 2010); future research should examine the effect of a HIIE and RT combination training program for improving health-related markers in all populations.

Considering diet plays an important role in controlling body composition (Layman et al. 2003) and a vegan and vegetarian diet helps protect against obesity (Tonstad et al. 2009); future research should examine the effect HIIE with a controlled diet has on body composition, specifically three different diets consisting of meat, vegan and vegetarian.

6.2 Conclusion

In this context, 6 weeks, three times weekly of progressively increased HIIE of 4x15 – 6x15 s at 7% body weight improved health markers such as left arm systolic blood pressure in both groups, increase time to exhaustion in groups, enhanced memory recall and VF L1 in the O group and increased in$\dot{V}O_{2\ peak}$ in the N group. Current research suggestion of alterations to skeletal muscle mitochondrial content and activity, increased sympathetic and parasympathetic nervous activity and vagal tone, enhanced cerebral blood flow, BNDF levels and catecholamine concentrations all give speculative evidence for the improvements in health markers. However, an important finding of the present study is the negative correlation found between $\dot{V}O_{2\ peak}$ and BMI in the O group. Based on

sharp decline of rpm in the O group and discrepancies in $\dot{V}O_{2\ peak}$ found among the N and O groups, the weight on the cradle in the O group may have been too high to elicit significant improvements to $\dot{V}O_{2\ peak}$, therefore a strategy to overcome the excessive body mass should be developed. The present study suggests HIIE is a time-efficient training method for improving health markers in both female populations compared to traditional exercise training methods, however to provoke further adaptations among the overweight and/or obese, a new strategy or percentage of body weight needs to be explored.

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8 Appendices

Appendix 1

Are you a female and looking to improve your health and fitness?

A 6 week protocol of high-intensity short bursts of exercise to improve health markers.

Do you want to take part in the study and see if you can improve your health?

For further details Contact Thomas Steer

Email:

Appendix 2

Abertay University

'The effect body composition has on adaptation to high-intensity interval exercise'

1. Invitation

You are being invited to take part in this research study. The criteria for selection are that you are aged 18-45, normal population; have a 'normal' BMI and overweight population; BMI of 'overweight', do not have experience of cycle sprinting, and are free from illness or injury that could affect your performance in the study. You should also be able to freely consent to participation. Before you decide whether or not to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information sheet carefully, and discuss it with others if you wish. Ask us if there is anything that is not clear, or if you would like more information. Take time to decide whether or not you wish to take part.

Ask us if there is anything that is not clear, or if you would like more information regarding the experiment. Take time to decide whether or not you wish to take part.

2. What is the purpose of the study?

We wish to determine whether 6 weeks of short bouts of exercise three times a week elicits differences in health implications among the normal and overweight population.

3. Do I have to take part?

No. It is up to you to decide whether or not to take part. If you have decided to take part you have been given this information sheet to keep and will be asked to

sign a consent form to confirm that you understand what is involved when taking part in this study. If you decide to take part you are free to leave the study at any time and without giving a reason.

4. What will happen to me if I take part?

If you agree to take part in this study then you will be asked to sign a consent form stating that you understand why the study is being done and agree to be part of it. You will be booked into the human investigation laboratory at the University of Abertay, at a time that is suitable for you, to allow us to familiarise you with the equipment. After the familiarisation session, you will have the following baseline tests measured; Immediate word recall, verbal fluency, body composition and segmental analysis, perception of happiness, subjective happiness, waist to hip ratio, right and left arm blood pressure, triglycerides, delayed word recall, $\dot{V}O_{2\ peak,}$ heart rate recovery between sprints. You will have the baseline tests measured before and after the 6 week training program. Training will then begin on a day and at a time that is suitable for you. During week 1 and 2, each session will consist of 4 single 15 second cycle sprints three times a week; during week 3 and 4, each session will consist of 5 single 15 second cycle sprints three times a week; during week 5 and 6, each session will consist of 6 single 15 second cycle sprints three times a week. All sprints will be completed against a resistance of 7% body weight and all sprints will be alternated with a 2 minute active recovery. Each session will last approximately 15 minutes separated by 1-2 days. After completing the training you will be asked to complete the baseline tests again.

Session 1: After the familiarisation session, you will be asked to complete an informed consent form and a health questionnaire in the form of a PAR-Q. After completion of these forms you will be invited back to the laboratory having fasted from 10pm the night before to have the following baseline measurements tested; immediate word recall, verbal fluency,body composition and segmental analysis, perception of happiness, subjective happiness, waist to hip ratio, triglycerides, delayed word recall, right and left arm blood pressure, $\dot{V}O_{2peak,}$

heart rate recovery between sprints. This means that you are not allowed to eat or drink anything for at least 10h before the baseline testing.

1. Immediate Word Recall: Participants will be given a unique set of fifteen words and have 60 seconds(s) to recall as many words as they could.
2. Verbal Fluency: Participants will be given three unique letter and have 60 s to write down as many words beginning with those letters.
3. Body composition & Segmental Analysis: Participants will stand bare foot on Tanita scales and the machine will request the following information; height, weight, age, gender, 'athletic' or standard'. A print out will indicate body fat percentage.
4. Perception of Happiness: Participants are invited to fill out and return the Oxford Happiness Questionnaire.
5. Subjective Happiness Scale: Participants are invited to fill out and return the subjective happiness questionnaire provided.
6. Waist to hip ratio: Participants will have their waist and hip measured. The ratio will be determined by dividing the waist measurement by the hip measurement.
7. Right and left arm blood pressure: 2 blood pressure cuffs will be attached to your arms and will inflate and deflate to record blood pressure. This will be taken 3 times.
8. Triglycerides: a single drop of blood will be taken from your index finger and will be used to measure circulating triglycerides.
9. Delayed Word Recall: Participants will be given 60 s to write down as many of the unique fifteen words as they can recall.
10. $\dot{V}O_{2peak:}$ This involves a gradual resistance increase of 35 kilowatts (KW) every 2 minutes, whilst the participants maintain a speed of 70 rpm on the Monark cycle Ergomedic bike.
11. Heart Rate Recovery between sprints: Participants heart rate recovery during the first and last sprint will be monitored to assess potential differences.

After both groups have completed these baseline tests, the training program will begin. After you have completed baseline testing you will begin a 6 week training program carrying it out three times a week.

Training protocol: You will be asked to cycle for 5 minutes at 60 rpm to warm up. You will then be asked to increase the speed so that you are cycling at 110 rpm when a weight will be added to the bike. You will be verbally encouraged to sprint for 15 s and then rest for 2 minutes between sprints. This will then be repeated until you have completed 4 15 second cycle sprints in weeks 1 and 2, 5 cycle sprints in weeks 3 and 4 and 6 cycle sprints in week 5 and 6.

After completing the training you will be asked to repeat the baseline testing shown in session 1.

5. What are other possible disadvantages and risks of taking part?

All procedures have been risk assessed to minimize the risk of injury during the testing. Your data will also be anonymous and will be kept secured at all times. You will not be identified in any report or publication.

6. What happens when the research study stops?

If you wish you will be kept informed of the progress of study and informed of the overall results. Results may be published in a scientific journal or presented at a scientific conference. The data will be anonymous and you will not be identified in any report or publication.

7. What if there is a problem?

If you have a concern about any aspect of this study, you should ask to speak with the researchers who will do their best to answer your question.

This completes Part 1 of the Information Sheet. If the information in Part 1 has interested you and you are considering participation, please continue to read the additional information in Part 2 before making any decision.

8. What will happen if I don't want to carry on with the study?

You are free to leave the study at any time and without giving a reason.

9. Will my part in this study be kept confidential?

All the information about your participation in this study will be kept confidential and data will be anonymous by using 'participant 1' instead of the subject's name. Only the investigators will have access to your name and contact details which will be kept on a password protected computer for 5 years to comply with legislation. The information you provide will be anonymous and audio recordings and transcripts will be either kept on password protected computers or in locked filing cabinets.

10. What will happen to the results of this study?

The results of the study will be available after it finishes. They may be published in a scientific journal or presented at a scientific conference. The data will be anonymous and you will not be identified in any report or publication. Should you wish to see the results of the study, or the publication, please let us know and we will arrange to provide you with these.

11. Who is organising and funding this study?

This is a University of Abertay led study.

12. Contact for further information

You are encouraged to ask any questions you wish, before, during or after the study. Should you have any queries or concerns at any time please contact Thomas Steer; or Dr John Babraj;

Appendix 3

Informed Consent Form

The aim and details of this study have been outlined and explained to me. I understand this study is designed to further sport science knowledge and the University of Abertay Dundee has approved all procedures.

- ☐ I have read and understand all the information provided including this consent form.
- ☐ I have had an opportunity to ask questions about what my participation involves.
- ☐ I understand that I am under no obligation to take part in this study.
- ☐ I understand that I have the right to withdraw from this study at any stage for any reason, and I am not required to provide an explanation for my reasons to withdraw.
- ☐ I understand that all the information I provide will be treated as confidential.
- ☐ I agree to participate in the study

Your name _____

Your signature _____

Signature of investigator _____

Date _____

Appendix 4

Physical Activity Readiness Questionnaire - PAR-Q (revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
☐	☐	**1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?**
☐	☐	**2. Do you feel pain in your chest when you do physical activity?**
☐	☐	**3. In the past month, have you had chest pain when you were not doing physical activity?**
☐	☐	**4. Do you lose your balance because of dizziness or do you ever lose consciousness?**
☐	☐	**5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?**
☐	☐	**6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?**
☐	☐	**7. Do you know of any other reason why you should not do physical activity?**

If you answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active – begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal – this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever – wait until you feel better; or
- if you are or may be pregnant – talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____ DATE _____

SIGNATURE OF PARENT or GUARDIAN (for participants under the age of majority) _____ WITNESS _____

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.

Appendix 5

Immediate Word Recall

Pre-test	*Post-test*
Nine	Horse
Swap	Colour
Cell	Baby
Ring	Sword
Plug	Desk
Lamp	Hold
Apple	Find
Table	Bird
Bird	Rock
Army	Mango
Bank	School
Fire	Fish
Hold	Water
Worm	Light
Clock	Student

Appendix 6

Verbal Fluency

Pre-test	*Post-test*
A S M	S C T

Appendix 7

The Oxford Happiness Questionnaire

Instructions

Below are a number of statements about happiness. Would you please indicate how much you agree or disagree with each by entering a number alongside it according to the following code:

1=strongly disagree; 2=moderately disagree; 3=slightly disagree; 4=slightly agree; 5=moderately agree; 6=strongly agree.

You will need to read the statements carefully because some are phrased positively and others negatively. Don't take too long over individual questions; there are no 'right' or 'wrong' answers and no trick questions. The first answer that comes into your head is probably the right one for you. If you find some of the questions difficult, please give the answer that is true for you in general or for most of the time.

1. I don't feel particularly pleased with the way I am (R).....
2. I am intensely interested in other people.....
3. I feel that life is very rewarding.....
4. I have very warm feelings towards almost everyone.....
5. I rarely wake up feeling rested (R.....
6. I am not particularly optimistic about the future (R).....
7. I find most things amusing.....
8. I am always committed and involved.....
9. Life is good.....
10. I do not think that the world is a good place (R).....
11. I laugh a lot.....
12. I am well satisfied about everything in my life.....
13. I don't think I look attractive (R).....
14. There is a gap between what I would like to do and what I have done (R).....
15. I am very happy.....
16. I find beauty in some things.....
17. I always have a cheerful effect on others.....

18. I can fit in everything I want to.....

19. I feel that I am not especially in control of my life (R).....

20. I feel able to take anything on.....

21. I feel fully mentally alert.....

22. I often experience joy and elation.....

23. I do not find it easy to make decisions (R).....

24. I do not have a particular sense of meaning and purpose in my life (R).....

25. I feel I have a great deal of energy.....

26. I usually have a good influence on events.....

27. I do not have fun with other people (R).....

28. I don't feel particularly healthy (R).....

29. I do not have particularly happy memories of the past (R).....

Calculate your score

Step 1. Items marked (R) should be scored in reverse.

For example, if you gave yourself a '1', cross it out and change it to a '6'.

Change '2' to a '5'

Change '3' to a '4'

Change '4' to a '3'

Change '5' to a '2'

Change '6' to a '1'

Step 2. Add the numbers for all 29 questions. (Use the converted numbers for the 12 items that are revers scored.)

Step 3. Divide by 29. So your happiness score = the total (from step 2) divided by 29.

Your Happiness Score: …….

Appendix 8

Subjective Happiness Scale

Instructions to participants: For each of the following statements and/or questions, please circle the point on the scale that you feel is most appropriate in describing you.

1. In general, I consider myself:

1	2	3	4	5	6	7
not a very happy person						a very happy person

2. Compared to most of my peers, I consider myself:

1	2	3	4	5	6	7
Less happy						more happy

3. Some people are generally very happy. They enjoy life regardless of what is going on, getting the most out of everything. To what extent does this characterization describe you?

1	2	3	4	5	6	7
not at all						a great deal

4. Some people are generally not very happy. Although they are not depressed, they never seem as happy as they might be. To what extend does this characterization describe you?

1	2	3	4	5	6	7
not at all						a great deal

THOMAS STEER
Matriculation number:
Programme: MSc/MBA/MTech/LLM By Research (SHS), year 11

Project Title: **The effect body composition has on adaptations to high-intensity interval exercise.**
Project Reference Number: SHS_R_2014-15_41
Supervisor: JB

Dear Thomas

You have been granted Full Ethical Approval for the above project.

Standard Conditions:

i You must remain in regular contact with your project supervisor.

ii Your supervisor must see a copy of all materials and your procedure prior to commencing data collection.

iii If you make any substantive changes to your proposed project, you must submit a new ethical approval application to the Committee. Application forms and the accompanying explanatory document are on the Intranet. Completed forms should be resubmitted through the Research Ethics Blackboard course.

iv Any changes to the agreed procedures must be negotiated with your supervisor.

Additional Conditions:
A couple of points of clarification: (i) what constitutes overweight in the sample (e.g. as per BMI) and is there a limit on how far overweight a person has to be to be ruled in or out?, and (ii) will participants be informed, or can they ask, if they are within the normal or overweight categories? Or is this information withheld? There may be an ethical point here about whether or not participants may or may not want to take part after they have been told they are within the normal weight bracket or are classified as overweight.

Failure to comply with these conditions will result in your ethical approval being revoked by the Ethics Committee.

Should you have any queries please contact your Supervisor.

Yours sincerely

Research Ethics Committee

School of Social & Health Sciences