

THE EFFECT OF HIGH INTENSITY INTERVAL TRAINING AND DETRAINING ON THE HEALTH-RELATED OUTCOMES OF YOUNG WOMEN

By

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DECLARATION

I, the undersigned, hereby declare that the work contained in this thesis is my own original work and that I have not previously in its entirety or in part submitted it at any university for a degree

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SUMMARY

There is a growing concern in South Africa and worldwide about the global epidemic of obesity and overweightness among the general population. Obesity mediates the pathogenesis of pathological conditions and is associated with a poor quality of life, high morbidity and mortality rates and a huge burden on an individual's and the health system's infrastructure and finances. The answer to this rising epidemic is weight loss. Endurance training has been shown to induce weight loss however, people usually cite lack of time as a barrier to meaningful participation in exercise programmes. High intensity interval training (HIIT) therefore emerges as a potential solution to these barriers as it takes a relatively short period of time compared to endurance training. Despite the differences in exercise durations the most cogent advantage is that HIIT elicits not just similar, but even superior central and peripheral adaptations. The central and peripheral adaptations have been shown to enhance weight loss, improve blood lipids and glucose levels, as well as decreasing blood pressure.

The challenge facing exercise physiologists is to find the optimal exercise intensity and duration of HIIT bouts which would be time efficient, safe and well tolerated by overweight and obese people. The shortcomings of literature are that most HIIT studies have focused on healthy, overweight and obese men and these studies cannot be extrapolated to women who have been shown to respond differently to training. Moreover, other interventions investigating the effects of HIIT in women and men have been longer term rather than short term interventions. In order to fill the gaps in the literature, the main aim of this study was to investigate the training and detraining effects of a short-term HIIT programme on selected health-related measures in young overweight and obese women.

To this end, a non-random sample of 20 overweight and obese women (aged 18-25) volunteered to participate in this study. Selected health-related outcomes were measured prior to training. The pre-training testing was followed by the HIIT intervention which was two

weeks and consisted of six sessions using the 10 – 15x1 minute running at 90% HR_{max} which was separated by one minute active recovery periods at 50-60% of HR_{max} . The HIIT intervention was followed by a post test in which baseline measurements were repeated. This was then followed by a two week detraining period and follow up testing.

The main finding of this study was that a period of two weeks of HIIT can elicit adaptations that can lower the risk profiles of young overweight and obese women. The results showed a statistically significant decrease in body mass (1.6%, $p = 0.001$), fat mass (3.7%, $p = 0.001$) and waist circumference (4.8%, $p = 0.001$), and an increase in lean mass of 1.9% ($p = 0.001$). There was also a decrease in blood glucose (11%, $p = 0.001$), total cholesterol (10.4 %, $p = 0.01$), systolic (3.4%, $p = 0.001$) and diastolic blood pressure (5.8%, $p = 0.001$) levels. Finally there was a statistically significant increase in relative VO_{2max} and exercise capacity after the HIIT

The follow-up testing after two weeks of detraining shows that the metabolic adaptations that were achieved by the HIIT protocol are relatively lasting or are at least not completely reversed. The weight loss induced by HIIT is important in that it is the major target in lowering the prevalence of overweightness and obesity. The HIIT protocol in this study emerges as a time efficient strategy in eliciting positive adaptations in clinical populations and healthy people. Moreover these findings suggest that 10 minute and 15 minute HIIT work bouts at near-maximal intensities are possibly the minimum amount of training that is needed to induce significant weight loss and other positive health-related outcomes.

OPSOMMING

Daar bestaan 'n toenemende besorgdheid in Suid-Afrika en wêreldwyd oor die globale epidemie van obesiteit en oorgewig onder die algemene bevolking. Obesiteit fasiliteer die patogene van verskeie siektetoestande en word met 'n swak kwaliteit lewe, hoë morbiditeit en mortaliteit en 'n geweldige las op 'n individu en die gesondheidsowerhede se infrastruktuur en finansies geassosieer. Een van die antwoorde op hierdie stygende epidemie is gewigsverlies. Dit is reeds gewys dat uithouvermoë oefening saam met 'n kalorie beperkende dieet gewigsverlies in die hand werk. Mense dui egter 'n tekort aan tyd as 'n hindernis tot betekenisvolle deelname aan 'n oefenprogram aan. Hoë intensiteit interval inoefening (HIIO) is dus 'n potensiële oplossing tot hierdie hindernis aangesien dit in vergelyking met uithouvermoë inoefening in 'n relatiewe korter periode van tyd uitgevoer kan word. Afgesien van die verskille in inoefenperiodes is die mees logiese voordeel dat die HIIO nie net soortgelyke nie, maar self beter sentrale en perifere fisiologiese aanpassing voortbring. Die sentrale en perifere aanpassing verhoog gewigsverlies, verbeter bloedlipiedes en glukose vlakke, en veroorsaak 'n afname in bloeddruk.

Alhoewel 'n aantal studies die voordele van HIIO by jonger en ouer populasies aandui, is baie min studies op vrouens uitgevoer. Bevindinge kan nie noodwendig na vrouens ekstrapoleer word nie omdat hulle dikwels verskillend op inoefening as mans reageer. Dit is ook nie bekend of 'n kort HIIO intervensie 'n betekenisvolle impak op oorgewig en vetsugtige vrouens sou hê nie, asook hoe blywend enige veranderinge sou wees nie. Die hoofdoel van hierdie studie was dus om die inoefening- en die geen-inoefening effekte van 'n korttermyn HIIO program op geselekteerde gesondheidskenmerke in jong oorgewig en vetsugtige dames te bepaal.

'n Nie-ewekansige steekproef van 20 oorgewig en vetsugtige vrouens (18-25 jaar) het vrywillig ingestem om aan hierdie studie deel te neem. Geselekteerde gesondheidskenmerke

is voor die aanvang van die inoefening gemeet. Die HIIO intervensie het twee weke geduur en het uit ses sessies bestaan (10 – 15x1 minuut draf by 90% HS_{maks} en een minuut aktiewe herstel by 50-60% HS_{maks}). Die HIIO intervensie is deur 'n na-toets gevolg waarin basislyn metings herhaal is. Dit is deur 'n twee weke geen-inoefening periode en opvolgtoetse opgevolg.

Die hoofbevinding van hierdie studie was dat ses sessies van HIIO fisiologiese aanpassings na vore gebring het wat die risiko profiele van jong oorgewig en vetsugtige vrouens verlaag het. Daar was statisties betekenisvolle afnames in liggaamsmassa (1.6%, $p < 0.001$), vetmassa (3.7%, $p < 0.001$) en heupomtrek (4.8%, $p < 0.001$) en 'n toename in vetvrye liggaamsmassa van 1.9% ($p < 0.001$). Daar was ook 'n afname in bloedglukose (11%, $p < 0.001$), totale cholesterol (10.4 %, $p = 0.01$), sistoliese (3.4%, $p < 0.001$) en diastoliese bloeddruk (5.8%, $p < 0.001$). Daar was ook statisties betekenisvolle verbeteringe in relatiewe VO_{2maks} en oefeningtoleransie na inoefening.

Die opvolgtoetse na twee weke van geen-inoefening het getoon dat metaboliese aanpassings wat deur die HIIO bereik is, relatief blywend van aard was of ten minste nie totaal omgekeerd was nie. Die gewigsverlies wat deur die HIIO veroorsaak was is belangrik in die sin dat dit die hoofdoelwit aanspreek om die voorkoms van oorgewig en vetsugtigheid te verminder. Die studie suggereer verder dat 10 – 15 minute HIIO werksessies, by naby maksimale intensiteite, moontlik die minimum hoeveelheid inoefening is wat benodig word om betekenisvolle gewigsverlies en ander positiewe gesondheidskenmerke te bereik.

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DEDICATION

I dedicate this thesis to my dad who has been my inspiration and role model.

LIST OF ABBREVIATIONS

AT	:	anaerobic threshold
ATP	:	adenosine diphosphate
BMI	:	body mass index
BIA	:	bioelectrical impedance analysis
Bpm	:	beats per minute
COX I	:	cytochrome oxidase subunit 1
COXIV	:	cytochrome oxidase subunit 4
CT	:	continuous training
CS	:	citrate synthetase
DBP	:	diastolic blood pressure
EPOC	:	excess post-exercise oxygen consumption
FBLA	:	fixed blood lactate accumulation
GLUT4	:	glucose transporter isoform 4
H ⁺	:	hydrogen ion
HIIT	:	high intensity interval training
HR	:	heart rate
HR _{max}	:	maximum heart rate
Kg	:	kilogram(s)
kJ	:	kilojoule(s)
Km/h	:	kilometres per hour
La _{max}	:	maximum lactate
LDH	:	lactate dehydrogenase
LT	:	lactate threshold
LM	:	lean mass

L/min	:	litres per minute
MDH	:	malate dehydrogenase
mmHg	:	millimetres mercury
ml/min/kg	:	milliliter per kilogram per minute
mM/l	:	millimole per liter
NADH	:	nicotinamide adenine dinucleotide hydrogen
N	:	number of people
PGC-1 α	:	Peroxisome proliferator-activated receptor γ coactivator 1 α
Q_{\max}	:	maximal cardiac output
RPE	:	ratings of perceived exertion
SDH	:	succinate dehydrogenase
SBP	:	systolic blood pressure
SD	:	standard deviation
VE_{\max}	:	maximum minute ventilation (l/min)
$VO_{2\max}$:	maximal oxygen uptake
WL_{\max}	:	maximum work load
Δ	:	change

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Chapter 1

INTRODUCTION

High intensity interval training (HIIT), a form of aerobic training, has been widely used in sport to develop physical fitness, induce physiological adaptations and improve health and sports performance. HIIT is an exercise method that is characterised by brief intermittent bursts of vigorous activity, interspersed by periods of rest or low intensity exercise (Gibala and McGee, 2008). Typical training programmes that have been described in the literature last between two and six weeks (Astorino *et al.*, 2012).

HIIT is also referred to as sprint interval training (SIT) and high intensity intermittent training. HIIT began to emerge in the 1960s in Sweden. Physiologists lead by Per Astrand performed ground-breaking research demonstrating how manipulation of work and rest durations could dramatically impact physiological changes to intermittent exercise (Astrand *et al.*, 1960a; Astrand *et al.*, 1960b; Christensen, 1960a; Seiler *et al.*, 2009). In later physiological fitness studies the outcomes of continuous training (CT) and HIIT interventions were compared in athletes and inactive individuals.

The physiological responses to CT are well described in the literature. It improves cardio-respiratory fitness by increasing cardiac output (in ~3wks) and arterial–venous oxygen difference (in ~4-6 wks), resulting in a greater maximal endurance capacity. CT also improves submaximal exercise capacity, reduces submaximal heart rate, changes substrate utilization (i.e. more fat oxidation) and decreases body mass. On the other hand, the physiological adaptations associated with HIIT cause enhanced aerobic performance within a period of 2 – 15 weeks, however, through similar mechanisms than CT. Previous studies have highlighted rapid skeletal muscle adaptations as shown by changes in oxidative enzyme

activity, resulting in enhanced skeletal muscle fat oxidation, improved glucose tolerance, improved insulin sensitivity and weight loss (Nybo *et al.*, 2010; MacPherson *et al.*, 2011; Trapp *et al.*, 2008; Daussin *et al.*, 2008; Talanian *et al.*, 2007). In addition, HIIT enhances cardiovascular function by increasing cardiac output (in ~4-6 weeks) and the arterial-venous difference (in ~2 weeks), thus improving maximal oxygen uptake (VO_{2max}) (Astorino *et al.*, 2012; Trilk *et al.*, 2011; Bailey *et al.*, 2009; Talanian *et al.*, 2007). Additionally, HIIT has the additional advantage of simultaneously enhancing anaerobic performance by increasing muscle buffering capacity, glycolytic enzymes and ionic regulation (Hazell *et al.*, 2010; Burgomaster *et al.*, 2007, 2006, 2005; Harmer *et al.*, 2000; Stathis *et al.*, 1994). Thus, despite the differences in total exercise duration, CT and HIIT induce similar physiological adaptations in the body when the programmes are matched for total work done. However, as shown by Gorostiaga (1991), HIIT may actually induce greater changes in VO_{2max} and peak power output (9-16%), compared to CT (5-7%) in untrained individuals when the two interventions are matched for total work.

Several studies have been done on team sport athletes, with most focusing on football (Driller *et al.*, 2009; Iaia *et al.*, 2009). The studies used HIIT training with work and rest intervals ranging from 15 sec to 4 min at 90 to 100% VO_{2max} , with heart rate values >90% of maximal heart rate and work to rest ratios of 1:1 – 4:1. It was shown in these studies that HIIT elicited increases in cardiovascular parameters such as heart size, blood flow capacity and arterial distensibility (Rakobowchuk *et al.*, 2009, 2008; Laughlin *et al.*, 2008;). These changes improved the capacity of the cardiovascular system to transport oxygen, resulting in faster muscle and pulmonary VO_2 kinetics and higher VO_{2max} . This enabled a greater amount of energy to be supplied aerobically, allowing a player to sustain intense exercise for longer durations, as well as recovering more rapidly between high intensity phases of the game (Iaia *et al.*, 2009).

Many studies use the Wingate test as a HIIT intervention (Astorino *et al.*, 2012; Bayati *et al.*, 2011; Trilk *et al.*, 2011; MacPherson *et al.*, 2011; Burgomaster *et al.*, 2008, 2007). Usually four to six bouts of work are done per session, separated by 4 minutes of recovery, giving a total of 2-3 minutes of intense exercise during a training session that lasts 20 minutes. As few as three sessions have been shown to improve skeletal muscle metabolite proteins normally associated with endurance training, such as glucose transporter isoform 4 (GLUT4) and cytochrome oxidase subunit 4 (COX4). For instance, Burgomaster *et al.* (2007) reported an increase in COX4 and GLUT4 of approximately ~17% and ~15%, respectively. GLUT4 transports glucose from the blood into the cell, while COX4 is used in the cell for the metabolism of glucose to produce ATP and it is a marker of mitochondrial content. An increase in these proteins causes an enhanced capacity for substrate transportation and utilization, glucose metabolism as well as fatty acid oxidation. This demonstrates the potency of HIIT to induce weight loss in individuals.

Although different HIIT exercise protocols are used in various studies, the majority of research shows that maximum exercise capacity can be significantly improved, even within a short period of six sessions. Furthermore, the physiological adaptations that are made are similar to a traditional continuous endurance training programme. What is not well described is the effect of detraining on these physiological adaptations. Furthermore, the effect of HIIT on the health-related outcomes of young overweight and obese women have also not been studied.

Chapter 2

HIGH INTENSITY INTERVAL TRAINING

2.1. INTRODUCTION

This chapter aims to explore the theoretical framework and underlying mechanisms of the physiological adaptations associated with HIIT. HIIT has been demonstrated to elicit similar physiological adaptations compared to endurance training matched for total work done, despite the differences in total exercise duration per session. It is suggested that HIIT may be an effective tool for promoting health and well-being in the general population.

2.2. TYPES OF AEROBIC TRAINING

2.2.2 Continuous Training

The traditional view of aerobic training has been continuous training (CT) which is characterised by long uninterrupted work ranging from low intensity to high intensity (Willmore and Costill, 1994). This then leads to two types of continuous training, namely high intensity continuous training and low-slow distance training. A constellation of sports make up aerobic training; these include running, swimming, cycling, walking, dancing, steps, aerobics and many other.

High intensity continuous training is characterised by work done at 80 - 95% of maximum heart rate (HR_{max}) or peak work rate (PWR) for a long duration (Vogiatzis *et al.*, 2002; Willmore and Costill, 1994). This type of training exerts a lot of stress on the human body and is usually gradually integrated into an athlete's training program. This type of training aims to simulate race times where athletes need to maintain speed and an even pace throughout a race.

Low slow distance exercise is characterised by work done at low intensities which usually elicit 50-75 VO_{2max} or HR_{max} (Helgerud *et al.*, 2006; Eddy *et al.*, 1977). This type of training

aims at enhancing muscle endurance (Gorostiaga *et al.*, 1991). A typical training session entails exercise done for longer than 30 minutes. This is the most common type of training in sport and wellness settings and is well tolerated by different populations, from the young to the elderly, as distance or time can be adjusted to suit each population. It can therefore be used to fulfill different outcomes, varying from those who want to stay fit or maintain weight and for conditioning purposes in competitive sport (Wilmore and Costill, 1994).

2.2.3 High intensity interval training

Interval training is characterized by repeated work bouts interspersed by recovery periods (Hargreaves, 1995; Elliot, 1999). It involves the manipulation of intensity and duration of work to rest ratios, where the resting periods allow the individual to rest and recover between sets. The rationale for this type of training is that more work can be done during a session if exercise is broken down into intervals, while the rest periods give time for recovery (Wilmore and Costill, 1994). This type of training stresses the physiological systems of the body to a greater extent than continuous training, because the sprint bouts are done at intensities that are higher than the anaerobic threshold.

The work done usually elicits approximately 85-95% HR_{max} (Kokkins, 2012) and rest intervals can either be passive or active. In the latter case light activity is done at 50 - 60% HR_{max} (Powers and Howley, 2004). Astrand (1980) suggested that the heart beat should drop to 120 beats per minute before the commencement of the next work bout. This type of training can be adapted according to the primary purpose of the activity. There are five variables that can be modified, namely i) rate and distance of the work interval, ii) number of repetitions and sets during a session, iii) duration of the rest interval, iv) type of activity during the rest interval and v) frequency of training per week (Wilmore and Costill, 1988; Fox and Mathews, 1974). Interval training has been shown to be more effective in exercising the principles of overload and progression, as one can adjust any of the five variables.

2.3. MODELS OF HIGH INTENSITY INTERVAL TRAINING

Two models of HIIT have been developed as a way of advancing and creating variation in physical activity.

2.3.1 Cycling Models

In the cycling model the most common method that has been used is the Wingate Test (Gibala, 2007). The Wingate Test consists of a 30-seconds all out cycling effort against a supra maximal workload. Usually four to six bouts of work are done, separated by 4 minutes of recovery, giving a total of 2-3 minutes of intense exercise during a training session that lasts 20 minutes in total. This test has been used in a number of studies (Burns *et al.*, 2012; Astorino *et al.*, 2012, 2011; Whyte *et al.*, 2010; Burgomaster *et al.*, 2008, 2007) and has been demonstrated to be a good stimulus for promoting skeletal muscle metabolic adaptations. The metabolic adaptations mainly include an increase in mitochondrial markers for protein proliferator-activated receptor γ coactivator 1 (PGC1-), carbohydrate oxidation (pyruvate dehydrogenase), lipid oxidation (3 hydroxyacyl CoA dehydrogenase maximal activity) and citrate synthase activity (Burgomaster *et al.*, 2008, 2005; Gibala *et al.*, 2006).

It is also important to note that the Wingate protocol has been reported in some studies to have some negative side effects on participants. This is because participants reported feelings of nausea, dizziness and severe fatigue (Astorino *et al.*, 2011). This kind of intervention may therefore be limited to certain populations who can tolerate this type of exercise.

A more practical HIIT cycling model was developed by Little *et al.* (2010). In this newer model eight to twelve 60-seconds work bouts at 100% VO_{2max} are done. These were split by 75 seconds of recovery giving a total exercise time of 30 min. This model increased resting muscle glycogen by 17%, GLUT4 by 119%, mitochondria transcription factor A (Tfam) total protein content by 37%, as well as the regulators of mitochondrial biogenesis, PGC1 (24%) and transcription factor (SIRT 1) (56%) in healthy active men (Little *et al.*, 2010). It also

increased mitochondrial enzymes, citrate synthase CS (16%), cytochrome oxidase subunit 4 COX (29%), and COX sub units II and IV (35-38%). Citrate synthase is involved in the synthesis of energy by the Krebs cycle, while cytochrome oxidases are involved in the synthesis of energy by the electron transport chain (ETC) (Katch *et al.*, 2011; McArdle *et al.*, 2001) An increase in these enzymes increases the aerobic energy deriving capacity of the body.

It is important to note that this model is still time efficient and induces the same physiological changes than the Wingate test. However, what makes this cycling protocol more superior is that it was well tolerated by participants as they did not report any feelings of dizziness, light headedness or nausea after the exercise (Little *et al.*, 2010).

The cycling models vary in their work to rest ratios from 1:1 to 1:6. These modifications may potentially cause differences in physiological adaptations. Hazell *et al.* (2010) addressed this issue in their study where they had three different groups exercising at different work to rest ratios and a fourth group which was the control group and did not exercise. The participants were physically active healthy men and women (Hazell *et al.*, 2010).

For the first group, the HIIT protocol consisted of 30-seconds work bouts interspersed by 4 minute active recovery periods. The second HIIT protocol consisted of 10 seconds work bouts interspersed by 4 minute active recovery periods, while the third group performed 10 seconds work bouts interspersed by 2 minute active recovery periods. The numbers of work bouts were increased from four in the first two sessions to five in the next 2 sessions and to six in the last two sessions to factor in progression.

The results from this study demonstrated that the work to rest ratios did not compromise training adaptations associated with HIIT, as all the protocols induced similar changes in terms of the increase in VO_{2max} . In group's one and two, VO_{2max} increased by 9.3% and 9.2%, respectively ($p < 0.05$). Group three improved by 3.8%, although this change was not

statistically significant ($p=0.06$). Peak power output also increased significantly by 9.5%, 8.5% and 4.2% in group's one, two and three respectively, while no significant difference was recorded between groups. The results of this study suggested that two- to four-minute recovery periods in HIIT protocols are enough to enable recovery for the next work bout of an all-out effort.

2.3.2 Advantages and disadvantages of cycling models

Given the knowledge that people usually mention a lack of time as a barrier to their participation in physical activity, the HIIT protocol addresses this as it takes a short period of time (which is nearly half that of endurance training) and is associated with long term adherence to exercise (Gibala, 2007). Moreover, the Wingate test has also been used in overweight and obese populations (Trilk *et al.*, 2011; Whyte *et al.*, 2010). On the other hand, the Wingate Test has disadvantages in that it can only be done on a specialized cycle ergometer and it requires high levels of motivation to be given to participants during the training period (Boutcher, 2011; Gibala, 2007). Feelings of discomfort, nausea and light headedness have been reported in some studies using the Wingate test, although this is not a universal finding in all studies (Astorino *et al.*, 2011).

2.3.3 Running Models

Running is an inborn ability and a natural progression from learning to walk. All individuals of different ages and body size can take part in walk/run exercise and still feel comfortable (Hawley, 2000). The running versions used to date in HIIT interventions vary in intensity, as well as in the work to rest ratios and their duration. The intensity has been reported to vary from 80 to 90% of VO_{2max} or HR_{max} , while the duration of the work and rest intervals ranges from one to four minute minutes (Tjonna *et al.*, 2013; Nybo *et al.*, 2010; Bravo *et al.*, 2008; Wisloff *et al.*, 2007). The warm up and cool down sessions range from five to ten minutes. Importantly, the running HIIT version has been used in healthy individuals and even in clinical populations, such as those with chronic obstructive pulmonary disease COPD (Orio *et*

al., 2008), diabetes and coronary artery disease (Gibala *et al.*, 2012), stroke, heart failure and overweight women (Smart, 2013). In a three month HIIT study of overweight adolescents, Tjonna *et al.* (2009) reported an 11% increase in VO_{2max} , while Warburton *et al.* (2005) reported a 31.8% increase in aerobic capacity in patients with coronary artery disease after 16 weeks of training.

Research also shows that running interventions cause similar physiological adaptations than cycling exercise. Bartlett *et al.* (2012; 2011) reported increases in PGC-1 α , messenger ribonucleic acid (mRNA), AMPK and p38MAPK phosphorylation, as well as exercise induced p53 phosphorylation which results in an increase in aerobic capacity.

HIIT running versions have also been used in sport studies (Fernandez-Fernandez *et al.*, 2012; Iaia *et al.*, 2009; Tanisho and Hirakawa, 2009) and mainly with soccer players. Bravo *et al.* (2008) showed a 5.9% and 12.5% increase in VO_{2max} and the Yoyo Intermittent Test, respectively, in football players. Fernandez-Fernandez *et al.* (2012) reported a 6% ($p=0.008$) increase in VO_{2peak} in competitive male tennis players after six weeks of training. Tanisho and Hirakawa (2009) also investigated the effect of 15 weeks of HIIT (10x10 sec separated by 20 sec active rest) in competitive lacrosse players. They reported a 9.9%, 6% and 9.5% increase in VO_{2max} , maximal anaerobic power and mean power output, respectively, thus demonstrating the effectiveness of HIIT in ball games (Tanisho and Hirakawa, 2009).

A recent novel finding by Tjonna *et al.* (2013) showed that one single bout of HIIT elicited similar adaptations than the 4x4 min running HIIT (4-HIIT) protocol in overweight men (Tjonna *et al.*, 2009). The single HIIT (1-HIIT) session consisted of one 4-minute running bout at 90% HR_{max} , and this was done three times a week for a period of 10 weeks. The results from this study are remarkable in that they recorded a reduction in fasting glucose of 6% (1-HIIT) and 5% (4-HIIT) and body weight loss of 1% (1-HIIT) and 2% (4-HIIT). VO_{2max} also increased by 10% (1-HIIT) and 13% (4-HIIT), while systolic blood pressure

(SBP) was decreased in both protocols by 6.2% and 3.2%, respectively. Similarly, diastolic blood pressure (DBP) also reduced by 7.7 mmHg (1-HIIT) and 6.3mmHg (4-HIIT). Overall, there were no significant differences between the two groups, implying that 1-HIIT induces similar adaptations to that of the 4-HIIT, which has also been demonstrated to trigger adaptations similar to endurance training. This reiterates the fact that HIIT is a time efficient strategy for promoting health and aerobic fitness.

2.3.4 Advantages and disadvantages of running models

The running HIIT models are advantageous in that taking up running as an exercise intervention does not need any specialist equipment hence it is a low cost intervention program (Bartlett *et al.*, 2011). Furthermore, participants undertaking this type of exercise have reported feelings of enjoyment compared to the continuous training which they described as 'boring'. This is particularly important as exercise enjoyment also determines exercise adherence (Bartlett *et al.*, 2011). The running HIIT models are also more favorable because no feelings of nausea and dizziness have been reported in studies.

Research studies have shown that running induces higher rates of fat oxidation compared to cycling when matched for exercise intensity (60 to 80% of VO_{2max}) in trained and untrained individuals (Capostagno and Bosch, 2010; Knechtle *et al.*, 2004 Achten *et al.*, 2003; Knechtle *et al.*, 2004). The higher fat oxidation with running may be attributed to more muscle fiber recruitment during exercise, in particular through type I fibers (Achten *et al.*, 2003; Carter *et al.*, 2000).

The disadvantage of some HIIT running protocols is that they take longer time, namely from 30 to 55 minutes, including warm up and cool down periods. This is relatively longer than the Wingate cycling protocols which take 15 minutes (Little *et al.*, 2011). This can be solved by adopting the 1-HIIT protocol which lasts for four minutes only, thus making it a time efficient

training strategy. Moreover, the 1 HIIT protocol also lasts a shorter time than the cycling protocols.

On the whole the two HIIT models have been shown to elicit physiological adaptations that are normally associated with traditional endurance training. The running HIIT models seem to have more advantages than the cycling protocols. More importantly is the duration of the HIIT sessions which last nearly half that of endurance training, which will also contribute to lower injury risks especially in those who are not accustomed to training. It is thus safe to assume that it will increase exercise adherence, help promote health and lower the risk of morbidity and mortality, not only in a healthy population, but also in overweight, obese and diseased populations.

2.4. METABOLIC EFFECTS AFTER HIIT

Exercise plays a major role in inducing weight loss by increasing the basal metabolic rate of the individual for several hours after an exercise session. DeVries and Housh (1994) showed increases in metabolic rate between 7.5% and 25% up to six hours after one hour of mixed aerobic exercise. They also suggested that to effectively induce weight loss, one should engage in vigorous endurance training, in a bid to maximize energy expenditure. In a review Hunter *et al.* (1998) reported that HIIT elicits a 5- 15% increase in resting energy expenditure (REE) after exercise, and this remains elevated for 24 - 48 hours after the last bout. This increase in REE is an advantage in that it induces a negative energy balance due to the differences in REE and food intake, provided the individual does not over eat after exercise. It is also important to note that HIIT has a tendency of suppressing appetite shortly after exercise and this may contribute positively towards a negative energy balance (Martins *et al.*, 2008; Hunter *et al.*, 1998; Kissileff *et al.*, 1990). However, this phenomenon may be limited to individuals with a healthy weight. Kissileff *et al.* (1990) demonstrated that vigorous exercise suppresses appetite more in non-obese subjects than moderate exercise during a three

day exercise programme. On the contrary, the overweight subjects partaking in the vigorous training did not reduce their food intake after the exercise (Kissileff *et al.*, 1990).

Little *et al.* (2011) measured the body's response to one bout of HIIT in habitually active men. The HIIT bout consisted of four 30-seconds maximal cycling interspersed with four minute rest periods. They reported an increase in maximal activities of citrate synthetase (CS) (14%, $p = 0.024$) and COX (19%, $p = 0.10$) at 24 hours after exercise. PGC-1 α increased by 66% at 3 hours post- exercise, however, this increase returned to pre-training values after 24 hours. They also reported an increase in the protein content of CS, COX II and COX IV of 30%, 29% and 43% respectively, and increases in the maximal activity of CS (14%, $p = 0.024$) and COX (19%, $p = 0.10$) 24 hours after the HIIT exercise. All these results thus indicate that an acute session of HIIT can significantly increase mitochondrial markers and enzymatic activities involved in aerobic metabolism and consequently enhance aerobic capacity (Gibala *et al.*, 2012; Little *et al.*, 2010).

2.5. ADAPTATIONS TO AEROBIC TRAINING

There is overwhelming evidence that regular physical activity enhances physical fitness, aids in weight loss and causes positive adaptations in the body ranging from hormonal, musculoskeletal and cardiorespiratory changes (Tjonna *et al.*, 2013; Ho *et al.*, 2012; Gibala *et al.*, 2012; Boutcher, 2011). Most studies contend that aerobic exercise plays a crucial role in promoting health and wellbeing (Golac *et al.*, 2010; Hagobian *et al.*, 2008; Nemato *et al.*, 2007; Baar *et al.*, 2002; Manson *et al.*, 2002).

Central adaptations attributed to continuous training and HIIT include an increase in muscle and cutaneous blood flow, cardiac output (specifically stroke volume), plasma volume as well as lowering resting heart rate. The peripheral adaptations occur at the working muscle and increase its utilization of oxygen. These adaptations include an increase in mitochondrial capacity and capillary density.

2.6. METABOLIC ADAPTATIONS TO HIIT

Adaptations to endurance training in the muscles can occur at two levels. On the one hand muscle adaptations can occur at a structural level where there is a modification of actin and myosin (Birch *et al.*, 1995). On the other hand adaptations to training can occur at a functional level whereby there is an increase in the maximal activities of cytoplasmic enzymes, as well as an increase in mitochondrial density (either an increase in the number of mitochondria or mitochondria size, or both) (Kraemer *et al.*, 2012). The increase in mitochondrial density triggers an increase in the aerobic enzymes (Kraemer *et al.*, 2012). For instance, Hawley and Stepto (2001) reported a 95% increase in succinate dehydrogenase (an enzyme involved in the Krebs cycle) in endurance trained cyclists.

2.6.1 Carbohydrate metabolism

Laboratory studies have demonstrated that the increase in oxidative capacity after HIIT training is a result of an increase in the mitochondrial enzymes. First there is an increase in the maximal activity of the enzyme citrate synthase (CS) which has been observed ranging from 5 to 35% in healthy subjects after HIIT (Little *et al.*, 2010; Perry *et al.*, 2008; Talanian *et al.*, 2007; Burgomaster *et al.*, 2006).

Burgomaster *et al.* (2006) reported an 11% up regulation of the maximal activities of CS in recreationally active men after two weeks of Wingate sessions. Talanian *et al.* (2007) in their study involving recreationally active women reported a 20% increase in CS following seven HIIT sessions over a two week period. Perry *et al.* (2008) also reported an increase in the activity of citrate synthase enzyme of 26% following six weeks of HIIT and Little *et al.* (2010) reported a 16% and 20% increase in the maximal activities and protein content of CS in their modified HIIT protocol (in section 2.3.1) in young healthy men. The improvement in the muscle's oxidative capacity enhances the oxidation of fat thus reducing the risks of metabolic disorders such as insulin resistance (Gibala, 2007).

Another enzyme that has been shown to increase its activity after HIIT training is Cytochrome oxidase subunit 4 (COX4) and this was also within as short a period as two weeks. This is especially interesting because these findings are similar to those shown after endurance training with a vast difference in total exercise times. Burgomaster *et al.* (2007) reported a 35% increase in COX 4 in healthy active men after only one week of HIIT, using the Wingate protocol. Subsequently Perry *et al.* (2008) reported an 18% increase in COX4 following 6 weeks of HIIT in physically active men, thereby confirming the earlier findings of Dudley *et al.* (1982) who also reported an increase in the activity of Cytochrome oxidase after a 6 week HIIT program.

Increases in the maximal activities of malate aspartate and pyruvate dehydrogenase have also been reported following HIIT. A 26% increase in the maximal activities of malate aspartate (enables the oxidation of NADH) and 21% increase in pyruvate dehydrogenase have been observed (Perry *et al.*, 2008), resulting in an increase in carbohydrate and fat oxidation capacities. Another enzyme, succinate dehydrogenase, which is a key enzyme in the Krebs cycle, has also been reported to increase after HIIT. MacDougall *et al.* (1998) reported a 65% increase in succinate dehydrogenase following seven weeks of HIIT (Wingate protocol) in physically active men.

Moreover, HIIT has also been shown to increase anaerobic capacity, as demonstrated by an up regulation of glycolytic enzymes. This was demonstrated by MacDougall and colleagues (1998) when they reported increases in the maximal activities of hexokinase (56%) and phosphofructokinase (49%) following HIIT.

To conclude, the increases in the muscle's oxidative capacity observed after HIIT is related to the fluctuations in workloads, rather than exercise duration and net total energy expenditure (Daussin *et al.*, 2008). Exercising at higher intensities subsequently decreases the ATP:ADP ratio, signaling an increase in the muscle's reliance on carbohydrate oxidation, resulting in

greater production of ATP per molecule of glucose (ATP : O₂ = 3) than fat (ATP: O₂ =2.8) (Atwood and Bowen, 2007; Noakes, 2001).

2.6.2 Lipid metabolism

During exercise an increase in blood flow to the adipose tissue induces an increase in the free fatty acid (FFA)/ albumin ratio thus resulting in an increase in FFA utilisation (Hargreaves 1995). Upon reaching the cell the enzyme carnitine transferase catalyses the release of FFAs into the mitochondria of the cell where β -oxidation takes place (Powers and Howley, 1994). Contradictory results have been reported on the effect of HIIT on fat metabolism, in that earlier studies investigating the effect of HIIT reported no changes in the maximal activity of hydroxyacyl-CoA dehydrogenase (HAD) which is a marker of β -oxidation (Burgomaster *et al.*, 2007, 2006). This is in contrast with other research studies, in which an up regulation of the maximal activities of HAD have been reported after HIIT.

Talanian *et al.* (2007) reported a change in lipid metabolism after seven sessions of HIIT. There was a significant increase in the maximal activity of HAD (32%, $p < 0.05$) and fatty acids binding protein (FABP_{pm}; 25%, $p < 0.05$) and thus an increase in whole body fat oxidation. This was further confirmed by Burgomaster *et al.* (2008) in their six week HIIT study, when they reported an up regulation of HAD activity (24%, $p < 0.05$). Similarly, Perry *et al.* (2008) (same protocol as Talanian *et al.*, 2007) reported an increase in fat oxidation in response to six weeks HIIT training in healthy, physically active men and women. They measured increases in fatty acid translocase (FAT/CD36; 16%, $p < 0.05$) and fatty acid binding protein (FABP_{pm}; 30%, $p < 0.05$). They also reported a 29% ($p < 0.05$) increase in the maximal activity of β HAD.

Burgomaster *et al.* (2008) suggested that a minimum volume of intense interval training is necessary to induce adaptations in lipid metabolism, as their results in 2008 were different from their earlier studies (Burgomaster *et al.*, 2007, 2006). However, at this point it is unclear what

the minimum amount of exercise is that would lead to increases in fat oxidation, although it seems that any of the protocols of Burgomaster *et al.* (2008), Talanian *et al.* (2007) and Perry *et al.* (2008) could be recommended for weight loss programmes.

Endurance training has also been shown to increase substrate availability which is demonstrated by an increase in carbohydrate and triglyceride availability (Kraemer *et al.*, 2012). HIIT has also been shown to increase the body's utilization of fat and reduce its reliance on glucose and glycogen (Gibala, 2007). This then increases glycogen content as shown by higher resting muscle glycogen levels (17%) following HIIT, whilst reducing the rate of glycogen utilization by the muscle (Little *et al.*, 2011). Perry *et al.* (2008) reported a 59% increase in glycogen content following six weeks of HIIT. The benefit of an increase in substrate availability is that it enhances one's capacity to sustain exercise for a longer duration.

2.6.3 Metabolite accumulation

HIIT causes a substantial increase in the local production of lactic acid and H⁺ (Edge *et al.*, 2006) which will lead to an increase in the acidity of the blood. An increase in acidity is detrimental to performance because it inhibits the optimal activities of enzymes involved in energy metabolism, such as phosphofructokinase (PFK), ATPases and glycogen phosphorylase (Kraemer *et al.*, 2012; Birch *et al.*, 2005). Furthermore, a lowering of pH affects the release of calcium from the sarcoplasmic reticulum and impairs the binding of calcium to troponin-C in the cross bridges (Kraemer *et al.*, 2012). However, lactate can be removed from muscle via the sarcolemmal transporters, MCT1 and MCT4 (Juel, 1999); these transporters are stereo selective for lactate and depend on the pH gradient for transportation.

It has been suggested that HIIT increases the muscle's buffering capacity (Edge *et al.*, 2006; Hashimoto *et al.*, 2007). Edge *et al.* (2006) compared the effects of HIIT and continuous training (CT) on muscle buffering capacity in young (20 ± 1 years) recreationally active

women in a five week cycling study. The HIIT and CT protocols were matched for total work done. They reported a significant increase in muscle buffer capacity (25%, $p < 0.05$) compared to the CT group (2%; $p > 0.05$). Thus it was suggested that the higher intensity of training might be a more potent stimulus to induce improvements in muscle buffer capacity compared to CT. These findings are supported by Hashimoto *et al.* (2007) who demonstrated how the addition of lactate ions to a tissue culture resulted in an increase in the expression of mRNA for MCT1 transporters.

However, *in vitro* studies did not yield the same results, as they found no significant change in MCT when lactate and H^+ were increased during exercise (Mohr *et al.*, 2007; Juel *et al.*, 2004). This absence of change in MCT can be attributed to the fact that it is not only lactate that accumulates during exercise, but H^+ ions as well, and this can have a detrimental effect on muscle buffering (Mohr *et al.*, 2007; Edge *et al.*, 2006; Juel *et al.*, 2004). Edge *et al.* (2006) suggested that the duration of the work bouts and the rest periods largely influences the muscle pH regulating systems. It seems that short intervals of one minute exercise and recovery each lead to a decrease in intracellular buffer capacity, while three minute rest periods between the one minute bouts resulted in an increase in intracellular buffering capacity. This suggestion was supported by Bishop *et al.* (2008), namely that short recovery periods between work bouts facilitate a decrease in the muscle's buffering capacity and no absolute change in the expression of MCT1. This could be attributed to the reduction of the intracellular buffers (phosphate) after training because of the great acidic load placed on the body during HIIT (Mannion *et al.*, 1993).

Studies investigating the accumulation of lactate and H^+ in muscle after training have reported a reduction in lactate and H^+ production (Bishop *et al.*, 2008; Krustup *et al.*, 2006; Harmer *et al.*, 2000). This reduction in lactate and H^+ is likely due to an increase in lactate removal or a reduction in lactate production (Philips *et al.*, 1995). Burgomaster *et al.* (2007) showed an

increase in the sarcolemmal lactate proton co-transporters after HIIT. An increase in these co-transporters indicates an enhanced rate of lactate removal. Burgomaster *et al.* (2007) reported a 50% increase in MCT1 and 44% increase in MCT4 after just a week of HIIT training. Similarly, Perry *et al.* (2008) reported a 14% and 16% increase in MCT2 and MCT4 protein content thus enhancing lactate removal capacity. In summary, HIIT training has been shown to enhance the rate of lactate removal in the muscles leading to an increase in exercise capacity.

2.6.4 Exercise capacity

Exercise also improves the capacity of the body to sustain strenuous activities for long periods, as well as improve exercise performance in tasks which rely on aerobic metabolism (Gibala and McGee, 2008; Simoneau *et al.*, 1985). Research evidence shows that HIIT also improves exercise capacity as shown by improvements in mean peak power output and time trial performances. Burgomaster *et al.* (2006) reported an improvement of 9.6% ($p = 0.04$) in the time taken to complete a 250 kJ cycle trial (equal to 10km) after two weeks of Wingate sessions, while mean power output increased by 5.4% ($p = 0.04$). Perry *et al.* (2008) also reported a 21% ($p < 0.05$) increase in peak power output in healthy, physically active men and women after six weeks of HIIT.

Improvements in exercise capacity were also demonstrated by Hazell *et al.* (2010) who used physically active men and women in their two week HIIT protocols (mentioned in 2.3.1). They reported an increase in time trial performance of 5.2%, 3.5 % and 3.0 % in the different groups (varying work to rest ratio). Similarly Little *et al.* (2010) reported an improvement in time trial performance of 11% ($p = 0.04$) and 9% ($p = 0.05$) in the 50kJ and 750kJ respectively. This was after a two week HIIT intervention program (section 2.3.1) in recreationally active men (Little *et al.*, 2010). Astorino *et al.* (2011) also reported an increase in mean power output of 10.4% in men and 10.9% in women during cycle time trials. These

studies collectively show that even very short HIIT interventions can significantly improve exercise capacity.

2.6.5 Peroxisome proliferator-activated receptor γ coactivator 1 α

Peroxisome proliferator-activated receptor γ coactivator 1 α (PGC-1 α), a key regulator of oxidative enzyme expression, is a transcriptional coactivator which serves to co-ordinate mitochondrial biogenesis in human muscle (Baar *et al.*, 2002). PGC-1 α is responsible for the activation of several transcriptional factors which lead to activation of mitochondrial and metabolic adaptations (Lin *et al.*, 2005; Wu *et al.*, 1999). This includes increases in insulin sensitivity, glucose uptake, anti-oxidant defense and protection against age related sarcopenia, as well as increasing the maximal activities of oxidative enzymes and exercise capacity (Gibala *et al.*, 2012; Bartlett *et al.*, 2012; Olesen *et al.*, 2010; Wende, 2007).

PGC-1 α being a master regulator for mitochondrial biogenesis is activated by a number of factors. In this particular review focus is placed on the exercise induced expression of PGC-1 α in muscle. This seems to be triggered by the disturbance of the homeostatic environment at the onset of exercise which includes an increase in the AMP/ATP ratio, reactive oxygen species (ROS), lactate, Ca²⁺, as well as a reduction in glycogen availability. This disturbance in homeostasis activates a number of protein kinases which also phosphorylate transcriptional factors or transcriptional coactivators; these then converge to regulate the expression of PGC1 α as shown in Figure 2.1 (Bartlett *et al.*, 2012). These factors include; adenosine monophosphate –activated protein kinase (AMPK), p38 mitogen-activated protein kinase (p38MAPK), calmodulin –dependent protein kinase (CaMK), reactive oxygen species (ROS) and sirtuin 1 (Olesen *et al.*, 2010; Koulmann and Bigard, 2006).

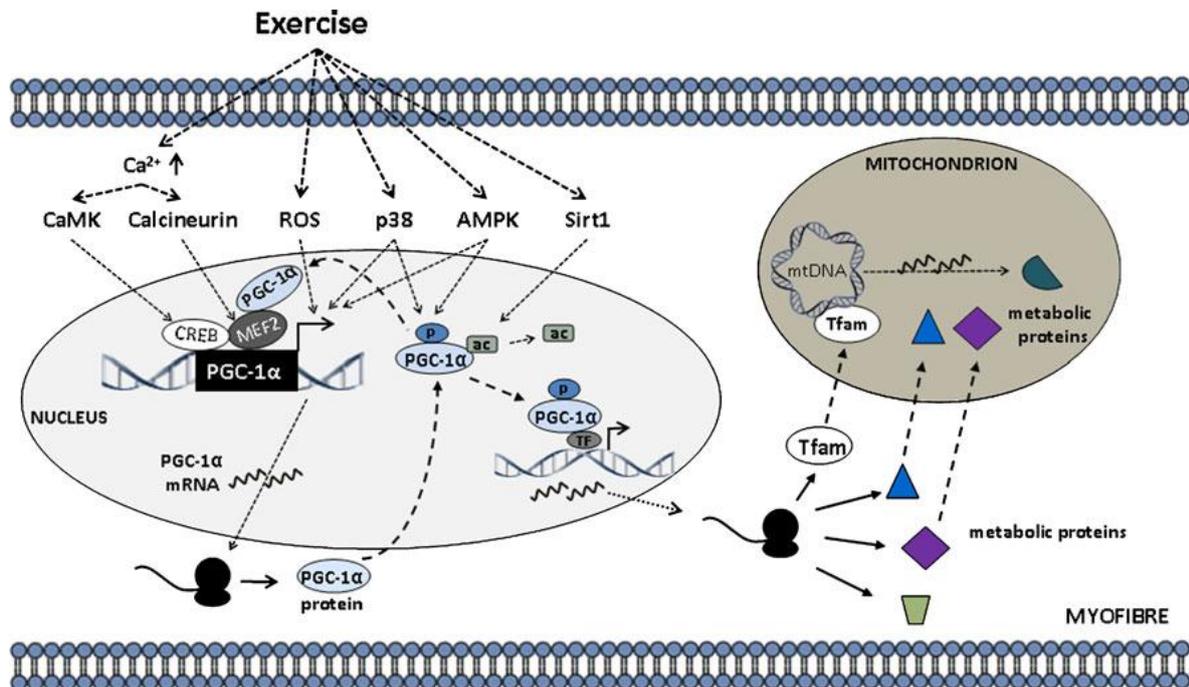


Figure 2.1: Diagram representing the exercise induced factors involved in the expression of PGC1- α .

peroxisome PGC1- α proliferator-activated receptor γ coactivators 1 α PGC1- α - adenosine monophosphate –activated protein kinase (AMPK), p38 mitogen-activated protein kinase (p38MAPK), calmodulin –dependent protein kinase (CaMK) reactive oxygen species (ROS) and sirtuin 1. (Sirt1), transcription factor (TF), mitochondria DNA (mtDNA). Transcription factor A mitochondria (TFAM) (Olesen *et al.*, 2010) with permission from Olesen

It has been shown that HIIT is a potent stimulus for the up regulation of the oxidative phenotype changes in skeletal muscle (Bartlett *et al.*, 2012; Gibala *et al.*, 2012; Little *et al.*, 2011; Gibala *et al.*, 2009a, 2000b). This is mediated through the signaling pathways that convert various intracellular and extracellular signals into changes in gene transcription (Hood, 2009). These adaptations are typical of endurance training (Baar *et al.*, 2002; Pilegaard *et al.*, 2003; Russell *et al.*, 2003). It has been shown that exercise intensity can influence PGC-1 α activation in human skeletal muscle (Bartlett, *et al.*, 2012; Little *et al.*, 2011; Egan *et al.*, 2010; Gibala, 2009). For instance, HIIT induces the activation of PGC-1 α by increasing its nuclear translocation, and increasing mRNA expression of several mitochondrial genes (Little *et al.*, 2010). These increases lead to an increase in mitochondrial content and oxidative capacity which is normally associated with endurance training (Calvo *et al.*, 2008; Watt *et al.*, 2004).

Pilegaard *et al.* (2003) reported an increase of 10 to 40 fold in PGC-1 α in physically active men after four weeks of endurance training (one legged knee extensor exercises) done five days a week. Russell *et al.* (2003) also reported an increase in PGC-1 α following six weeks of endurance training in type I, IIa and IIx fibers in men. The endurance training program consisted of running for 40min at 60% VO_{2max}. This increase in PGC-1 α is important in that it plays a key role in the up regulation of mitochondrial biogenesis.

More recent studies have demonstrated how HIIT can induce an acute increase in PGC-1 α mRNA. Gibala *et al.* (2009) reported significant increases in PGC-1 α and mRNA during recovery after HIIT. Similarly, Little *et al.* (2011) reported an increase in PGC-1 α of 66% three hours post recovery, with a return to baseline after 24 hours. This then confirms that HIIT has the ability to induce similar PGC-1 α adaptations which are usually associated with endurance training.

2.7. CAPILLARY DENSITY

An increase in capillary density has been reported as part of the adaptations invoked by endurance training. This is an important adaptation as capillary density determines the delivery of oxygen, blood glucose and triglycerides to working muscles, as well as the rate of removal of carbon dioxide, lactate and other metabolic by-products. The increase in capillary density can be observed in three ways, which include (i) an increase in capillary number, (ii) an increase in the number of capillaries per muscle fiber and (iii) and increase in the number of capillaries per square millimeter (Saltin and Gollinick, 1983).

The increase in the number of capillaries surrounding each muscle fiber is important in that it helps maintain conditions which are conducive for aerobic metabolism by increasing the efficiency of the muscles oxidative capacity. Daussin *et al.* (2008) compared the effect of eight weeks of endurance training and HIIT on muscle capillary density in sedentary women and men. They reported an increase of 3% and 2% for the endurance training and HIIT,

respectively. This showed that HIIT caused similar changes in capillary density to endurance training. It is important to note that energy expenditure was matched for the two groups in the latter study.

Aerobic metabolism recruits primarily type I muscle fibers which have a high oxidative capacity as they have more mitochondria, high capillary density and a higher resistance to fatigue (Willmore and Costill, 1994). Simoneau *et al.* (1985) investigated the effect of a 15 week HIIT programme in sedentary men and women. The HIIT protocol consisted of 15 to 90 seconds on a cycle ergometer at 60 to 90% of an individual's maximal work and recovery periods were long enough to ensure that the heart rate reduced to 120-130 bpm. They reported a shift in muscle fiber type from IIb (fast twitch) to type I fibers (slow twitch). Dawson *et al.* (1998) also reported a decrease in the proportion of type II fibers in fit males who trained for six weeks using six 40min sprints interspersed by 24 seconds of recovery. These results show that HIIT has the potential to alter muscle fiber composition towards greater endurance capacity.

Endurance training also increases cardiorespiratory fitness. This is important in that it lowers one's risk of developing cardiovascular disease and metabolic disorders. The assessment of cardiorespiratory fitness is measured using the VO_{2max} test. The cardiorespiratory adaptation to endurance training and HIIT will be discussed in Chapter 3.

2.8. COMPARISON OF HIIT AND ENDURANCE TRAINING

A series of studies have compared the effects of HIIT and endurance training (ET) and have reported similar and even superior adaptations with the HIIT program (Bartlett *et al.*, 2012; MacPherson *et al.*, 2011; Nybo *et al.*, 2010; McKay *et al.*, 2009; Gorostiaga *et al.*, 1991; Eddy *et al.*, 1977). Bartlett *et al.* (2012) reported similar increases in p38, p53, AMPK and PGC-1 following an exercise session of HIIT and ET in healthy recreationally active men. Hottenrott *et al.* (2012) also reported similar reductions in body mass, visceral fat and heart

rate following twelve weeks of ET and HIIT in endurance runners - this was despite the differences in training intensities. Nybo *et al.* (2010) also compared HIIT and ET over a 12 week period and they reported an 8 mmHg ($p < 0.05$) reduction in SBP after the intervention in both groups. However, reductions in HR and DBP were less after HIIT compared to endurance training. Despite the differences in total exercise time (20 min for HIIT and one hour for ET), HIIT induced a greater increase in VO_{2max} ($14 \pm 2\%$, $p < 0.05$) than ET ($7\% \pm 4\%$, $p < 0.05$).

The effect of HIIT has also been investigated in patient populations and has been shown to induce similar adaptations to ET. For example, Wisloff *et al.* (2007) studied heart failure patients during a 12 week HIIT and ET program. The HIIT protocol consisted of four 4-minute uphill walking intervals at 90 to 95% HR_{max} interspersed by three minute active recovery periods. In contrast the ET group walked for 47 minutes at 70 to 75%, three times a week. HIIT resulted in a greater increase in endothelial function, mitochondrial function and VO_{2peak} (46% vs 14%, $p = 0.001$) in the HIIT and ET groups, respectively.

All the above findings confirm that HIIT induces physiological adaptations equivalent to, or even more than traditional ET.

2.9. CONCLUSION

From the review of literature it was shown that HIIT elicits similar metabolic adaptations to that of endurance training. This was shown by changes in muscle oxidative capacity, exercise capacity, glycogen content and capillary density. However, optimum intensity at which HIIT can induce positive adaptations in lipid metabolism over a two week period still remains unknown. Although HIIT has been reported to induce feelings of nausea and dizziness exercise physiologists have come up with alternative modes of HIIT which have been shown to be well tolerated by individuals giving the potential for more people to adhere to training programmes. Last but not least is the fact that HIIT takes a shorter period of time compared to

endurance training and it can thus be used as a strategy for promoting health in both healthy and clinical populations as people usually cite a lack of time as barriers to participation.

Chapter 3

Role of Exercise in Managing Health Related Outcomes

3.1. INTRODUCTION

The “big four” primary risk factors for the pathogenesis of cardiovascular disease are smoking, hypertension, high blood lipid levels and physical inactivity (Foss and Keteyian, 1998; Wilmore and Costill, 1988). It is interesting to note that three of these factors are actually modifiable through exercise. High blood sugar levels and obesity are also risk factors for the development of diabetes, metabolic syndrome and cardiovascular disease. Of particular concern is the poor quality of life and high morbidity and mortality rates associated with cardiovascular disease and other chronic diseases such as diabetes, metabolic syndrome and cancers. In this review the health-related outcomes discussed are related to hypertension, blood lipids, physical activity, blood glucose and obesity. Despite the bleakness associated with chronic diseases, physical activity seems to provide a break through as studies have shown the beneficial effects of exercise in managing and in some cases reversing chronic diseases. Exercise can thus be used as a non-pharmacological option in the treatment of chronic diseases which would eventually lower the rate of mortality.

3.2. GLUCOSE METABOLISM AND EXERCISE EFFECTS

The regulation of glucose is a very sensitive homeostatic process; dysfunctions arising from a disruption in this system can lead to either hypoglycaemia (< 4.0 mM/l) or hyperglycaemia (≥ 11.1 mM/L). The American Diabetes Association Expert Committee on Diagnosis and Classification of Diabetes Mellitus recognise three categories for glucose levels, using fasting plasma glucose (ADA, 2013). The ADA, (2013) states that prior to the measurement of resting glucose, a fasting period of at least 8 hours should be adhered to. Chronic hyperglycemia may result in long term organ failure. The organs mostly affected include eyes, kidneys, nerves, blood vessels and the heart.

Table 3.2: Categories for the diagnosis of diabetes (ADA, 2013)

Classification	mMol.l⁻¹
Normal (Low Risk)	≤ 5.6
(Prediabetic) High Risk	5.6 – 6.9
Diabetic	≥ 7.0

Diabetes is a metabolic disease which is characterised by chronic hyperglycemia caused by a defect in insulin action, insulin secretion, or both. Diabetes is detrimental to health in that it may result in retinopathy, nephropathy, sexual dysfunction, hypertension and cardiovascular disease (ADA, 2013). It is important to note that there are two etiopathogenetic categories for diabetes. In the first category is type 1 diabetes which is caused by a deficiency in the amount of insulin secreted, while the second category, type 2 diabetes, is a result of insulin resistance and failure of β cells to secrete enough insulin to compensate for insulin resistance. Type 1 diabetes accounts for 5-10% of diabetes worldwide, while type 2 diabetes affects 90-95% of the diabetic population (ADA, 2013). Diabetes can be detected by measuring fasting plasma glucose or by the oral glucose load and by using the hemoglobin A1c.

The glucostatic centre in the hypothalamus plays a major role in controlling the release of hormones in the body which maintain glucose homeostasis by activating key transporter molecules (GLUT4) and enzymes involved in energy synthesis and resynthesis processes. The glucostatic centre has a high metabolic rate thus it is very sensitive and can detect any changes in blood glucose levels. This enables it to invoke feelings of satiety, soon after a meal and hunger, several hours after a meal.

Elevated blood sugar levels after the consumption of a meal prompt the release of insulin by the β cells of the Islets of Langerhans in the pancreas (Petersen and Shulman, 2002; Saltiel, 2001). Insulin promotes the uptake of glucose into the skeletal muscle where it is either used for ATP production or converted and stored as glycogen.

The target tissues for insulin are adipose tissue, liver and muscle. In adipose tissue glucose is converted into lipids and stored in adipocytes. Insulin also inhibits lipolysis in adipocyte by deactivating the enzyme lipase (Saltiel, 2001). Insulin has an antagonistic effect in its other target organs which is the liver. Here it attenuates the production of glucose by the processes of glycogenolysis and gluconeogenesis.

The insulin released into the blood during periods of elevated blood levels binds to the insulin receptor (outer α subunit) which phosphorylates the tyrosine residues (of the inner β subunit) which also phosphorylates other intracellular proteins (Brooks *et al.*, 1995). The intracellular proteins initiate a cascade of events that end up stimulating the translocation of glucose transporters (GLUT 4) from intracellular compartments to the cell surface, thereby increasing the rate of glucose uptake by 10 to 40 times (Bouskila *et al.*, 2010).

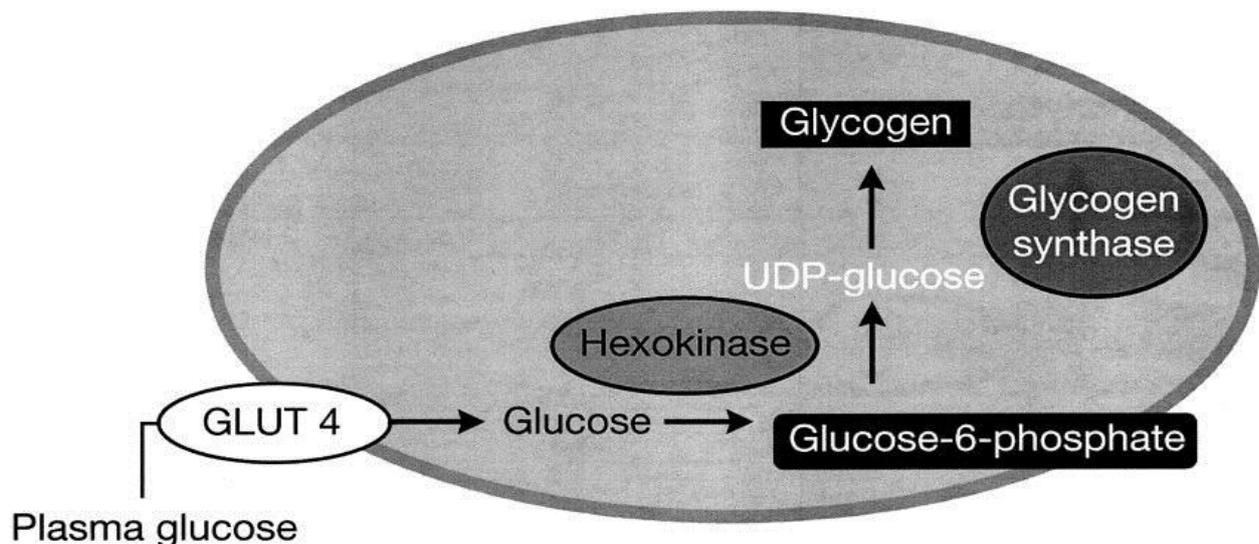


Figure: 3.1 the pathway of muscle glycogen synthesis. GLUT 4 = glucose transporter 4; UDP = uridine 5-diphosphate adapted from Petersen and Shulman (2006).

Upon entering the cell glucose is phosphorylated by the enzyme hexokinase to glucose-6-phosphate (G6P) as shown in Figure 3.1. G6P is either used in ATP synthesis or in the synthesis of glycogen. G6P is converted to Uridine 5' phosphate which is then polymerized into glycogen by the enzyme glycogen synthetase which is activated by insulin through

covalent phosphorylation and allosteric regulation (Bouskila *et al.*, 2010; Petersen and Shulman, 2006; Roach *et al.*, 2002; Friedman and Lerner, 1963).

During a period of starvation the hormone glucagon is released from the α -cells of the pancreas and it triggers the conversion of the stored glycogen to glucose and is released into the circulation. Glucagon, an antagonistic hormone to insulin, is also released when blood glucose levels are below normal (< 4.0 mM/L); its effect is to stimulate the release of glucose from the glycogen stores into the bloodstream, thus helping to maintain glucose levels (Saltiel *et al.*, 2001).

3.2.1. Insulin Resistance

Due to prolonged periods of elevated glucose levels, the peripheral target tissue insulin receptors (IR) fail to respond to the hormone insulin, thus attenuating glycogen synthesis (Bouskila, *et al.*, 2010), resulting in insulin resistance. Saltiel (2001) specified a number of factors that have been implicated in the pathogenesis of insulin resistance. These include obesity, aging and genetic predisposition. Insulin resistance leads to an increase in fatty acid release through lipolysis, and insulin also fails to exert its antilipolytic effects on adipocytes. To compensate for this the β cells of the pancreas increase the basal and postprandial insulin secretion resulting in an increased glucose uptake by skeletal muscles as well as attenuating the release of free fatty acids (Petersen and Shulman, 2006).

However, when the β cells fail to compensate (as a result of genetics, aging or obesity) it may lead to glucose intolerance and diabetes (Figure 3.2). Saltiel (2001) claims that about 5 to 10% of people diagnosed with glucose intolerance become diabetic.



Figure 3.2: Metabolic Staging of Type 2 Diabetes adapted from Saltiel A.R (2001)

3.2.2. Effect of exercise on glucose levels, glucose metabolism and insulin resistance

During exercise there is a drop in blood sugar levels which is detected by the glucostatic centre; this activates the release of norepinephrine from the sympathetic nerves and the release of epinephrine from the adrenal medulla (Talanian *et al.*, 2007). These amine hormones activate the production of ATP in the muscles by attaching to receptors in the plasma membrane to start the production of cyclic adenosine monophosphate (cyclic AMP). The cyclic AMP activates the protein kinase which activates other active enzymes to breakdown glycogen to glucose which can be used in the aerobic pathway for production of ATP. They also trigger lipolysis in the adipose tissue, glucogenolysis in the liver and use of fatty acids for energy production thus sparing the glucose for the heart and the brain. Growth hormone is also released to stimulate the mobilization of fatty acids and glycerol by the adipose tissue.

Exercise training enhances insulin action and glycemic control; these changes are not dependent on changes in body composition and occur despite no reductions in body weight

(Richards *et al.*, 2010; Babraj *et al.*, 2009). An increase in insulin sensitivity has been shown to be proportional to increases in GLUT4; which then enhances the body's glucose uptake and assists glucose homeostasis (Durstine *et al.*, 2013; Kessler *et al.*, 2012). This maintenance of glucose homeostasis is important in both prediabetic and diabetic individuals. Moderate to vigorous intensities can both induce changes in insulin action and glycemic control, with higher intensities producing more favourable changes in insulin sensitivity (Babraj *et al.*, 2009). In a review Boutcher, (2011) mentioned that HIIT studies have reported 23 to 58% increases in insulin sensitivity. Studies done on healthy subjects have reported an increase in insulin sensitivity that ranges from 23% to 33% after HIIT.

Babraj *et al.* (2009) revealed the efficacy of HIIT in their study conducted in sedentary and recreationally active men who took part in a two week HIIT intervention program where they used the Wingate protocol (4-6, x30s all out cycling efforts). They reported a 12%, 37% and 26% reduction in the area under the curve for glucose, insulin and non-esterified fatty acids (NEFA) concentrations, respectively. Insulin sensitivity also improved by 23% while time trial performance increased by 6%. A reduction in NEFA is important as NEFA regulates insulin sensitivity. Similarly, Richards and colleagues (2010) observed an increase in insulin sensitivity from 6.3 ± 0.6 to 8.0 ± 0.8 mg/kg/min. in recreationally active adults after two weeks of HIIT (Wingate protocol) in which six sessions were done.

Nybo *et al.* (2010) reported a significant increase in (9%) glucose levels after a twelve week HIIT intervention; which was similar to the (9%) continuous training group reduction in healthy adults (see Table 3.4). This then shows the efficacy of HIIT as an intervention for promoting health. Burgomaster *et al.* (2007) illustrated the potency of a very short HIIT intervention (three sessions of six min exercise each) where they reported a significant 20% increase in GLUT 4 after one week, which remained significantly higher than pre-training values after six weeks of detraining. This adaptation could help in the uptake of glucose and

thus help prevent the development of insulin resistance. This finding is consistent with other studies utilizing various HIIT interventions (Gibala *et al.*, 2009; Perry *et al.*, 2008). From a review of the literature, Boutcher, (2011) proposed that the increase in insulin sensitivity observed following HIIT can be attributed to the increase in metabolic oxidative capacities of skeletal muscles, and an increase in GLUT4 transporters.

3.3. CHOLESTEROL AND EXERCISE EFFECTS

A high level of cholesterol (≥ 5.2 mM/L) in the body is a primary health risk indicator in the development of cardiovascular disease (Dishman *et al.*, 2004). Nevertheless, Manson and colleagues (1992) suggested that a reduction of total cholesterol levels of one percent results in a two to three percent reduction in the risk of coronary heart disease (CHD). It is therefore important to have an understanding of cholesterol metabolism and its role in the pathogenesis of cardiovascular diseases and how physical activity can lower people's health risk profiles.

Table 3.3: Cholesterol classifications of risk categories (ACSM, 2006)

Classification	mM/L
Low Risk	≤ 4.2
Moderate Risk	4.3 – 5.1
High Risk	5.2

Cholesterol is a sterol with a complex ring structure and is synthesised from acetate by the liver, adrenal cortex and gonads. Cholesterol can either be obtained exogenously from the diet (animal foods) or derived *in vivo*, from the liver. In instances where cholesterol is obtained from the diet it is taken up into the lymphatic vessels and is transported by chylomicrons to the liver where it inhibits its endogenous synthesis.

Cholesterol plays a major role in the body and the body synthesizes enough to enable it to fulfil its responsibilities. Cholesterol is used in the production of bile, and is a precursor for many vitamins, sex hormones and adrenal gland hormones (McAdele *et al.*, 2010). Cholesterol

also forms a crucial component of cell membranes as the phospholipid layer (McAdle *et al.*, 2010). Cholesterol is packaged in the liver into lipoproteins from where it is transported to various tissues. Different types of lipoproteins exist, namely low density lipoproteins (LDL), high density lipoproteins (HDL) and very high density lipoprotein (VHDL) (Brooks *et al.*, 1995).

There exists an inverse relationship between the density of lipoproteins and the risk of coronary heart disease as HDLs have been reported to have a protective role against the pathogenesis of CHD (Durstine *et al.*, 2013). HDL comprises 50% protein, 5% triglycerides, 25% phospholipids and 20% cholesterol and they are often referred to as the “good cholesterol”. HDL and VHDL play a vital role in the transportation of cholesterol from the blood and artery walls to the liver for excretion or utilization in the production of bile acids (Vella *et al.*, 2001). Additionally, HDLs bind to the LDL receptor sites, thus intercepting the uptake of LDLs into tissues (Kravitz and Heyward, 1994). HDLs and VHDLs have a scavenging role as they catalyse VLDLs and chylomicrons via the enzyme lipoprotein lipase (Brooks *et al.*, 1995). Moreover, HDLs also have a cardio protective role as they contain the enzyme lecithin cholesterol acid transferase (LCAT). LCAT reverses the cholesterol transfer system by gathering all free cholesterol (from cell renewal or death) and returning it to the liver where it is excreted into the bile (Foss and Keteyian, 1998; Brooks *et al.*, 1995). This reverse cholesterol transport system is vital in that it impedes the pathogenesis of atherosclerosis.

LDLs are responsible for the transportation of cholesterol to tissues for use in hormone and cell membrane synthesis and are commonly referred to as the bad cholesterol. They consist of 50% cholesterol, 20% phospholipids, 20% protein and 10% triglycerides (Brooks *et al.*, 1995). LDLs have a high affinity for the arterial wall leading to high fat deposits in the arterial wall. This is prompted by the fact that exposure to endothelial cells of LDLs may

result in LDLs being oxidised which mediates the accumulation of esters in the macrophages (Foss and Keteyian, 1998). An excess LDL becomes hazardous in that it may cause hyperlipidemia which may lead to atherosclerosis (Brook *et al.*, 1995). Atherosclerosis is a result of cholesterol and calcium deposition in the inner walls of the artery, where they form calcified cholesterol plaques (McAuley *et al.*, 2010; Heyward, 2002). These plaques can eventually lead to the development of chronic diseases such as hypertension, ischemia, coronary thrombosis, heart attack and strokes (Dishman *et al.*, 2004; Heyward, 2002).

On the one hand endurance training induces an increase in the amounts of HDLs in the blood and the maximal activities of the enzymes lipoprotein lipase and LCAT, thus enhancing the excretion of cholesterol from the body (Boreham *et al.*, 2000; Brooks *et al.*, 1995). The elimination of cholesterol may also be associated with a decline in one's risk for developing coronary heart disease (Brooks *et al.*, 1995). On the other hand, endurance training reduces LDLs and triglycerides by increasing insulin receptor activity in the body (Brooks *et al.*, 1995). Moreover endurance training increases the HDL-C to LDL-C ratio. The increase in HDL-C is also partly responsible for the lowering of LDL-C as it plays its role of scavenging on LDL-C.

Weintraub *et al.* (1989) shows overwhelmingly that physical exercise, done within a period of seven weeks on a treadmill for 30 minutes per session, reduced the fasting and postprandial lipoprotein levels. This was despite the fact that the participants did not lose weight, thus demonstrating the potential for exercise to further improve health. They also reported a 16% increase in the maximal activities of lipoprotein lipase as well as decreasing the chylomicrons by 37%. These results showed that exercise increases endogenous and exogenous triglyceride catabolism (Weintraub *et al.*, 1989). Laaksonen *et al.* (2000) conducted a 12 to 16 week endurance training study in type 1 diabetic men. The endurance program consisted of 20 to 60 minutes of continuous running at 50 to 80% VO_{2peak} three to five times a week. They

observed a significant decrease in total cholesterol and HDLs. They also reported an increase in HDLs and in the HDL/HDL ratio. The favourable changes observed in the lipid profile were despite the fact that they did not report any changes in body weight. This then suggests that changes in lipid profiles accrued during endurance training are independent of changes in body mass. These changes are of importance as Manson *et al.* (1992) mentioned that a 1% decline in cholesterol levels reduces the risk of coronary heart disease by 2-3%.

Tjonna *et al.* (2009) compared the effect of three months of HIIT (AIT) and endurance training in overweight adolescents (protocol in Table 3.2). They reported a 0.11mM/L ($p < 0.05$) increase in HDL-C in the AIT group, while in the endurance training group there was only a 0.09 mM/L ($p > 0.05$) decrease in the HDL-C (Tjonna *et al.*, 2009). The results from this study show that HIIT is superior to endurance training in improving lipid profiles as it increases the HDL-C levels.

3.4. BLOOD PRESSURE

Zanabria *et al.* (2003) defines hypertension as “a blood pressure equal to or greater than 140/90mmHg.” The blood pressure classification used in this study is in accordance with the ACSM (2006) guidelines as shown in Table 3.3. The 2013 WHO report estimates that in South Africa 34.9% of women and 39.9% of men have hypertension (WHO, 2013). Hypertension has been shown to increase the afterload of the heart which imposes more stress on the heart as it has to pump harder to deliver the same amount of blood to the periphery and lungs. This chronic stress can lead to myocardial hypertrophy resulting in an increase in myocardial oxygen uptake.

Table 3.4: Blood Pressure classification of risk categories (ACSM, 2006)

Classification	Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)
Low Risk	<120	<80
Moderate Risk	120 – 139	80-89
High Risk	≥140	≥90

Hypertension is also known as the “Silent Killer”. This is because it does not have any ostensible symptoms during the early stages. It mediates the development of atherosclerosis which eventually leads to myocardial infarction, thrombosis, ischemia and stroke. Hypertension is linked to atherosclerosis in that it increases shear force, torsion and lateral wall pressure which causes damage to the endothelial cells lining the arteries. This damage in the endothelial cells results in the migration of blood platelets, white blood cells, monocytes and macrophages which forms a fibrous plaque, which narrows the lumen of the arteries. Atherosclerosis may lead to the formation of a clot (thrombosis) if the fibrous plaque ruptures. The clot /thrombi can subsequently lead to angina and myocardial infarction (heart attack). Myocardial infarction occurs when the plaque / thrombi block the coronary arteries which cuts off the tissue’s supply of oxygen leading to death of heart cells. Chobanian *et al.* (2003) reported that a reduction as little as 2mmHg can significantly decrease the incidence of stroke by 6%, while coronary heart disease risk is reduced by 4%.

Hypertension commonly co-exists in obese individuals and this may be attributed to three pathophysiological mechanisms. Firstly, is the association of obesity with increased sympathetic activation of the hypertrophied fat cells which release molecules which have vasopressor and anti-natriuretic effects as they stimulate angiotensin and aldosterone formation. Secondly, is the identification of local renin-angiotensinogen which may act independently than the known plasma renin–angiotensinogen system. Lastly is the fact that that obesity results in the production of pro-inflammatory cytokines which could influence the

endothelial cells and vascular modulation (Rheume, 2009). Individuals with high visceral adipose tissue have been shown to have a higher incidence of developing hypertension in future (Yanai *et al.*, 2008; Chuang *et al.*, 2006; Poirier *et al.*, 2005).

To clarify this issue, Rheume *et al.* (2009) conducted a study on 407 men and women to investigate whether the relationship between cardiovascular fitness and blood pressure is related to visceral adiposity levels. The findings from their study showed that visceral adipose tissue (VAT) has a strong correlation with blood pressure with high levels of visceral adipose tissue being associated with higher systolic and diastolic blood pressure (Rheume *et al.*, 2009). Individuals with lower visceral adipose tissue had lower systolic and diastolic blood pressure and consequently lower cardiovascular risk. It is also important to note that the strong correlation between VAT and blood pressure is not dependent on either cardiorespiratory fitness or BMI (Rheume *et al.*, 2009). These authors actually suggested that the lower SBP and DBP observed in fit individuals are related to the fact that these individuals have a healthy body composition and low levels of VAT (Rheume *et al.*, 2009). The strong correlation between VAT and blood pressure has serious implications in the prevention, treatment and management of hypertension. It is thus recommended that VAT becomes a clinical target in the treatment of blood pressure (Rheume *et al.*, 2009). This then provides a link with exercise training as it has been shown to reduce VAT (Rheume *et al.*, 2009).

Endurance training has favourable effects on blood pressure as it has been demonstrated to lower both systolic and diastolic blood pressure. The mechanisms involved in this regulation include a decline in autonomic nervous activity as shown by lower plasma renin and noradrenaline activity resulting in a reduction in systemic vascular resistance (Fagard, 2006).

Stassen *et al.* (1989) reported that aerobic exercise can significantly reduce systolic and diastolic blood pressure coupled by a reduction in weight. For every kilogram lost by

participants, their SBP and DBP were also reduced by 1.6 and 1.3 mmHg, respectively. This was further confirmed by Hagberg, (1990) when he reported a 10 to 12 mmHg decrease in both SBP and DBP for every kilogram lost in body mass with aerobic exercise. Endurance training reduces BP by decreasing total peripheral resistance (McArdle *et al.*, 2010). The reduction in total peripheral resistance is caused by the vasodilation of the muscle which is mediated by local factors in the muscle resulting in an enhanced blood flow. Endurance training increases nitric oxide production (in endothelial cells) which facilitates the dilation of blood vessels and consequently decreasing vascular resistance resulting in an enhanced blood flow (McArdle *et al.*, 2010.)

In a meta-analysis reviewing 72 trials and 105 study groups Fagard, (2006) reported a decrease of 3.0/2.4 mmHg and 3.3/3.5mmHg in resting and daytime ambulatory BP, respectively, after endurance training. The reduction in blood pressure accrued during endurance training is associated with a decrease in cardiovascular risk factors demonstrating the viability of exercise as a non-pharmacological treatment for hypertension (Millen *et al.*, 2013; Kokkinos, 2012; Fagard, 2006).

It is also interesting to note that short-term HIIT studies (Table 3.2.1) have surprisingly reported a 4.7% reduction in SBP in overweight and obese men after 24 hours post-training (Whyte *et al.*, 2010). However, this was not sustained at 72 hours post exercise. These authors attributed the reduction in SBP to an increase in nitric oxide-mediated vasodilatation and a reduction in sympathetic nervous activity (Whyte *et al.*, 2010). Furthermore, studies comparing the effects of HIIT and endurance training on BP have reported that HIIT elicits similar and even superior adaptations compared to traditional endurance training (Guimeres *et al.*, 2010; Ciolac *et al.*, 2010; Nybo *et al.*, 2010; Tjonna *et al.*, 2009; Rakobowchuck *et al.*, 2008; Nemoto *et al.*, 2007).

It is also important to note that exercise does not always reduce BP regardless of exercise intensity. For example, Gormley *et al.* (2008) showed no significant declines in BP in healthy men and women who exercised at 50% VO_2R (difference between resting and maximal VO_2), 75% VO_2R , or 95% VO_2R . They attributed this to the fact that their subjects had low BP at baseline (108/65mmHg). This then indicates that initial levels of BP also determine the extent to which BP adaptations occur following exercise.

3.5. MAXIMAL EXERCISE

The $\text{VO}_{2\text{max}}$ test is considered the gold standard for testing cardiorespiratory fitness. Kreamer *et al.* (2012) defines $\text{VO}_{2\text{max}}$ as “the rate of oxygen consumption attainable during maximal or exhaustive exercise.” Hence $\text{VO}_{2\text{max}}$ evaluates the maximum energy released by aerobic processes and the rate of oxygen consumption by the body because it measures the functionality of both the oxygen transport system (a- vO_2 : oxygen extraction) and the circulatory system (cardiac output) (Astrand *et al.*, 2003; Reilly *et al.*, 1993). It is used as an indicator of cardiorespiratory endurance, aerobic fitness and as a measure of the effectiveness of training programs (Wilmore and Costill, 1988).

An inverse relationship exists between $\text{VO}_{2\text{max}}$ and risk of all-cause mortality. In a sense a high $\text{VO}_{2\text{max}}$ reduces or minimizes an individual’s risk of cardiovascular diseases (Trilk *et al.*, 2011; Blair *et al.*, 1989).

A series of studies have demonstrated the effectiveness of aerobic training in improving cardiorespiratory fitness in both healthy and diseased populations as shown in Table 3.1 (Trilk *et al.*, 2011; Nybo *et al.*, 2010; Burgomaster *et al.*, 2008; 2007, 2005; Gibala 2008; Bailey, 2004; Laursen and Jenkins, 2002; McDougall, 1998). Endurance training has been shown to increase $\text{VO}_{2\text{max}}$ and this increase was accompanied by increases in stroke volume and lower heart rate values during exercise at submaximal intensities.

Consequently, HIIT has also been shown to induce an increase in $\text{VO}_{2\text{max}}$ in healthy and clinical populations. The improvements in $\text{VO}_{2\text{max}}$ by HIIT have been equal and even superior to that of continuous endurance training. The improvements in $\text{VO}_{2\text{max}}$ of 4.2% to 17.2% observed following two to eight weeks of HIIT can be attributed to two factors. Firstly an increase in $\text{VO}_{2\text{max}}$ can be attributed to the increase in oxygen availability which occurs as a consequence of an increased cardiac output (Sloth *et al.*, 2013; Helgerud, *et al.*, 2006). This increase in cardiac output is mediated by an increase in stroke volume which is induced by an increase in cardiac contractility and plasma volume (Sloth *et al.*, 2013; Boyd *et al.*, 2013; Trilk *et al.*, 2011; Daussin *et al.*, 2008; Helgerud, *et al.*, 2006; Warburton *et al.*, 2005, Nagashima *et al.*, 1999; 2000; Green *et al.*, 1984). Secondly, the improvement in $\text{VO}_{2\text{max}}$ accrued through HIIT can be attributed to peripheral changes which enhance the muscle's oxidative capacity and oxygen extraction (Sloth *et al.*, 2013).

Table: 3.5 Changes in VO_{2max} and health parameters.

Study : Subject characteristics	Sam ple size	Study Design	Duration & Intensity,	Adaptations to Training
Tjonna <i>et al.</i>, 2013 Overweight healthy men	26	Treadmill – 10wks. 1-AIT=11 4-AIT = 13	1-AIT; 1x4 min at 90% HRmax 4-AIT - 4x4 min at 90%HRmax, 3min active recovery	1-AIT: 10% increase in VO _{2max} , 1% reduction in BMI& body weight, 7.1% and 7.7% decrease in SBP and DBP, 6% reduction in fasting glucose 4-AIT:13% increase in VO _{2max} , 2% reduction in BMI and body weight, 2.6% and 6.1 decrease in SBP and DBP, 5% reduction in fasting glucose
Astorino <i>et al.</i>, 2012 Recreationally men & women	20	Cycle ergometer 2 wks. HIIT= 11 CON = 9	HIIT; 4-6, 30s all out sprints, 4 min active recovery, 3 d/wk. CON did not exercise	4.7 % increase in VO _{2max} No change in BP And HR
Bayati <i>et al.</i>, 2011 Active young males	24	Cycle ergometer 4 wks. G1 G2	G1:3-5 x30 s all out sprints, 4 min rest G2: 6-10 x30 at 125% Pmax sprints, 2 min rest 3 d/wk G3: No training	G1:9.6% increase in VO _{2max} , 12.8% & 10.3%increase in power at VO _{2max} & mean power G2: 9.7% increase in VO _{2max} , 12.8% increase in power at VO _{2max} , no increases in mean poweroutput.
Metcalfe <i>et al.</i>, 2011 Sedentary males and females	29	Cycle ergometer 6 wks. Total exercise time 30mins	WK1: 1x10s al out sprints WK2-3: 2x15s all out sprints WK3-6: 2x 20s all out sprint. 3d/wk	15% & 12% increase in VO _{2max} in men and women. 28% increase in insulin sensitivity in men and none in women.
Trilk <i>et al.</i>, 2011 Overweight /obese women	28 F only	Cycle ergometer 4 wks. SIT= 14 CON=14	SIT; 4-7 X 30s all out sprints, 4min active rest, 3d/wk. CON; no training	12% increase in VO _{2max} 11.4% increase in SV 4% increase in plasma volume -8.1% decrease in resting HR No significant change in HRmax
Hood <i>et al.</i>, 2011 Overweight	7	Cycle ergometer 2wks.	HIIT; 10X60s at 80-95%, 60s active rest, 3d/wk.	35% increase in insulin sensitivity at 72h
MacPherson <i>et al.</i>, 2011 Healthy & recreationally active	20 F = 8 M= 12	Treadmill 6 wks. SIT = 10 ET = 10	SIT :4-6 X 30s all out runs, 4min rest ET: 30- 60 min/ at 65 % VO _{2max}	SIT : 11.5% increase in VO _{2max} , 12.4% decrease FM, 1% increase in LM, 4.6 % increase TTP ET : 12.5% increase in VO _{2max} , 5.8% decrease FM, 1% increase in LM, 5.9% increase in TTP, 9.5% increase in Q max

Whyte et al., 2010 Overweight /obese	10 M	Cycle ergometer 2 wks.	4-6 X30s all out sprints, 4.5 active rest, 3d/ wk.	9.5%increase in VO _{2max} 5% & 1.1% decrease in SBP &WC respectively 25%, 15% decrease in fasting glucose &AUC insulin respectively, 23% increase in insulin sensitivity.
Nybo et al., 2010	28	Treadmill 12 wks. AIT = 8 CME = 9 CON = 11	AIT ; 5x2 min at 95% HR _{max} , 3min active recovery CME 47 min continuous run at 60-70% HR _{max}	AIT ; 14.2% increase in VO _{2max} , 6% decrease in SBP, 9% decrease in fasting glucose CME ; 7% increase in VO _{2max} , 6% decrease in SBP , 9% decrease in fasting glucose. No changes in lipid
Ciolac et al., 2010 Normotensive women with a family history of hypertension and normal BMI	34 Fem ales	Treadmill 16wks. AIT= 11 CME = 11 CON = 12	AIT; 13x1 min at 80-90% VO _{2max} , 2 min active recovery CME 40min run at 60- 70% , 3d/wk	AIT ; 15.8% increase in in VO _{2max} , 1.8% and 2.8 % decrease in SBP and DBP respectively CME ; 8% increase in VO _{2max} , 2.4% and 2.8% in SBP and DBP respectively
Moholdt et al.,2009 Overweight adults, post coronary artery bypass graft surgery	59	Treadmill 4wks AIT = 28 CME = 31.	AIT; 4X4min at 90%HRmax, 3min active CME; 46 min continuous run at 70% HRmax	AIT ;12.2% increase in VO _{2max} CME ; 8.8%increase in VO _{2max} No changes in fasting glucose, HDL-C, LDL-C or TG in both AIT and CME
Wallman et al., 2009 Obese adults	21	Cycle ergometer 8 wks. AIT =7: CME = 6: CON= 8	AIT; 10x1min at 90% VO _{2max} , 2 min active rest, 4d/wk. CME; 30min continuous cycling at 50% VO _{2max}	AIT : 24% increase in VO _{2max} CME ; 9% increase in VO _{2max}
Munk et al., 2009 Overweight adults with coronary artery stents	40	Treadmill 6months AIT = 20 CON = 20	AIT; 4X4 min at 95% HRmax, 3 min active recovery 3d/wk.	AIT : 16.8% increase in VO _{2max} , 2.2% increase in BMI CON ; 7.8% increase in VO _{2max} , 1.8% increase in BMI. No change in BP in both groups
Tjonna et al., 2009 Obese adolescents.	42	Treadmill 12wks. AIT= 20 CON =22	AIT:4x4 min at 90% HRmax with 3min rest at 70%HRmax, 2d/wk CON:followed physician orders on exercise and had group meetings3 times a month	AIT ; 11% increase in VO _{2max} , 2.1% ,2.1%, 1.3%,7%, 8% 8% decrease in BMI, BF%, SBP, DBP and MAP respectively, 9.7% increase in HDL-C, 6% & 29% decrease in fasting glucose and insulin, 12% &27% decrease in glucose and insulin (2hrs post glucose load), 24% increase in insulin sensitivity. CME ; no change in VO _{2max} , HDL-C, DBP, MAP, fasting glucose and WC
Tjonna et al., 2008	28	Treadmill	AIT; 4x4 min at	AIT ; 35 % increase in VO _{2max} .

Overweight adults with metabolic syndrome		16wks. AIT =11 CME =8 CON = 9	95% HR _{max} , 3 min active recovery 3d/wk. CME; 47 min continuous run at 70% HR _{max} 3d/wk. CON; followed physician advice	3%, 2.3% , 4.7% decrease in BM , BMI and WC respectively. SBP, DBP, MAP decreased by 6.2%, 6.3% and 5.4% respectively. HDL-C increased by 22%. Fasting glucose decreased by 4.3 %. 15% increase in Insulin sensitivity. CME ; 16 % increase in VO _{2max} , 4%, 4.1%, 5.7% decrease in BM, BMI and WC respectively. SBP, DBP, MAP decreased by 7.6%, 0% and 6.9% respectively. HDL-C increased by 22%. Fasting glucose decreased by 4.3 %. 15% increase in Insulin sensitivity.
Tsekouras <i>et al.</i>, 2008 Borderline overweight	15	Treadmill 8 wks. AIT = 7: CON = 8	AIT; 4x4 min at 90% VO _{2max} , 4min active recovery, 3d/wk.	AIT ; 18 % increase in VO _{2max} , 28% decrease in VLDL-C- TG
Burgomaster <i>et al.</i>, 2008 Healthy recreational active men	20	Cycle ergometer 6 wks SIT : CME	SIT; 4-6 X 30 S all out efforts, 4,5 min rest , 3d/wk. CME; 40-60 min, 5d/wk.	SIT ; 7.3% VO _{2max} , CME ; 12.5% VO _{2max} No change in in body weight
Schjerve <i>et al.</i>, 2008 Obese middle aged adults	27	Treadmill 12 wks. AIT =14: CME=13	AIT ; 4x4 min at 85-95% HR _{max} , 3min active recovery CME; 47 min continuous run at 60-70%HR _{max}	AIT ; 33% increase in VO _{2max} 2% ,1.6 % , , 2.2% 7%increase in BM, BMI, BF% and DBP respectively, no change in WHR CME ; 16% increase in VO _{2max} , 3%, 3%, 2.5% and 9% decrease in BM, BMI, BF% and DBP respectively
Wisloff <i>et al.</i>, 2007 Older adults with post infarction heart failure	27	Treadmill 12 wks. AIT = 9 : CME = 9: CON = 9	AIT; 4x4min at 90-95% HR _{max} , 3min active recovery, 2d/wk. CME; 47 min continuous walking at 70- 75% HR _{max} 2d/wk. CON: followed physician advice	AIT : 46% increase in VO _{2max} CME : 14% increase in VO _{2max}
Helgerud <i>et al.</i>,2006 Recreational endurance male runners	40	Treadmill 8 wks LSD= 10 LT= 10 15/15=10 4x4= 10	LSD:45min continuous run at 70% HR _{max} LT:24.25min continuous run at 85%HR _{max} 15/15: 47 x 15s at 90-95% HR _{max} , 15s rest at 70% HR _{max} 4x4: 4x4 min run at	LSD: no increase in VO _{2max} LT: no increase in VO _{2max} 15/15:5.5% increase in VO _{2max} 4x4:7.2% increase in VO _{2max}

			90-95% HR _{max} with 3min rest at 70% HR _{max}	
Warburton <i>et al.</i>, 2005 Overweight adults with CAD treated with a bypass or angioplasty.	14	Treadmill 16 wks. AIT = 7 CME = 7	AIT; 8x2min at 90% VO _{2res} , 2min active recovery, 2d/ wk. CME; 30 min of continuous running at 65% VO _{2res} , 2d/ wk.	AIT ; 31.8% increase in VO ₂ at anaerobic threshold, 3.4% increase in BM CME ; 9.5% increase in VO ₂ at anaerobic, 4.7% increase in BM
Rognmo <i>et al.</i>, 2004 Overweight adult with CAD	17	Treadmill 10wks. AIT = 8 CME = 9	AIT; 4x4 min at 80- 90% VO _{2max} , 3min active recovery, 3d/wk. CME; 41 min continuous run at 50- 60% VO _{2max} , 3d/wk.	AIT ; 17.9% increase in VO _{2max} CME ; 7.9% increase in VO _{2max}

VO_{2max} AIT - aerobic interval training CON – control group, HR- heart rate , HR_{max} – maximum heart rate, VO_{2R}- VO₂ Reserve, TTP- Time trial performance; Q_{max} – cardiac output, MI –moderate intensity group, VI- vigorous intensity group, NMI- near maximal intensity group, BM – body mass, CME, VO_{2res}, BMI- body mass index, CAD- coronary artery disease, VLDL-C- TG, HDL-C, LDL-C ,TG- triglycerides SBP- systolic blood pressure, DBP diastolic blood pressure, MAP- mean arterial pressure, VO₂, WC- waist circumference, WHR- waist to hip ratio, BF%-percentage body fat, LSD- long slow distance running, LT- lactate threshold, P_{max}- maximum power.

From the above table it is apparent that an increase in VO_{2max} after HIIT is not a universal finding. Those researchers who reported positive results, observed changes between 4.2% and 46% in VO_{2max}. Astorino *et al.* (2012) suggested that any changes in VO_{2max} may be dependent on the initial fitness levels of the participants, and since subjects in all studies varied from sedentary to healthy active, it is quite possible that this may be the main reason for the inconsistent results. Furthermore, the duration of the training intervention, as well as the duration of exercise intervals per session may be additional factors that may cause disparate results. HIIT has also been demonstrated to cause more superior changes in VO_{2max} in clinical populations which include patients with coronary artery disease, heart failure, hypertension and overweightness or obesity. Nybo *et al.* (2010) reported a 14% ± 2% increase in VO_{2max} after a 12 week HIIT intervention; this was superior to the CT group which reported a 7 % increase in VO_{2max}.

Far beyond the simple paradigm of $\text{VO}_{2\text{max}}$ being increased by volume of work as stated by Pollock *et al.* (1998), Gormeley and colleagues (2008) indicated that there seems to be a direct relationship between an increase in $\text{VO}_{2\text{max}}$ and high intensity exercise of up to 95% of $\text{VO}_{2\text{max}}$. Thus it seems that the improvement in $\text{VO}_{2\text{max}}$ seems to lie on an intensity continuum with higher intensities inducing greater improvements in $\text{VO}_{2\text{max}}$ than moderate intensities. This has been demonstrated by exercise studies comparing continuous training and HIIT training, where the total volume of exercise was matched for both training regimes (Gormley *et al.*, 2008; Daussin *et al.*, 2008, 2007; Warburton *et al.*, 2005), yet exercise intensity per session were higher during HIIT than during continuous training.

3.6. OBESITY , OVERWEIGHT AND EFFECTS OF EXERCISE

According to the World Health Organisation (WHO, 2000), obesity or being overweight is a result of an abnormal or excessive accumulation of fat in the body. In general it is believed that this excessive accumulation of fat is caused by an energy imbalance where energy intake exceeds energy expenditure over some period of time leading to a positive energy balance and resulting in weight gain (Durstine *et al.*, 2013; Goedecke *et al.*, 2006; Hill and Wyatt, 2005). The consumption of excess energy and/or low physical activity levels result in the surplus energy being deposited in the adipose tissue as fat (Durstine *et al.*, 2013). This surplus fat is detrimental to health in that it can lead to the alteration of the body's physiological function resulting in insulin resistance, dyslipidemia and the metabolic syndrome. However, obesity can also be caused by genetics. One method to classify overweight and obesity is by computing the body mass index (BMI). There is a classification of obesity done by the WHO (2000) in which the results are categorized to measure a person's health risk profile as shown in the table below.

Table 3.6: Categories for body mass index (WHO, 2000)

Classification	Kg/m²
Underweight	< 18.5
Normal	18.5 – 24.9
Overweight	25 – 29.9
Obese	30 – 39.9
Morbid Obesity	≥ 40

Obesity is associated with type 2 diabetes, dyslipidemia, hypertension, cardiovascular disease, ischemic stroke, sleep apnoea, gallstones, certain cancers, and degenerative joint disease (Katch *et al.*, 2011). The investigations by the WHO denote that more than 1.4 billion adults in the world are overweight, while more than 200 million men and 300 million women are obese (WHO, 2011). In the United States the NHANES surveys reported that 68.8% of the population is either overweight or obese implying more than two thirds of America's population have excessive accumulation of fat. Within South Africa an estimated 42.8 % women are obese, while 23.2% men are obese (WHO, 2013). Diseases associated with obesity lead to extensive costs on the worldwide economy. With USA spending approximately 117 billion dollars on the treatment of these diseases, while in South Africa an estimated 3 million worth of rands is used in the treatment of these diseases.

Consequently it is imperative that obese individuals lose weight, in particular reduce fat mass so as to reduce their risk of attaining chronic metabolic disease and cardiovascular disease (Sarsan *et al.*, 2006). Exercise can be used as a strategy to impede the development of obesity and help contain this global epidemic. This is important because Kelly *et al.* (2008) estimated that if the current secular trends are not changed by 2030 the global trend of overweight and obesity will increase to 2.16 billion (from 937 million in 2005) and 1.12 billion (from 396 million in 2005). This increase will confer enormous cost on public health systems and could also increase mortality rates.

The majority of people with obesity are likely to have six times more chances of having hypertension compared to their sedentary counterparts. Research evidence has shown that weight reduction is an effective tool in the management of obesity and an antihypertensive strategy, as a 10 % reduction in body weight is associated with a reduction in BP in obese hypertensives (Poirier and Despres, 2001). Moreover, dyslipidemia, diabetes and metabolic syndrome also coexist with obesity, and abdominal adiposity specifically is related with high levels of triglycerides, LDLs and low HDLs. Weight loss through exercise has been shown to increase the HDL levels in blood, while reducing the LDLs and triglyceride levels (Poirier and Despres, 2001).

Research evidence demonstrates that exercise favorably impacts on body composition by inducing weight loss (Tjonna *et al.*, 2013; Tjonna *et al.*, 2009; Irving *et al.*, 2008). Of paramount importance is the effect of exercise on fat mass and lean mass. Weight loss during exercise is a result of a loss of fat mass which therefore increases the amount of lean mass. This increase is particularly important as lean mass is metabolically more active, thus increases total energy expenditure and resting energy expenditure (Hill and Wyatt, 2005). This effect of exercise on fat mass gives it an edge over other weight loss interventions (dieting), where weight loss is usually due to a loss of lean mass (Birch *et al.*, 2005). It is also important to note that although exercise can induce weight loss, people should still not over eat so as to maintain higher total energy expenditures (Birch *et al.*, 2005).

Of particular interest is the new emerging HIIT that seems to be an economically viable and effective strategy for preventing obesity as numerous studies have demonstrated how it can induce fat loss in overweight and obese individuals (Boutcher, 2011; Tjonna *et al.*, 2009; Irving *et al.*, 2008). Exercise studies propose that HIIT induces fat mass and body weight loss by enhancing the lipolytic enzymes, expending more energy during exercise, having a greater negative balance and through excess post-exercise oxygen consumption (EPOC) (Hazell *et*

al., 2012; Boutcher, 2011; Houmard, 2008; Irving *et al.*, 2008; Imbeault *et al.*, 1997). HIIT also increases the catecholamine response which increases lipolysis and the availability of free fatty acids which results in an increase in overall fat oxidation during and after HIIT (Boutcher, 2011; Bracken *et al.* 2009; Trapp *et al.*, 2008). Bracken *et al.* (2009) reported a 6.3 and 14.5 fold increase in plasma epinephrine and norepinephrine at the end of sprinting exercise in healthy adults. Trapp *et al.* (2008) also reported decreases in leptin concentration. β -adrenergic receptors found in the abdominal fat have been reported to be increased by endurance training (Rebuffe-Scrive *et al.*, 1989; Crampes *et al.*, 1986), and this increase in abdominal β adrenergic receptor sensitivity is associated with a decline in abdominal fat in both endurance training and HIIT (Boutcher, 2011).

Exercise also increases fat oxidation rate and this is important as it could compensate for the impairment of fat oxidation that occurs with obesity. This was demonstrated by Talanian *et al.* (2007) who reported a 36% increase in whole body fat oxidation following two weeks of HIIT training in recreationally active women. This was accompanied by a reduction in net glycogen utilization. Increases in the enzymes involved in fat oxidation have been reported following HIIT and these enzymes increase the skeletal muscle's fat oxidation capacity (Talanian *et al.*, 2007; Tremblay *et al.*, 1994). Talanian *et al.* (2007) also reported a 32% increase in β -hydroxyacyl coenzyme after two weeks showing the viability of HIIT in inducing weight loss. This was further confirmed by Trapp *et al.* (2008) who also reported a 31% increase in the maximal activity of β - hydroxyacyl-CoA dehydrogenase. These findings show the potency of HIIT in increasing the muscle's capacity to oxidise fat which may contribute to weight loss.

Studies comparing HIIT and endurance training have reported that HIIT induces greater reductions in body mass and fat mass (MacPherson *et al.* 2011; Tjonna *et al.*, 2009; Irving *et al.*, 2008). Macpherson *et al.* (2011) observed a 12.4% loss in body fat in healthy active men

and women after HIIT, while the endurance training group reduced body fat by 5.8%. This was consistent with earlier findings by Similarly, Trapp *et al.* (2008) reported a 11.2% and 9.5% decrease in fat mass and abdominal fat in their HIIT exercise group, while the endurance training group did not lose fat mass, but decreased their abdominal fat by 10.5%. Tjonna *et al.* (2009) suggested that the greater weight loss observed using HIIT could be attributed to the fact that the high exercise intensities stimulates larger amounts of fat to be metabolized and induce a greater EPOC.

3.7. BODY MASS INDEX

Body mass index (BMI) is an indirect measure of body composition and is obtained from the measurement of body mass and weight. Body composition analysis is used to classify an individual's health risk profile in terms of the development of cardiovascular disease, diabetes, metabolic syndrome, hypertension and cancer (Powers and Howley, 2004). A number of factors interplay to influence body composition and include age, sex, heredity and physical activity (McAdele *et al.*, 2010).

The primary limitation of BMI is that it does not take the individual's morphology into account. In individuals who are mesomorphic, their BMI tends to be higher, and individuals may be classified as overweight /obese. A case in point is wrestlers who have big muscles. Similarly, in ectomorphic individuals adiposity can be underestimated and this may lead to an individual being classified as underweight (Breytenbach, 1996). Dudina *et al.* (2011) in a review of studies calculated that a unit increase in BMI is associated with a 1.14 mmHg increase in SBP, a 0.055 mM/L increase in total cholesterol and a 0.024 mM/L decrease in HDL.

3.8. WAIST CIRCUMFERENCE

Waist circumference is the circumference of the abdomen at its narrowest point between the lower costal (10th rib) border and the top of the iliac crest, perpendicular to the long axis of

the trunk (Marfell –Jones *et al.*, 2006). Hans *et al.* (1997) mentions that three categories have been identified as a way of classifying risk profiles in waist circumference as shown in Table 3.8.

Table 3.8: Waist circumference classifications of risk categories (Hans *et al.*, 1997)

Classification	Women Cm	Men Cm
Low Risk	<80	<93
Moderate Risk	80 – 88	94 – 102
High Risk	≥88	≥102

Waist circumference has been used as an estimate of intra-abdominal fat and some researchers are of the opinion that this parameter is a better indicator of cardiovascular disease and insulin resistance than BMI (Tabata *et al.*, 2009; Hans *et al.*, 1997). In men Tabata *et al.* (2009) demonstrated that waist circumference between 90-94cm was associated with a 15-fold increase in insulin resistance, while waist circumferences greater than 95cm was associated with a 45-fold increase in insulin resistance. This is because fat accumulation in the visceral fat compartment of the abdominal area has been associated with a higher risk for the development of insulin resistance, diabetes, dyslipidemia, hypertension, metabolic syndrome and atherosclerosis (Goedecke *et al.*, 2006, Despres, 2001). This could be attributed to the fact that there is a greater change in waist circumference (-4.6 cm) during interventions designed to promote health and encourage weight loss, compared to BMI (-1.9) (Hans *et al.*, 1997).

Tabata *et al.* (2009) showed in their study of 4800 men that there is a linear relationship between waist circumference and insulin resistance ($r = 0.78$). Additionally, Lapidus and colleagues (1984) reported a strong association between waist circumference and myocardial infarction. Moreover, Pouliot *et al.* (1994) indicated that women with waist circumferences above 87cm have a higher risk of developing cardiovascular disease and this increases even

further when waist circumference reaches the high risk category. Leung *et al.* (2011) further affirmed that waist circumference is an independent risk factor for the pathogenesis of hypertension. Changes in waist circumference are not necessarily dependent on weight loss; which implies that reductions in waist circumference occur even without weight loss. High waist circumference was associated with a 2.4 fold increased risk of developing hypertension (Leung *et al.*, 2011).

In summary, waist circumference is more superior to BMI and waist to hip ratio in the prediction of cardiovascular risk in that it is related to body mass and fat distribution, and its measurement is simple (Lean *et al.*, 1995).

HIIT has been shown to be able to induce waist circumference reductions in visceral fat. Whyte *et al.* (2010) reported that two weeks of HIIT resulted in a 1.1% reduction in waist circumference. Kay and Singh (2006) also mentioned that a reduction in waist circumference is correlated with an enhancement of glucose metabolism. The decrease in visceral adiposity could be attributed to the fact that the adipocytes in visceral fat have a greater number of β -adrenergic receptors thus making them more lipolytically active (Goedecke *et al.*, 2006).

3.9. CONCLUSION

Drawing from the literature, this chapter explained the effects of obesity on the human body and how it can lead to the development of pathological conditions such as diabetes, hypertension and cardiovascular diseases. It also demonstrated using literature the therapeutic role of exercise (HIIT and endurance training) in lowering the incidence of coronary heart disease and chronic diseases such as atherosclerosis and diabetes. The mechanisms by which HIIT lowers body mass, high glucose, cholesterol, and blood pressure levels were also explained.

Chapter 4

DETRAINING

4.1. INTRODUCTION

This chapter examines research evidence that shows the time course and mechanisms in which the adaptations gained in the various physiological systems during training are lost following a termination or reduction of training stimuli. It also aims to determine the extent to which this reversal in physiological adaptations occurs.

Physical activity stimulates specific adaptations in the body which depend upon the type, intensity and the duration of exercise performed (Coyle, 2000). It is unfortunate that these adaptations are not permanent and a cessation of training or insufficient training stimuli can result in the loss of adaptations acquired during training, prompting the popular cliché “*USE it or LOSE it.*” This loss in the gains acquired during training is known as the principle of disuse or the principle of reversibility (Wilmore and Costill, 2000). Detraining can be caused by inadvertent circumstances such as an injury, illness and surgery or intentional circumstances as in the case of (one taking) a vacation, travelling and rest at the end of a competition season (Mujika and Padilla, 2001b; Wilmore and Costill, 1988; Simoneau *et al.*, 1987).

According to Mujika and Padilla (2001a, 2001b) the principle of reversibility states that “whereas regular physical training results in several physiological adaptations that enhance athletic performance, stopping or markedly reducing training induces a partial or complete reversal of these adaptations, compromising athletic performance.” Wilmore and Costill define detraining as “the cessation of regular physical training”, while Mujika and Padilla (2000) define it as “the partial or complete loss of training- induced adaptations, in response

to an insufficient training stimulus.” For the purpose of this study the detraining definition by Mujika and Padilla will be used.

It is well documented that aerobic training, in particular HIIT, induces physiological adaptations that enhance muscular endurance and cardiorespiratory fitness. Likewise detraining is characterised by a reduction in the cardiorespiratory and muscular endurance and thus it is important to understand the effects and mechanisms involved (Mujika and Padilla 2000b). The reversal of adaptations gained during aerobic training seems to occur within a few days, weeks and months of training cessation and are influenced by the duration of aerobic training (Birch *et al.*, 2005) and whether training cessation is partial or complete. On the one hand individuals who have undergone a short period of training lose the adaptations gained through physical activity quickly. On the other hand the onset of detraining in individuals or athletes who have trained for longer periods and have had a history of training years seem to be delayed, with athletes being able to retain their fitness levels for a longer period (Wilber and Moffat, 1994; Coyle *et al.*, 1984).

4.2. MUSCULAR CHARACTERISTICS OF DETRAINING

In the muscle detraining can trigger a decrease in the mitochondrial size and enzymatic activities resulting in a decrease in the muscle's oxidative capacity. This is evident by the decline in the activities of key metabolic enzymes involved in aerobic metabolism. For example, a 40-60% decline in the activities of succinate dehydrogenase (Krebs cycle) and cytochrome oxidase (ETC) have been reported following a two week detraining programme in swimmers (Wilmore and Costill, 2000). Coyle *et al.* (1985) also reported a ~20% decline in succinate dehydrogenase, beta-hydroxyacyl-CoA dehydrogenase and malate dehydrogenase after three weeks of detraining and 40% after 56 days of detraining in endurance trained athletes. This is consistent with the findings of Chi *et al.* (1983) where they reported an average decrease of 36% in the maximal activities of succinate dehydrogenase (SDH), beta –

hydroxyacyl-CoA dehydrogenase and malate dehydrogenase in endurance trained cyclists and runners after a 12 week detraining period.

Similar responses were reported in highly trained distance runners after 15 days of inactivity (Houston *et al.*, 1979), while rugby players who detrained for six weeks showed a 25% decline in SDH (Allen, 1989). Beta-hydroxyacyl-CoA dehydrogenase also declined (12%) in endurance athletes who reduced their training volume from six to ten hours a week to 35 minutes per week in which they did a one high intensity work bout during a four week detraining period (Madsen *et al.*, 1993). They also reported a decline in exercise capacity of 21% in the detraining period. The results from these studies confirm that detraining decreases the muscle's oxidative capacity which brings about a decline in the endurance capacity (Madsen *et al.*, 1993).

A decline in the maximal activities of the enzyme citrate synthase (CS) has also been reported in a number of studies. For instance, a decline of 25 to 40% has been reported following detraining periods ranging from two to eight weeks (Amigo *et al.*, 1998; Bangsbo and Mizuno, 1988). These detraining effects were evident in endurance runners (Houmard *et al.*, 1992), soccer players (Bangsbo and Mizuno, 1988) and triathletes (McCoy *et al.*, 1994).

Muscle glycogen storage capacity has also been reported to decline following a detraining period. Costill *et al.* (1985) reported a 40% reduction in muscle glycogen content of endurance trained swimmers following four weeks cessation in training. It is also important to note that the swimmers had done intense training for five months, which was then followed by the detraining period. This then demonstrates that gains acquired during a long period (five months) of training can be quickly lost after a short period (four weeks) of detraining.

In terms of fat metabolism, two effects are evident. On the one hand detraining is associated with a lowering of the enzymatic activities of lipoprotein lipase in the muscle thus limiting the availability of FFA for fat metabolism. On the other hand there appears to be an increase in

the activities of the adipose tissue enzyme lipoprotein lipase which stimulates the storage of fat in the adipose tissue (Simsolo *et al.*, 1993). This was demonstrated by Narici *et al.* (1989) who reported a 45-75% decrease in muscle lipase activity during two weeks of inactivity, while this enzyme's activity increased by 86% in the adipose tissue.

The cessation of training has also been shown to affect the anaerobic capacity of the muscle, as it results in a decrease in enzymes of the glycolytic pathway. It is interesting to note that the rate at which the enzymatic activities of glycolysis are reversed is slower than that of the oxidative enzymes. This was demonstrated by Coyle *et al.* (1985) when they reported no decline in maximal activities of glycolytic enzymes following 84 days of detraining in endurance athletes although significant decreases in aerobic enzyme activities were noted.

Detraining also results in an increase in blood lactate levels during exercise and reduces muscle buffering capacity. Neuffer *et al.* (1989) demonstrated this in swimmers who performed a standard swim of 183-m at 90% of the swimmer's best time following a four week detraining period. Subsequently, blood lactate concentrations increased from 4.2 (\pm 0.8) to 9.7 (\pm 0.8) mmol/l after detraining. Burgomaster *et al.* (2007) further reported a total reversal in the lactate transporter proteins (MCT1 and MCT4) to pre-training values following six weeks of detraining in physically active men who had trained for six weeks. This reduction may impede the rate of lactate removal leading to an increase in blood lactate levels during exercise. Moreover, the lactate threshold would shift to the left and occur at a lower percentage of VO_{2max} .

The responses to detraining in previously sedentary people who partook in exercise have also been investigated in several studies (Table 4.1). The detraining periods induced similar reductions in the maximal activities of mitochondrial enzymes of both the oxidative and glycolytic pathways, as well as reduced capillary density as seen in individuals with a training history.

Table 4.1: Summary of studies investigating effects of detraining on muscle adaptations in previously sedentary individuals.

Study & Subject characteristics	Duration & Intensity	Adaptations to Training	Detraining	Results
Moore <i>et al.</i> (1987) Sedentary individuals	7 weeks		3 weeks	25 % decrease in CS
Wibom <i>et al.</i> (1992) Sedentary	6 weeks		3 weeks	Decreases in CS, GDH, cytochrome c oxidase
Klausen <i>et al.</i> (1981) Sedentary	30min –left and right leg cycle ergometer exercise 3d/week 8 wks	Increases 14.6 % in VO_{2max} , 20% capillary density, 30-40% in SDH and cytochrome-c oxidase	8 weeks	Decreases in SDH, cytochrome –c oxidase to pre training 10% decrease in capillary density
Simoneau <i>et al.</i> (1987) Sedentary men and women	15 wks of mixed CT and HIIT	Significant increases in MDH, HADH, OGDH and PFK, LDH. No significant change in CK	7 weeks	Decreases in HADH (21.2%), and OGDH (27.1%) No decrease in the PFK and LDH
Fournier <i>et al.</i> (1982) Sedentary adolescent boys	3 months sprint and endurance training	Significant increase in ST and FTa fiber area, SDH, VO_{2max} in endurance training Sprint training: increases in PFK	6 months	SDH, VO_{2max} and PFK maximal activities declined to pre training values

ST: slow twitch FTa: fast twitch, SDH: succinate dehydrogenase, VO_{2max} : maximal oxygen uptake PFK: phosphofructokinase, MDH: malate dehydrogenase, HADH- β hydroxyacyl-CoA dehydrogenase, OGDH- oxoglutarate dehydrogenase, CK: creatine kinase CS: citrate synthetase; LDH: lactate dehydrogenase; CT: continuous training. GDH- glutamate dehydrogenase.

4.3. CARDIORESPIRATORY CHARACTERISTICS OF DETRAINING

A large body of research evidence has shown that termination of CT or HIIT compromises cardiorespiratory fitness as shown by a decrement in maximal oxygen uptake, pulmonary ventilation, maximal ventilation equivalent and oxygen pulse (Orio *et al.*, 2008; Mujika and Padilla, 2000, 2001; Coyle *et al.*, 1986; Fringer and Stull, 1974).

The decline in VO_{2max} as a result of detraining depends on the period of detraining and the initial level of fitness of the individual (Mujika and Padilla, 2000, 2001). A cessation in

training shorter than 4 weeks in previously trained athletes has been reported to prompt a rapid decline in maximal oxygen uptake (4 – 14%) (Mujika and Padilla, 2001a). In previously sedentary individuals who have been recently trained, the loss in VO_{2max} during a 2 to 4 week period is less (3.6 - 6%) (Mujika and Padilla, 2000, 2001a). These findings are in line with the earlier results of Coyle *et al.* (1984) who reported a correlation of 0.93 between trained VO_{2max} and percentage reversal of VO_{2max} with detraining therefore, the higher the VO_{2max} , the greater the losses. The loss in training adaptations is not age dependent in previously trained individuals. The decrease in VO_{2max} after two months of detraining in young (range 19 – 25 years) and older (range 50 – 65 years) endurance athletes with many years of training was 16.3% and 16.9%, respectively (Giada *et al.*, 1998).

A number of studies investigating the time course of training adaptation reversals in athletes who are endurance trained have been done and the results indicate that loss of adaptations occur at a different rate compared to their sedentary counterparts (Giada *et al.*, 1998; Coyle *et al.*, 1984). Coyle *et al.* (1984) investigated the effects of detraining over a 12, 21, 56 and 84 days. In the first 12 and 21 days they observed a 7% decline in VO_{2max} which they attributed to a change in stroke volume as it rapidly declined to equal those of sedentary counterparts (at 12 days). The athletes were then tested again after 56 days of training cessation. The results showed a 16% reduction of VO_{2max} (Coyle *et al.*, 1984). After 84 days of inactivity the endurance athletes showed higher VO_{2max} (17%) values than those of the sedentary group. The authors suggested that the retention of VO_{2max} gains could be attributed to the second component of VO_{2max} which is the arterio-venous difference (Coyle *et al.*, 1984). This is because they observed that capillary density remained 50% higher than those of sedentary counterparts while there was only a partial loss of mitochondria content (citrate and succinate dehydrogenase activity which remained 50% higher) thus allowing an increased rate of oxygen transport and utilisation by muscles (Coyle *et al.*, 1984). This shows that central adaptations are rapidly lost during as short a period as 12 days of physical inactivity despite

years and years of training, while the peripheral adaptations tend to last a little longer. Despite the loss of VO_{2max} in endurance athletes associated with detraining, VO_{2max} still remains relatively higher than in sedentary people even after longer periods of up to two months.

In previously sedentary individuals detraining periods of longer duration (8 to 12 weeks) resulted either in a partial or complete loss of VO_{2max} . The retention of some of the training-induced VO_{2max} adaptations has been reported in a number of studies. Fringer and Stull (1974) had 44 college women undertake a ten week CT programme which was then followed by either a five (group one) or ten week detraining (group two) period. On the one hand group one showed a 35.5% increase in VO_{2max} after the continuous training, while detraining (five weeks) induced a 17.9% decline in VO_{2max} . However VO_{2max} remained 11.2% higher than pre-training values. On the other hand, group two reported a 38.7% increase in VO_{2max} after continuous training, however there was a total reversal of adaptations after the 10 week detraining period, as VO_{2max} was 1.3% lower than pre-training values.

Ready and Quinney, (1982) involved males in their study and had them train for nine weeks for 30minutes at 80% VO_{2max} four days per week. They reported a 19.4% ($p < 0.05$) increase in VO_{2max} . This training period was then followed by a nine week detraining period which resulted in the tapering of VO_{2max} , However this tapering remained higher than the pre-training values (Ready and Quinney, 1982). This was also consistent with the findings of Simoneau *et al.* (1987) who examined the responses of sedentary men and women during a 15 week training programme of mixed CT and HIIT. They reported a 17.6% ($p \leq 0.01$) increase in VO_{2max} after CT and HIIT training, while seven weeks detraining induced a significant decrease in VO_{2max} of 8.4% ($p \leq 0.05$). However the reduction observed in VO_{2max} after detraining was higher than pre-training values by 7.4%.

Other studies report a complete reversal of maximal exercise capacity after detraining. Marles *et al.* (2007) investigated the effect of six weeks of HIIT and detraining in sedentary

men. HIIT did not induce any statistically significant change in $\text{VO}_{2\text{max}}$ ($p = 0.24$), but there was a significant decline in $\text{VO}_{2\text{max}}$ ($p = 0.04$) following the six weeks detraining period. Wang *et al.* (1997) had sedentary women partake in an eight week CT programme which was followed by a four to twelve week detraining period. They had an initial increase of approximately 36% ($p < 0.05$) following CT and then four weeks of detraining resulted in a 15.6% reduction in $\text{VO}_{2\text{max}}$. However there was a total reversal of $\text{VO}_{2\text{max}}$ to pre-training values, after twelve weeks of detraining. Klausen *et al.* (1981) also reported a 14.6% ($p < 0.05$) increase in $\text{VO}_{2\text{max}}$ following eight weeks endurance training however the eight week detraining period resulted in total reversal of $\text{VO}_{2\text{max}}$ gains acquired during training similar to that of pre-training levels.

A majority of the studies done on previously sedentary individuals seem to indicate that a detraining period equivalent to that of the training period (eight to twelve weeks) invokes a total reversal in the $\text{VO}_{2\text{max}}$ changes accrued during training (Kemi *et al.*, 2004; Wang *et al.*, 1997; Klausen *et al.*, 1981; Fringer and Stull, 1974). However, detraining periods shorter than the training programs seem to invoke a partial loss of $\text{VO}_{2\text{max}}$ adaptations accrued during training.

The first initial declines in $\text{VO}_{2\text{max}}$ during three to four weeks have been attributed to a decrease in the CO which is mainly attributed to a loss in stroke volume. The reason for the decline in stroke volume is that there is also a rapid decline in blood volume which mediates the decrease in $\text{VO}_{2\text{max}}$. Coyle *et al.* (1986) showed that the decline in $\text{VO}_{2\text{max}}$ in the first weeks is attributed to changes in stroke volume as they reported a 6% to 12% reduction in $\text{VO}_{2\text{max}}$, stroke volume and plasma volume, respectively, after two to four weeks of detraining. The other reversals occurring in $\text{VO}_{2\text{max}}$ even after four weeks of training seem to be caused by a decrease in arterio-venous difference.

Neufer *et al.* (1987) surmised that partaking in moderate activity during a detraining period can help retain or slow the rate of deconditioning. They reported retention in VO_{2max} in collegiate swimmers who reduced their training to three days a week which required approximately 30% of the energy required by their prior endurance training. However, this was not the case with the other two groups of swimmers who reduced their training to one day per week and those who did not train at all. This then shows that moderate exercise and reducing training volume during a detraining period may act as an effective tool in avoiding the tapering off effect of detraining on cardiorespiratory adaptations.

4.3.1 Cardiac output

The decline in cardiorespiratory fitness, as shown by a reduction in VO_{2max} , is mainly due to the rapid, initial reduction in cardiac output (CO), followed by decreases in the oxidative capacity of muscles. The decrease in CO is mostly mediated by a reduction in stroke volume, rather than changes in heart rate, especially at higher exercise intensities.

Miyashata *et al.* (1978) reported a 6.9% decrease in maximal cardiac output after a six month detraining period in previously sedentary men who underwent a 15 week training programme. Coyle *et al.* (1984) also reported that the decrease in maximal CO stabilises at 8% below trained values after 21 days of detraining in endurance trained individuals.

4.3.2 Stroke volume

The decline in stroke volume (SV) after detraining is mainly attributed to changes in the blood volume. The decline in CO mentioned earlier by Coyle *et al.* (1984) was attributed to a 10% decline in SV within 12 days of inactivity and this reduction was also observed to stabilise at 10-14% below trained levels throughout the 12 to 84 days of detraining. Martin *et al.* (1986) reported a 17% lowering of SV in endurance athletes who refrained from training for three to eight weeks and said this decline is mediated by a decrease in the left ventricular mass and left ventricular end diastolic volume. The change in SV resulted in a 20% decline in

VO_{2max} . Since the increase in SV contributes at least 50% to the increase in VO_{2max} , it follows that exercise capacity will decrease significantly with a decline in SV.

4.3.3 Heart rate

Research studies have reported an increase in heart rate values during submaximal and maximal activities after detraining in endurance athletes (Houmard *et al.*, 1992; Coyle *et al.*, 1986; Culline *et al.*, 1986). This increase is a mechanism in which the body tries to counteract the effect of a reduced SV, however, this is not enough hence CO continues to decline, which ultimately leads to a decrease in VO_{2max} .

Coyle *et al.* (1986) reported an 11% increase in heart rate during exercise at submaximal and maximal intensities in endurance athlete after two to four weeks of detraining. This was consistent with the findings of Culline *et al.* (1986) who observed 5% increases in submaximal and maximal heart rates following 10 days of inactivity in endurance runners. Houmard *et al.* (1992) also reported an increase in heart rate (11 beats per minute) in endurance runners who refrained from training for 14 days. These studies all show that short periods of training cessation increases heart rate values during exercise at submaximal and maximal intensities as a way of compensating for the declines in SV.

4.3.4 Blood volume

As mentioned previously, one of the reasons why SV decreases with detraining is due to a reduction in blood volume. Blood volume reductions have been observed during short and long periods of training cessation, and in both athletes and sedentary individuals. In sedentary individuals who had trained for six days, Shoemaker *et al.* (1998) and Pivarnik and Senay, (1986) observed declines in resting plasma volume to pre-training values following six days and four weeks of inactivity, respectively. Similarly, Houmard *et al.* (1992) and Culline *et al.* (1986) reported reductions in plasma volume in order of 5% with 10 to 14 days of inactivity.

4.4. CHARACTERISTICS OF DETRAINING IN HEALTH MARKERS

Detraining has detrimental effects on health markers which include glucose levels, cholesterol levels, blood pressure and overweightness and obesity.

Detraining induces a decline in the muscle's uptake and utilisation of glucose as shown by a decrease in GLUT4 transporters which is mediated by insulin and exercise training. Vukovich *et al.* (1996) showed a 29.5% decline in GLUT4 transporters in endurance runners after six days of detraining, while McCoy *et al.* (1994) reported a 33.2% decrease in triathletes after 10 days of detraining.

Orio *et al.* (2008) reported that a 12 week detraining period (complete cessation) induced a total reversal of the adaptations in glucose, lipid profiles and BMI in overweight women attained during a 12 week CT programme. The reversal of glucose levels were attributed to the decrease in GLUT4 transporters. However, reduced activity during a period of detraining has been shown to retain in part the GLUT4 adaptations acquired through training. Burgomaster *et al.* (2007) investigated the effect of detraining following six weeks of HIIT and reported a 15% and 8% decrease in GLUT4 after one and six weeks of detraining, respectively. During the detraining periods they stopped the HIIT training but they continued their previous active lifestyle. These findings are related to a study done by Houmard *et al.* (1993) who reported a 92% retention in GLUT4 in participants who reduced their training volume by 50%, while those who stopped training completely had a total reduction of GLUT4.

A cessation in training also causes a reversal of blood pressure as well as a regain in body weight (Mujika and Padilla, 2000b; Motoyama *et al.*, 1995). Martin *et al.* (1986) reported an 11.6% increase in mean blood pressure following a 12 week detraining period in highly trained endurance cyclists and runners. They attributed this increase to a rise in total peripheral resistance. By contrast, Drinkwater and Hovarth, (1972) reported no changes in

blood pressure in women runners after 12 weeks of inactivity in the post competitive season this could have been a result of them being active.

Thompson *et al.* (1984) reported a 10% increase in low density lipoprotein cholesterol, while there was a 15% reduction in high density lipoprotein in endurance athletes who refrained from training for ten days. Similarly, Hardman *et al.* (1994) reported a 5% increase in high density lipoprotein after 12 weeks of brisk walking by sedentary women, but a 6.3% decrease (thus more than the training effect) in the high density lipoproteins following 12 weeks of detraining. Hardman *et al.* (1998) observed a 28.2% and 7.5% increase in very low density lipoproteins and the ratio of total cholesterol to high density lipoprotein cholesterol, respectively, in endurance athletes after 6.5 days of detraining.

Gutin *et al.* (1999) endurance trained overweight and obese children for four months (40 min, five d/wk), after which a four month detraining period followed. They reported an increase in body mass of 1.6% ($p = 0.001$) and bone density ($p = 0.045$) after the endurance training programme. However these changes were not sustained after the four month detraining period as they reported a 1.3% increase in body fat following the detraining period. Ferguson *et al.* (1999) also reported a reduction in insulin sensitivity (-25.4 pmol^{-1}) and plasma triglyceride concentration ($-0.24 \text{ mmol.l}^{-1}$) following a four month endurance training programme (40 min, five d/wk.) with obese children. The endurance training programme was then followed by a four month detraining period which resulted in an increase in insulin sensitivity ($+26.64 \text{ pmol}^{-1}$) and plasma triglycerides concentration. The results from these studies show that improvements accrued through training can be totally reversed when there is a cessation in training.

Lo *et al.* (2011) conducted a study in healthy college men who partook in a 24 week endurance training programme (30 minute run at 70-80% $\text{HR}_{\text{reserve}}$) which was then followed by 24 weeks of detraining. There was a significant decrease in the body fat percentage and

body mass of 0.4 and 0.6%, respectively after training. These changes were reversed to pre-training levels after the detraining period.

In summary a cessation of training invokes a reversal of adaptations gained during training. However reducing the exercise stimuli (for example training frequency or volume) can help maintain the gains obtained during training during a period of detraining.

4.5. CONCLUSION

Drawing from the literature this chapter explained the effects of detraining in endurance athletes and sedentary individuals on the muscular and cardiorespiratory adaptations which were acquired through aerobic training. This chapter also reviewed the effect of detraining on important health-related markers. A universal finding in the literature was that detraining resulted in a partial or total loss of metabolic and cardiorespiratory adaptations which are apparent within one week of inactivity. Similar tendencies were noted in the health markers. However, the initial level of fitness and duration of aerobic training determine the extent to which deconditioning occurs. In previously sedentary people a detraining period equal to that of the training period invokes a total reversal of cardiorespiratory changes gained during training. In athletes with longer histories of continued training some adaptations are retained, even after four weeks of detraining.

Chapter 5

PROBLEM STATEMENT

5.1. INTRODUCTION

Obesity is a serious condition which can lead to enormous financial costs incurred by the individual, as well as health systems and organizations. Many epidemiological studies have shown that physical inactivity, and being overweight and obese are risk factors for developing cardiovascular disease, hypertension, type 2 diabetes, cardio metabolic syndrome, various cancers (colon, endometrial, breast cancer), osteoporosis, dyslipidemia, obstructive sleep apnea and several psychological disorders. There is a close relationship between overweight and obesity and these diseases and an increase in overall morbidity and mortality as a result therefore (Helmert *et al.*, 1993; Paffenbarger *et al.*, 1993, 1990).

Regular physical exercise plays a vital role in promoting health and well-being. In light of this the American College of Sport Medicine's guidelines on physical activity state that 150 to 250 minutes of moderate physical activity per week is sufficient and effective to counteract weight gain (Donnelly *et al.*, 2009). In spite of the widespread acceptance that participation in physical activity helps reduce the risk of many diseases, participation remains low. Studies investigating barriers to participation in physical activity report that overweight and obese individuals mentioned a lack of time, lack of money, not being the sporty type, fear of injury, disliking exercising as some of the reasons (Felipe *et al.*, 2007; Zunft *et al.*, 1999).

Exercise prescription of a shorter duration represents a potential valuable approach to increase physical activity levels and health in the general population. High intensity interval training (HIIT) thus has the potential to provide a solution to these barriers, as it has been shown to be a time efficient strategy to induce physiological adaptations normally associated with

traditional endurance training (Gibala *et al.*, 2012, 2008; Boutcher, 2011). HIIT has been shown to elicit improvements in cardiorespiratory fitness, skeletal muscle oxidative capacity, glucose tolerance, weight loss, as well as lowering of systolic blood pressure. Moreover it has been used in the rehabilitation of patients with coronary artery disease (Warburton *et al.*, 2005) and as an intervention in cancer patients (Quist *et al.*, 2006), as well as obese men and women who are presumably the ‘not sporty’ type (Trilk *et al.*, 2011; Whyte *et al.*, 2010).

Increasing number of studies report the effectiveness and potential benefits of HIIT (Todd *et al.*, 2012; Fernandez-Fernandez *et al.*, 2012; Bartlett *et al.*, 2011; Iaia *et al.*, 2009; Gibala *et al.*, 2008; Burgomaster *et al.*, 2008; Perry *et al.*, 2008; Talanian *et al.*, 2007). However, there still remains a gap in knowledge as to the duration of the physiological adaptations induced by HIIT training. This is important as it will help in the prescription of training programmes and as a way of incorporating variations in physical activity which will help in exercise adherence. Moreover, the fact that most HIIT sessions last approximately 20 minutes, it means that they can be used by companies and organizations as an intervention during working hours to promote good health. In addition HIIT can be used when one has upper body injuries to avoid the effects of deconditioning.

5.2. PRIMARY AIM

The primary aim of this study was to investigate the training and detraining effects of a short-term HIIT programme on selected health-related measures in young overweight and obese women.

5.2.1 SPECIFIC AIMS

The specific aims of this study were:

- i.** To determine whether six HIIT sessions affect the body composition, maximal exercise capacity and health-related outcomes of young overweight and obese women.

- ii.** To quantify the magnitude of the training adaptations after six sessions of HIIT.
- iii.** To quantify the magnitude of the detraining effects after two weeks of HIIT.

Chapter 6

METHODOLOGY

6.1. STUDY DESIGN

This study follows an experimental design with two pre-tests, an intervention and two post-tests. Young women who met the inclusion criteria were asked to volunteer for the study. The participants acted as their own controls.

6.2. SUBJECTS

The recruitment of participants for this study was done through advertisements on campus. Fifty young university female students volunteered, however upon screening 25 students met the inclusion criteria for the study. Five of the participants did not complete the HIIT intervention because of knee pains from previous injury and shin splints (three), while the other two people fell ill. Twenty young women who were overweight or obese took part in this study. The body mass index (BMI) was used to determine those who met the inclusion criterion for overweight or obesity. The participants were asked to complete a Physical Activity Rating Questionnaire (PAR-Q) (Appendix A) and a Health Screening Questionnaire (Appendix B) which was also used as part of the selection criteria. The participants were asked not to change their dietary intake during the study. No dietary advice was given to participants during the research period. All participants were tested individually and all body composition measures were done in a private room.

Inclusion and exclusion criteria

Individuals were included in the study if:

- i. They were between the ages of 18– 25 years
- ii. They had been living a sedentary lifestyle (structured exercise \leq 1 day per week)

- iii. They had a BMI $\geq 25 \text{ kg/m}^2$ - 35 kg/m^2
- iv. They passed the preliminary screening procedure (physical activity and health screening).

Exclusion Criterion

Individuals were excluded from the study if:

- i. They had type 1 or type 2 diabetes
- ii. Stage 1, 2 or 3 hypertension (SBP $>130 \text{ mmHg}$ and DBP $> 90 \text{ mmHg}$)
- iii. They were on medication (e.g. antidepressant, anti-anxiety, thyroid or hypertension medication) that would affect their exercise responses
- iv. They had musculo-skeletal abnormalities or injuries.
- v. Individuals on a special diet.

6.3. ASSUMPTIONS

Firstly it was assumed that the participants were honest in reporting their daily activities in the physical activity readiness questionnaire, as well as in reporting that they are not on any medication stated in the exclusion criteria. Secondly it was assumed that the participants followed the instructions given to them to refrain from training during the detraining period. Thirdly it was assumed that the participants were honest in continuing with their habitual dietary intake.

6.4. DELIMITATIONS

In this study, instead of random sampling, a sample of convenience was used; this means that not everyone in the proposed study population had an opportunity to be selected for participation in the study, rather only those eligible, reachable and willing to participate. This implies that the findings from this study cannot be generalised to all women between the ages of 18 and 25 years.

6.5. PLACE OF STUDY

The study was conducted in the Sport Physiology Laboratory of the Department of Sport Science at Stellenbosch University. This is a fully equipped laboratory, which had all the apparatus that was needed for this project.

6.6. EXPERIMENTAL DESIGN

There were a 14 of visits by the participants to the laboratory on different occasions. The participants were tested individually and privately during all testing times (baseline, post and follow up testing). Moreover participants were informed about how changes in diet could affect the results of the study and were requested not to change their normal diet and activity levels. The experimental design is shown in Figure 6.1.

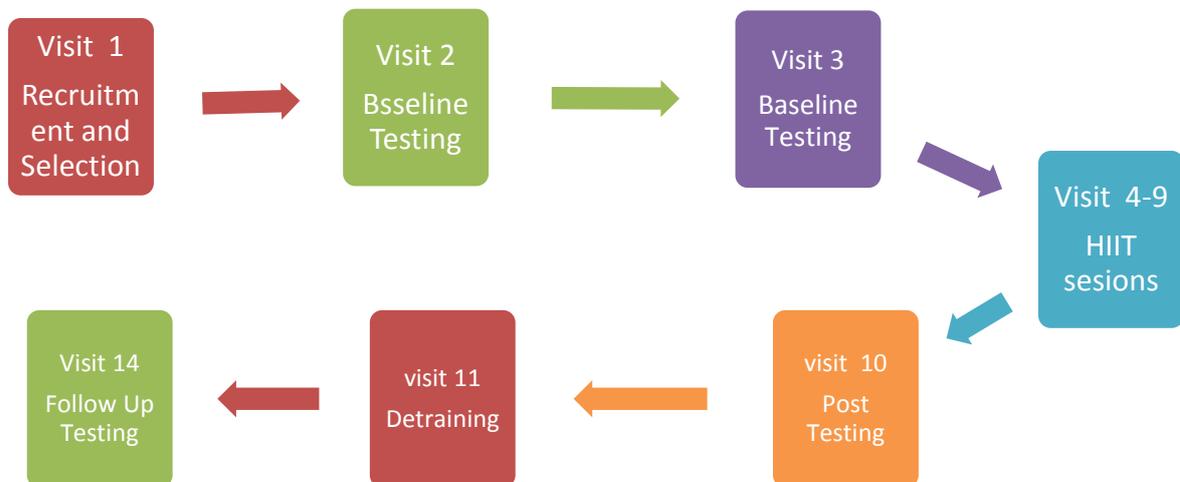


Figure 6.1: Schematic representation of the research design.

6.7.1 Visit 1 Screening and Inclusion

During the first visit to the laboratory, a full explanation of the purpose and procedures of the study was given to participants. They were then measured for height and body mass, so as to obtain their BMI (kg/m^2). If they qualified, they were then asked to complete the PAR Questionnaire (Appendix A) and Health Risk Assessment Questionnaire (Appendix B).

Thereafter resting blood pressure was measured. The participants, who met the inclusion criteria, were then asked to volunteer to participate in the study and complete the informed consent form.

6.7.2 Visit 2

The subjects were asked to take a twelve hour overnight fast prior to this laboratory session. As a way for controlling for fluctuations in blood glucose and cholesterol levels that can be induced by diet the participants were asked to write down a 24 hour food record which they were then asked to repeat during post testing and follow up testing. However the 24 hour diet record was not analysed in this study. Blood samples were taken to measure blood glucose and total cholesterol levels. Other variables measured were body mass, height, percentage body fat, lean mass, waist circumference, hip circumference and blood pressure. Body mass index was calculated from body mass and height while the waist to hip ratio was calculated from the waist and hip circumference.

6.7.3 Visit 3

The VO_{2max} test was performed on the treadmill, while blood lactate concentrations were measured.

6.7.4 Visit 4– 9 HIIT sessions

The participants were asked to perform six HIIT sessions under supervision in the laboratory over a two week period. The first three sessions lasted 30 minutes. Each session consisted of ten 1-minute running intervals at 90 to 95% of HR_{max} separated by one minute active recovery intervals. The last three sessions lasted 40 minutes and consisted of fifteen 1-minute running intervals at 90 to 95% HR_{max} and these were also separated by one minute active recovery intervals. Subjects ran for five minutes during warm up and five minutes during cool down at an intensity eliciting 60% of HR_{max} .

6.7.5 Visit 10 Post Testing

The first post testing was done within 48 hours of the last session of the HIIT programme. Testing protocols used were the same as for baseline testing. These included body mass, height, blood pressure, blood glucose level, percentage body fat, waist circumference, hip circumference, total cholesterol, VO_{2max} and lactate concentrations. Body mass index was calculated from body mass and height while the waist to hip ratio was calculated from the waist and hip circumference.

6.7.6 Visit 11 Detraining

The participants were asked to refrain from any structured exercise for 2 weeks.

6.7.7 Visit 12 Follow up

The participants were assessed within 48 hours after two weeks of detraining. All tests were done following the baseline protocols and procedure.

6.7. PROCEDURES AND PROTOCOLS

6.8.1 Anthropometric measurements

The anthropometric measurements consisted of; stature, body mass, waist circumference and hip circumference. Waist and hip circumference were used to determine the waist to hip ratio, while bioelectrical impedance analysis (BIA) was used to determine percentage body fat and lean mass.

6.8.2 Body mass

Body mass was measured using a calibrated electronic scale (UWE BW-150, Brisbane, Australia) and recorded to the nearest 0.1 kg. Subjects were asked to stand barefoot in the middle of the scale, distributing their weight evenly on both legs. They were clothed in minimal and light weight clothing.

6.8.3 Stature

Stature is defined as the perpendicular distance between the transverse planes of the vertex and the inferior aspects of the feet (Marfell-Jones *et al.*, 2006). The stretch stature method was used, using a stadiometer (SECA, model 220, Hamburg, Germany). The participants were asked to stand barefoot on the scale with their heels together. The heels, buttocks and upper part of the back touched the scale. The subject's head was in the Frankfort position. This is when the orbital (lower edge of the eye socket) and the tragion (the notch superior to the tragus of the ear) are horizontally aligned. The subject was asked to take a deep breath and then the researcher placed the head board firmly down the vertex compressing the hair as much as possible. The measurement stature was taken to the nearest 0.1 cm. The measures for height and mass were used to determine BMI.

The classification of obesity that was used in this study was those from the World Health Organization (WHO), based on BMI. The WHO (2000) designations include the following:

- i. Grade 1 overweight (commonly and simply called overweight) - BMI of 25-29.9 kg.m²
- ii. Grade 2 overweight (commonly called obesity) - BMI of 30-39.9 kg/m²
- iii. Grade 3 overweight (commonly called severe or morbid obesity) - BMI greater than or equal to 40 kg/m².

6.8.4 Girths

Waist and hip girths were measured using a Ross craft (Canada) non-extensible anthropometric tape. The cross hand technique was used for measuring. The tape was held at right angles to the limb/body segment and the tension in the tape was constant, so as to ensure that gaps and indentations were minimized. The researcher's eyes were at the same level as the tape when taking the reading so as to avoid any error of parallax.

6.8.5 Waist girth

Waist girth is defined as the circumference of the abdomen at its narrowest point between the lower costal border (10th rib) and the top of the iliac crest, perpendicular to the long axis of the trunk (Marfell–Jones *et al.*, 2006). The subjects were asked to stand relaxed and fold their arms across the thorax. The tape was placed around the waist and the measurement was taken at the end of a normal expiration and noted to the nearest 0.01 cm.

6.8.6 Hip girth

The hip girth measurement was taken to the nearest 0.1 cm at the level of the greatest posterior protuberance, perpendicular to the long axis of the trunk. The subjects were asked to stand relaxed and fold their arms across the thorax, with the feet together and the gluteal muscles relaxed.

6.8.7 Bioelectrical impedance analysis (BIA)

Subjects were asked to refrain from taking diuretics such as caffeine and alcohol 12 and 24 hours before the test respectively, as well as not to participate in any exercise 24 hours before testing time (Heyward and Wagner, 2004). Before the commencement of the procedure subjects were asked to void their bladders. BIA was done using the Biostat Quadscan 4000 (Isle of Man, United Kingdom). The subjects were then asked to lie down quietly on the table in a supine position for ten minutes after which the measurement began.

This procedure lasted approximately five minutes and involves sending a very small electrical current through the body (800 μ A at 50 kHz), which cannot be felt. This small current measures the resistance of the tissue. Lean tissue provides less resistance than adipose tissue because of its greater composition of water and electrolytes. The subjects lay quietly in a supine position with the arms 30° from the body. The limbs should not touch each other or the center of the body. The sites for electrodes attachment were cleaned with an alcohol swab. Two electrodes were placed on the dorsal side of the hand; one centimeter proximal to the

knuckle of the middle finger and on the wrist between the head of the ulna and radius. Two electrodes were placed on the dorsal side of the bare right foot between the lateral and medial malleoli and at the base of the toes between the hallux and the third phalange. The cables were attached to the electrodes and the analyser was switched on. The resistance and reactance were recorded when the measurement stabilized.

6.8.8 MAXIMAL EXERCISE CAPACITY TEST (VO_{2max})

The VO_{2max} test protocol was conducted on a treadmill. The objective of the test was to measure the subjects' endurance capacity and to determine the exercise intensity for the HIIT sessions. The h/p/cosmos Saturn treadmill (Nussdorf-Traunstein, Germany) is interfaced with specialized computer software of the Cosmed Quark CPET metabolic system (Rome, Italy). By using breath by breath analysis together with a telemetric heart rate monitor (POLAR, Polar Electro Oy, Finland), the CPET software calculates and records exercise intensity and selected cardio-respiratory parameters continuously throughout each test. The gas analysers were calibrated prior to each test with atmospheric gas and known gas concentrations (16% O_2 , 4% CO_2 balance N_2) and the turbine flow meter was calibrated with a 3 L calibration syringe.

The subjects were fitted with an adjustable safety harness on the treadmill. The participants first warmed up for 2 minutes at 5 km/hr and 0% gradient. The participants were then allowed to drink water, before the face mask was fitted. The test started at a speed of 5 km/hr and an incline of 0%, thereafter the speed and the incline of the treadmill were increased at three minute intervals (Table 3) until at least two of the test termination criteria were reached. This protocol was used instead of the Bruce protocol because in our experience this protocol is well tolerated by overweight /obese individuals and causes less local muscle fatigue than the Bruce protocol. This then allows the participants to extend the time to exhaustion thus give a more accurate measure of their cardiorespiratory fitness.

These criteria, were as follow: (i) the VO_2 does not increase by more than 150 ml per successive workload, (ii) a respiratory quotient (R-value) equal or above 1.15 is reached, (iii) heart rate is more than 90% of the age predicted maximal heart rate, (iv) the rating of perceived exertion is above 19 on the 6-20 Borg scale and (v) the subject indicates she is exhausted (McArdle *et al.*, 2010).

During the three different test times (baseline, post and follow up testing) the temperature and humidity in the laboratory ranged from 17°C to 25°C, while humidity was 47.5%.

Table 6.1: The $\text{VO}_{2\text{max}}$ Running Protocol.

Stage	Time	Incline	Speed Km/hr.
1	3	0	6
2	3	0	7
3	3	0	8
4	3	0	9
5	2	1	10
6	2	1	11
7	2	1	12
8	1	2	13
9	1	2	14
10	1	2	15

6.8.9 Measurement of blood [lactate]

A finger stick blood sample was taken before the $\text{VO}_{2\text{max}}$ test commenced to determine the resting blood [La]. The finger was cleaned with an alcohol swab and then pricked once with an Accucheck soft click (Roche diagnostics, Mannheim, Germany). The blood was drawn into the capillary tube of the Lactate Pro meter (ARKRAY, Inc. Kyoto, Japan). Further measurements were taken at the end of each completed stage of the $\text{VO}_{2\text{max}}$ test.

6.8.10 Measurement of total cholesterol

Blood samples were obtained after a 12 hour overnight fast hence all samples were taken in the morning. Subjects were required to sit for five minutes before blood sampling. The subject's finger was first cleaned with an alcohol swab and then pricked once using an Accucheck soft click. The first blood sample was wiped away, and then the second drop was put on the cholesterol strip in the meter (Accutrend Plus, Roche, Mannheim, Germany) and the lid was closed. Results were shown after approximately two minutes. The calibration of the cholesterol meter was done each day before testing.

6.8.11 Measurement of blood glucose

The Hemocue Glucose 201+ Analyser (Angelholm, Sweden) has an inbuilt electronic self-test that verifies its performance every time the device is switched on. Blood samples were obtained after a 12 hour overnight fast hence all samples were taken in the morning. Subjects were required to sit for five minutes to stabilize plasma volume, before blood sampling. The subject's finger was first cleaned with an alcohol swab and then pricked once using an Accucheck soft click. The first blood was wiped away and the second blood was drawn into the cuvette using capillary action and put into the glucometer.

6.8.12 Measurement of blood pressure

The subjects were asked to sit for ten minutes before blood pressure was measured (Nemoto *et al.* 2007; Dengel *et al.*, 1998). The blood pressure was measured on the left arm using an Ergoscan duo Recorder (Munich, Germany). The correct cuff size was used and put on the arm, making sure it closely surrounded the arm but did not exert any pressure on the veins. Subjects were instructed not to talk or move during measurement. The blood pressure measurements were taken two minutes apart and the mean was calculated and used for data analysis (Astorino *et al.*, 2012; Leung *et al.*, 2011).

6.8. HIIT EXERCISE PROTOCOL

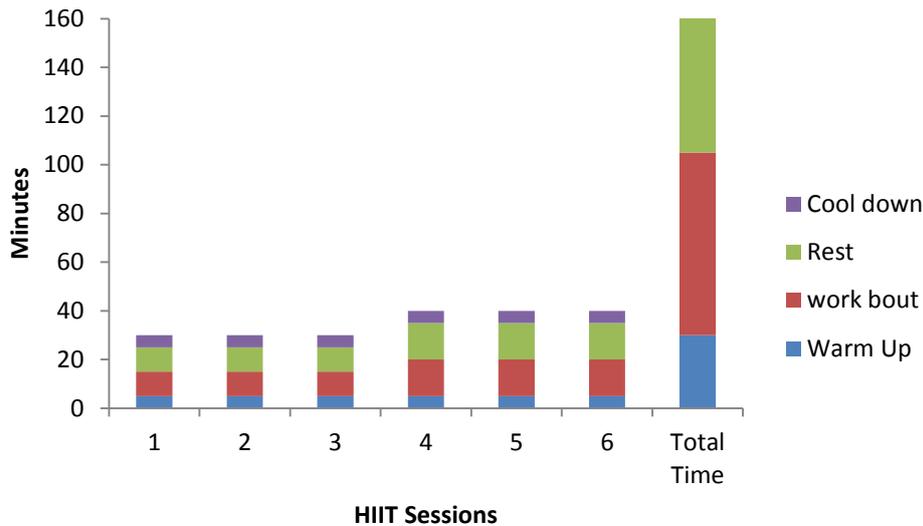


Fig 6.2: Duration of the HIIT sessions

The participants followed a training protocol modified from Hood *et al.* (2011) and Little *et al.* (2011). These studies showed that this protocol is well tolerated by participants compared to the Wingate protocol where participants reported feeling nauseated and light headed. Moreover the pilot study showed that the participants could not maintain the high intensity bouts for longer than one minute. The HIIT intervention programme was done over two weeks in which six sessions were done. The first three HIIT sessions were done during the first week whereas the last three were conducted during the second week. The total exercise time for each session was 30 minutes which consisted of a five-minute warm up period (at approximately 60% HR_{max}) followed by ten one-minute high intensity work bouts at 90 - 95% of HR_{max} which were interspersed by ten one-minute rest intervals at approximately 50% of HR_{max} . The last one-minute rest interval was then followed by a five-minute cool down period (at approximately 60% HR_{max}). During the second week three HIIT sessions were done but total exercise time increased from 30 minutes to 40 minutes. The total exercise time for each session consisted of a five minute warm up period (approximately 60% HR_{max}), followed by fifteen one-minute high intensity work bouts at 90 - 95% of HR_{max} which were interspersed by fifteen, one-minute rest intervals (50% HR_{max}). The last one-minute rest interval was then

followed by a five-minute cool down period (approximately 60% HR_{max}). Subjects exercised with heart rate monitors (POLAR, Polar Electro Oy, Finland) so as to ensure that they were exercising at the correct intensities.

6.9. POST TRAINING AND DETRAINING MONITORING

After completion of the six HIIT sessions, participants were assessed using the same baseline procedures. They then resumed their daily activities with no constructive exercise for two weeks. At the end of the two weeks follow up testing was undertaken.

6.10. ETHICAL ASPECTS

The study protocol was approved by the Ethics Committee of Research Subcommittee A of Stellenbosch University (DESC_Terblanche2012). All participants in this study were volunteers. The study protocol was explained to them and they were free to withdraw from the study anytime during the study period. All possible precautions were taken to minimize the risk of injury during testing and training.

6.11. STATISTICAL ANALYSIS

The data was analysed using SPSS v. 15.0 (SPSS, Inc., Chicago, IL, USA). The descriptive statistics were reported as means and standard deviations (SD). The one way repeated measures ANOVA was used to determine if there was any difference in the three test times for the following variables: body mass, percentage body fat, lean mass, waist circumference, waist to hip ratio, glucose, cholesterol, blood pressure, VO_{2max} and lactate concentrations. If the F-value was statistically significant, then the Fischer's Least Significant Difference (LSD) *post hoc* test was done to identify the test times that were statistically significant from the rest. Pearson product-moment correlations were calculated for changes between pre to post values and post to follow up values. Statistical significance was set at $p < 0.05$.

Chapter 7

RESULTS

7.1. Subject characteristics

Fifty young university female students volunteered to participate in the study, however, upon screening 25 students were eligible for the study. All participants replied “NO” to the PAR-Q questions, which then showed that it was safe for them to participate in physical activity. In addition the subjects reported that they were engaged in physical activity of less than one day per week. The participants were between the ages of 18 – 25 years and living a sedentary lifestyle. Of these 25 participants, five did not complete the HIIT intervention because of knee pains from previous injury and shin splints (three), while the other two people fell ill. This left a total of 20 participants who completed the intervention; however, two of these participants did not complete the final VO_{2max} test after detraining because of travel arrangements.

Table 7.1 shows the subject characteristics at pre-training. According to the WHO (2000) BMI risk categories the participants were either overweight or obese (29.0 ± 3.40). In fact, Figure 7.1a shows a bar graph for the actual frequency distribution of participants among the BMI risk categories. The mean waist circumference at pre-training was 84.1 cm and this meant that the sample fell in the category of moderate risk which ranges from 80 – 88cm (Hans *et al.*, 1997). The average waist to hip ratio was above 0.80 showing that most participants were at high risk of developing cardiovascular diseases (Srikanthan *et al.*, 2009; Wellborn and Dahlia, 2007).

Table 7.1: Participant Characteristics at Pre-training

Variable (n= 20)	Mean	SD	Range		
Age (years)	21.2	1.93	19.0	–	25.0
Height [cm]	160.0	6.66	145.8	–	172.7
Body mass [kg]	74.3	10 .00	61.0	–	103.1
BMI	29.0	3.40	25.0	–	35
Fat Mass [%]	35.5	5.50	25.9	–	45.5
Lean Mass [%]	64.5	5.50	54.5	–	74.1
Waist Circumference [cm]	84.1	9.50	68.0	–	101.0
Hip Circumference [cm]	107.7	8.20	93.0	–	124.8
Waist to Hip Ratio	0.8	0.07	0.65	–	0.95

* Pre-training versus Post-training: $p < 0.05$; # Post versus Follow Up: $p < 0.05$; † Follow Up versus Pre-training $p < 0.05$

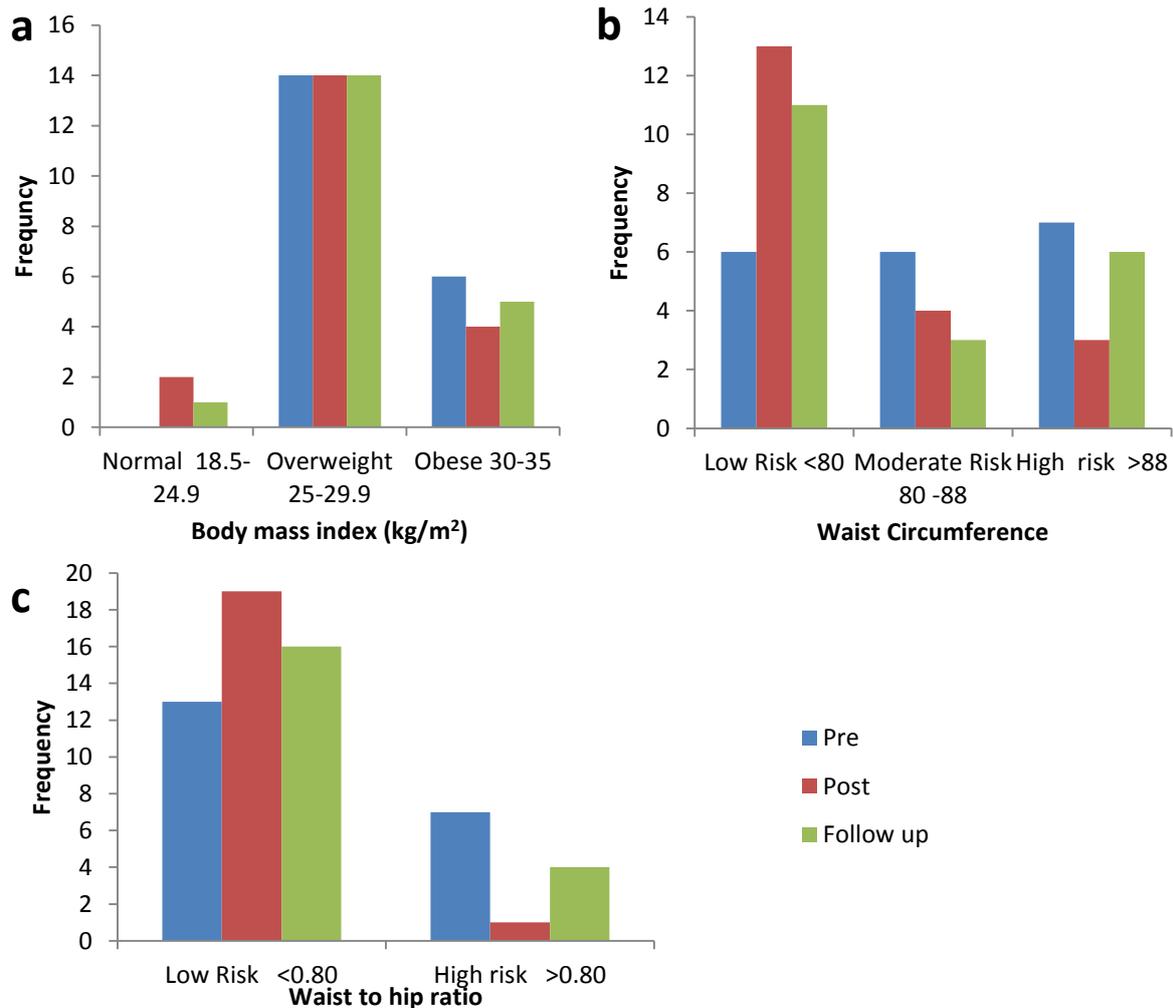


Figure 7.1: The distribution of the study sample in the risk categories for (a) BMI, (b) waist circumference and (c) waist to hip ratio.

Figure 7.1a shows that the HIIT intervention reduced the number of people in the high risk category for BMI. The number of people in the obese category reduced by 10% and this number even remained lower after detraining. The graph also shows that training resulted in a shift of 10% towards the normal weight category. After detraining 5% of the sample remained within this category.

The HIIT intervention increased the number of people in the low risk category for waist circumference by 35%. However, after the detraining period there was a 15% increase in the number of people in the high risk category showing that people regained weight after the detraining period which caused a partial reversal of the improvements obtained during the

HIIT program. In addition, HIIT also reduced the number of people in the high risk category for the waist to hip ratio by 30%, however, there was a 15% increase in the number of people in the high risk category after the detraining period again signifying a partial reversal.

7.2. The effect of training and detraining on body composition

On the one hand Table 7.2 shows that two weeks of detraining resulted in a reversal of the statistically significant ($p < 0.05$) improvements in body mass, percentage body fat and lean mass that had been acquired during the HIIT intervention. Percentage body fat increased to pre-training levels whereas body mass and lean mass declined almost back to pre-training values. This trend meant there were no statistically significant differences between the pre-training and the follow up values for these variables. On the other hand the detraining period induced a partial loss in mean waist circumference and BMI which were significantly higher than pre-training values (1.6 cm and 0.03cm, respectively).

Table 7.2: Changes in body composition variables between pre-training and follow up

Variables	Pre-training		Post-training		Follow Up	
	Mean	SD	Mean	SD	Mean	SD
Body Mass	74.3	9.97	73.1	10.26*	73.6	10.13
Percentage Body Fat	35.5	5.53	34.2	5.88*	35.5	5.62
Lean Mass	64.5	5.53	65.7	6.11*	64.4	5.79
Waist Circumference	84.1	9.52	80.1	8.65*	82.5	10.76 ^{#†}
Waist to Hip Ratio	0.78	0.07	0.76	0.04*	0.77	0.06
BMI	29.0	3.36	28.5	3.48*	28.7	3.45 [†]

* Pre-training versus Post-training: $p < 0.05$; # Post versus Follow Up: $p < 0.05$; † Follow Up versus Pre-training $p < 0.05$

Figure 7.2 illustrates the changes in body composition following the HIIT intervention. Firstly, there was a statistically significant reduction of -1.6% ($p = 0.001$) in the body mass for the 20 participants. However, this weight was regained over the two weeks detraining

period to a level equal to the pre-training measurements. Secondly, percentage body fat decreased significantly by -3.7% ($p = 0.001$) from pre-training to post-training. Thirdly, lean mass (LM) increased significantly by 1.9% ($p = 0.001$) from pre- to post-training. Statistically significant changes were also observed in the measures of central adiposity, where results showed a -4.8% ($p = 0.001$) reduction in waist circumference from pre-training to post-training. The waist circumference reduction also remained significantly lower (-1.9%, $p = 0.001$) than pre-training values after the two weeks of detraining. Waist to hip ratios also reduced significantly by -2.6% ($p = 0.01$) following HIIT. However, the changes were not sustained over the detraining period and they returned to pre-training values. Finally, the BMI was also significantly reduced by 1.7% ($p = 0.001$) after training and remained significantly lower compared to pre- training by 1.1% ($p = 0.02$).

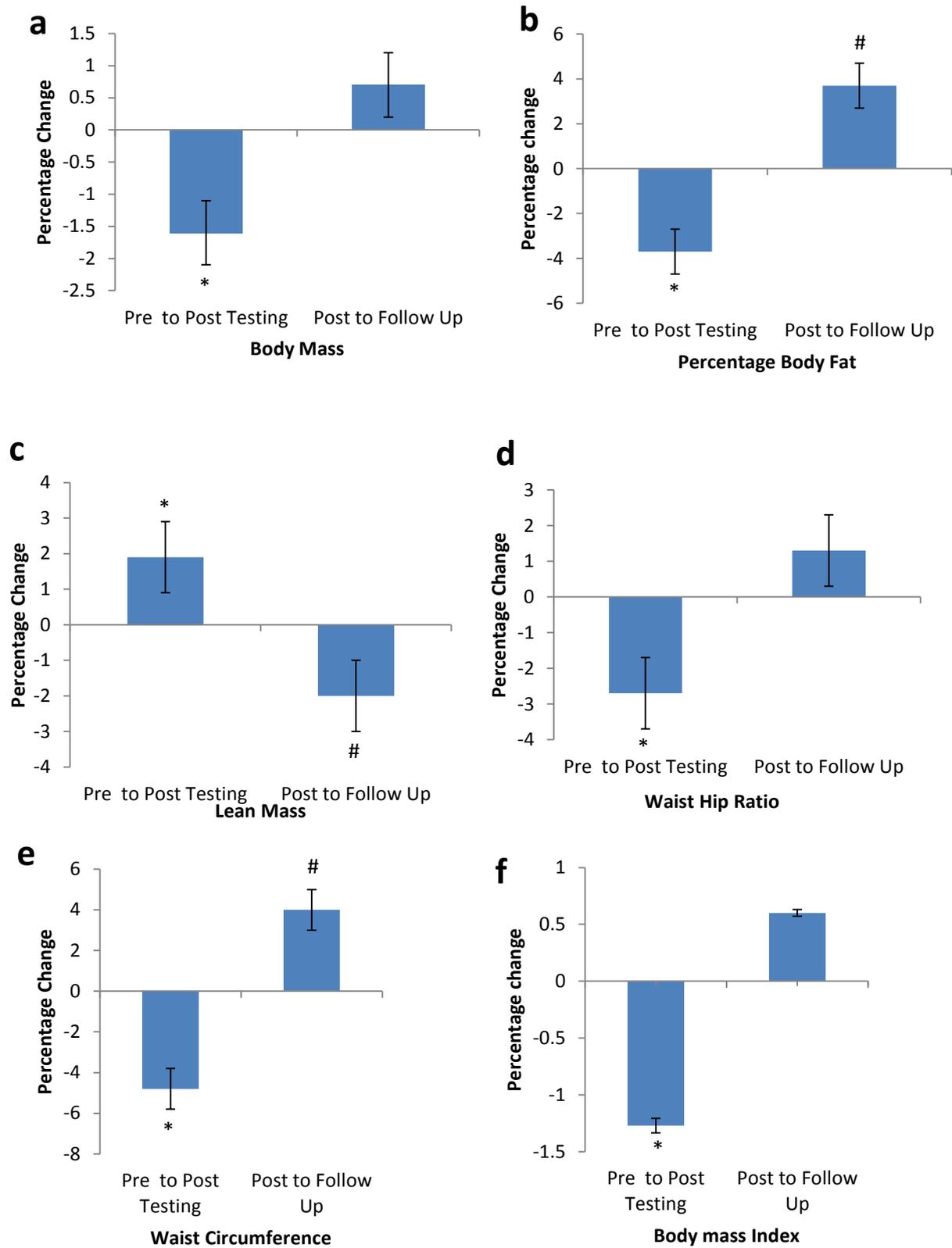


Figure 7.2: Changes in body composition after HIIT.

* Pre-training versus Post-training: $p < 0.05$; # Post versus Follow Up: $p < 0.05$; † Follow Up versus Pre-training $p < 0.05$

7.3. The effects of training and detraining on blood glucose and total cholesterol levels

The HIIT intervention induced a statistically significant change in both glucose and cholesterol levels (Figure 7.3). The glucose levels were significantly reduced by 11% ($p = 0.001$) from pre- to post-training. Despite the two week detraining period glucose levels remained significantly lower (4.9%, $p = 0.01$) than pre-training levels. A statistically significant reduction of 10.4 % ($p = 0.01$) was observed in cholesterol levels after the two weeks of HIIT training, and even remained lower (8.76%, $p = 0.02$) than pre-training values after detraining.

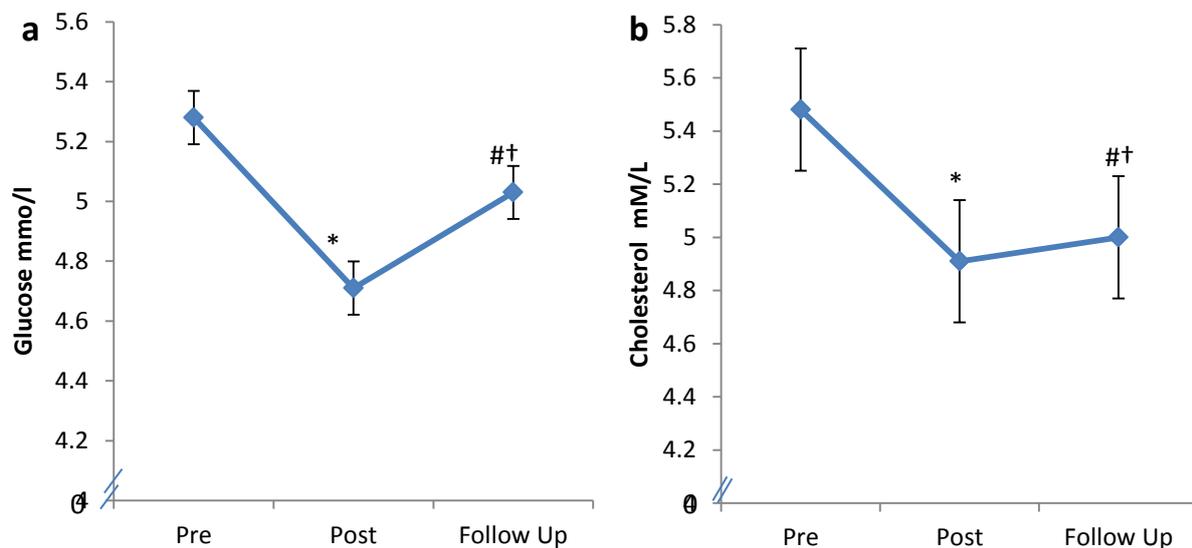


Figure 7.3: Changes in Glucose and Cholesterol levels before and after HIIT and detraining.

*Pre-training versus Post-training: $p < 0.05$; # Post versus Follow Up: $p < 0.05$; † Follow Up versus Pre-training $p < 0.05$

7.4. The effects of training and detraining on blood pressure

The resting systolic blood pressure (SBP) decreased significantly by 3.4% ($p = 0.001$) after the HIIT programme, while the resting diastolic pressure (DBP) decreased significantly by 5.8% ($p = 0.001$) (Figure 7.4). However, after detraining, the SBP reversed to pre-training values. On the contrary, DBP remained 1.4% lower than pre-training values.

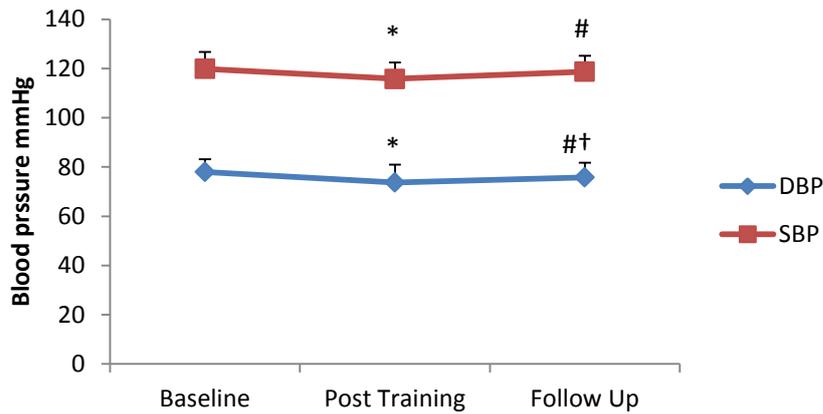


Figure 7.4: Changes in systolic and diastolic blood pressure levels before and after HIIT and detraining

*Pre-training vs. Post-training: $p < 0.05$; # Post vs. Follow Up: $p < 0.05$; † Follow Up vs. Pre-training $p < 0.05$

7.5. The effects of training and detraining on maximal aerobic capacity

Table 7.3 illustrates the performance of participants during the maximum aerobic capacity test. There were statistically significant increases in the relative VO_{2max} (27.8 ± 5.69 to 29.3 ± 5.06 , $p = 0.00$) and maximum workload (11.0 ± 2.16 to 12.2 ± 2.43 , $p = 0.04$) after HIIT. Both variables returned to pre-training after the two week detraining period.

Table 7.5: Changes in maximal exercise capacity

Variables	Pre-training		Post-training		Follow Up	
	N = 20		N = 20		N = 18	
	Mean	SD	Mean	SD	Mean	SD
VO_{2max} [ml/kg/min]	27.8	5.69	29.3	5.06*	27.6	4.33
VO_{2max} (L/min)	2.01	0.38	2.1	0.30	1.99	0.25
VE_{max} [l/min]	86.0	13.85	93.1	15.15*	89.1	14.10
AT%	70.6	8.79	68.5	8.74	69.4	8.83
HR_{max} [min-1]	194.4	7.37	191.7	7.97	192.9	9.45
RE_{max}	1.2	0.07	1.1	0.07	1.1	0.05
La Max [mM/L]	10.1	1.88	11.0	1.97*	10.3	1.76
RPE	19.0	1.21	19.3	1.21	19.1	1.26
WL_{max}	11.0	2.16	12.2	2.43*	11.4	2.63
VO_{2max}: maximal oxygen uptake; VE_{max}: maximum minute ventilation; AT: anaerobic threshold; HR_{max}: maximum heart rate; RE_{max}: maximum respiratory exchange ratio; La_{max}:—maximum lactate concentration; RPE: rating of perceived exertion; WL_{max}: maximum workload.						

* Pre-training versus Post-training: $p < 0.05$; # Post versus Follow Up: $p < 0.05$; † Follow Up versus Pre-training $p < 0.05$

Figure 7.5 shows that the HIIT intervention induced a 5.1% ($p = 0.02$) increase in relative VO_{2max} which was statistically significant. However this was not sustained over the two week detraining period as it induced a total reversal in relative VO_{2max} (5.8%, $p = 0.35$) which was gained during the HIIT intervention.

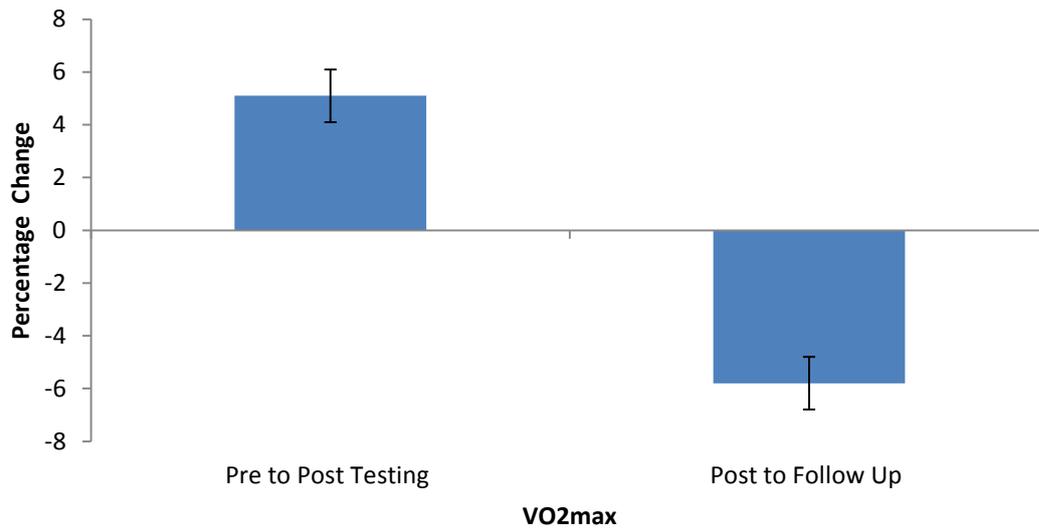


Figure 7.5: The effect of HIIT and detraining on maximal aerobic capacity of the participants.

* Pre-training versus Post-training: $p < 0.05$; # Post versus Follow Up: $p < 0.05$; † Follow Up versus Pre-training $p < 0.05$

7.6. The effect of training and detraining on the exercise time to reach critical parameters

Fig.7.7 summarises the results of the time taken to reach the anaerobic threshold, FBLA at 4-mM/L and VO_{2max} during the maximal exercise test. These indices are indicators of exercise tolerance. The time taken to reach the anaerobic threshold increased significantly from pre-training to post-training ($200.0 \pm 96.14s$ to $348.0 \pm 171.60s$, $p = 0.001$). The time taken to reach FBLA at 4 mM/L also increased significantly from pre-training to post-training ($484.5 \pm 347.55s$ to $641.0 \pm 267.15s$, $p = 0.001$). The increase in FBLA observed after the HIIT intervention was sustained after the detraining period, as results showed significant differences between pre-training and follow up times taken to reach FBLA at 4 mM/L ($632.7 \pm 293.95s$, $p = 0.001$). Similarly, time taken to reach relative VO_{2max} also increased significantly following the HIIT intervention ($837.3 \pm 230.74s$ to $954.9 \pm 213.29s$, $p = 0.001$).

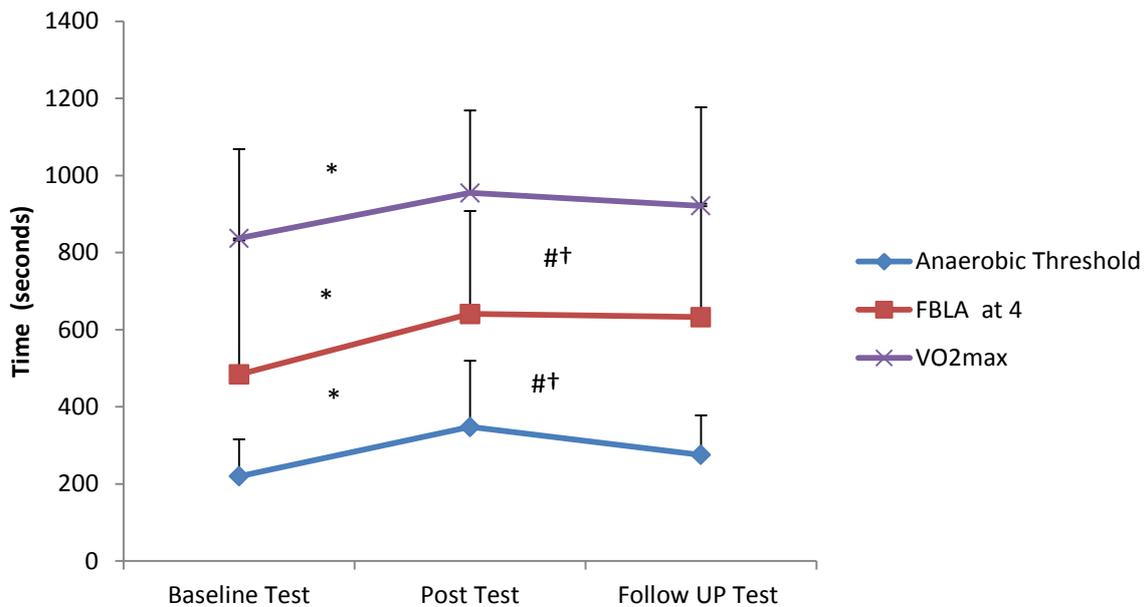


Figure 7.6: Time taken to reach aerobic threshold, FBLA at 4mM/L, and VO₂max.

* Pre-training versus Post-training: $p < 0.05$; # Post versus Follow Up: $p < 0.05$; † Follow Up versus Pre-training $p < 0.05$

7.7. The relationship between changes in exercise capacity and health outcomes

Table 7.4 shows the Pearson product-moment correlation coefficients between changes in variables from pre-training to post-training. The table shows that there exists a weak negative correlation between the change in relative VO₂max and change in body mass and lean mass ($r = -0.15$, $p > 0.05$ and -0.28 , $p > 0.05$). However, there was a moderately weak negative correlation between relative VO₂max and resting glucose concentration ($r = -0.41$, $p > 0.05$), as well as a moderately weak positive correlation between relative VO₂max and anaerobic threshold ($r = 0.41$, $p > 0.05$). The results also showed that there was a moderately strong positive correlation between the change in relative VO₂max ml/kg/min and the change in DBP ($r = 0.53$, $p < 0.02$ and $r = 0.55$, $p < 0.02$).

Table 7.6: Pearson product-moment correlations between changes in exercise capacity and health outcome measures after training from pre to post

Variable	ΔVO_{2max} ml/min/kg	ΔVO_{2max} L/min	ΔWL_{max}
Body Mass	-0.15	-0.09	-0.12
Fat Mass	0.28	0.22	0.00
Lean Mass	-0.28	-0.22	0.00
Waist Circumference (cm)	0.03	0.01	-0.25
Waist to Hip Ratio	-0.13	-0.11	-0.30
Glucose (mM/L)	-0.41	-0.33	-0.39
Cholesterol (mM/L)	0.36	0.40	-0.08
SBP (mmHg)	0.03	0.12	-0.05
DBP (mmHg)	0.53#	0.55#	-0.04
VO_{2max} (L/min)	-0.96	1.00	-0.34
VO_{2max} (ml/min/kg)	1.00	0.96	-0.25
Anaerobic Threshold	0.41	-0.35	0.41
Maximum Workload	-0.25	-0.12	1.00
SBP: systolic blood pressure; DBP: diastolic blood pressure; WL max: maximum work load			

*= $p < 0.05$; # = $p < 0.02$

Table 7.5 shows the correlations between changes in variables from pre-training to post-training. Moderate negative correlations exist between changes in relative VO_{2max} and changes in BM, and DBP ($r = -0.30$, $p > 0.05$ and $r = -0.30$, $p > 0.05$). The results also showed moderate correlations between changes in relative VO_{2max} and anaerobic threshold and maximum work load ($r = -0.40$, $p > 0.05$ and $r = 0.36$, $p > 0.05$).

Table 7.7: Pearson product-moment correlations between changes in exercise capacity and health outcome measures after detraining from post to follow up.

Variable	$\Delta\text{VO}_{2\text{max}}$ ml/min/kg	$\Delta\text{VO}_{2\text{max}}$ L/min	$\Delta \text{WL}_{\text{max}}$
Body Mass	-0.30	-0.16	0.13
Fat Mass (%)	0.14	0.09	0.49
Lean Mass (%)	-0.14	-0.09	-0.49
Waist Circumference (cm)	0.30	0.37	-0.08
Waist to Hip Ratio	0.13	0.17	0.05
Glucose (mM/L)	0.05	-0.06	0.25
Cholesterol (mM/L)	0.17	0.13	-0.14
SBP (mmHg)	-0.11	-0.02	-0.24
DBP (mmHg)	-0.30	-0.27	-0.08
$\text{VO}_{2\text{max}}$ (L/min)	0.94	1	0.20
$\text{VO}_{2\text{max}}$ (ml/min/kg)	1.00	0.94	0.36
Anaerobic Threshold	-0.40	-0.41	0.10
WL max	0.36	0.20	1.00
SBP: systolic blood pressure; DBP: diastolic blood pressure; WL max: maximum work load			

*= $p < 0.05$; # = $p < 0.02$

7.8. Response rate to HIIT intervention for the outcome variables

Waist circumference had the greatest improvement in response to the exercise intervention in that it reduced in 100% of the participants. Second to waist circumference was resting glucose levels (reduction towards the 4 mM/L norm) which improved in 90% of the participants following the HIIT intervention. This was followed by body mass, in which 85% of the participants lost weight, and reduced diastolic blood pressure.

Table 7.8: The number (and %) of participants who responded positively, negatively or had no change in health indicators after the HIIT intervention

Variable	Improvement %	N	Worse %%	N	Same %	N
Waist Circumference (cm)	100	20	0	0	0	0
Glucose (mM/L)	90	18	5	1	5	1
Body Mass	85	17	5	1	10	2
DBP (mmHg)	85	17	5	1	10	2
Body Mass Index	85	17	5	1	10	2
Cholesterol (mM/L)	80	16	15	3	5	1
SBP (mmHg)	75	15	10	2	15	3
Percentage Body Fat	75	15	15	3	10	2
Lean Mass	75	15	15	3	10	2
Waist to Hip Ratio	65	13	15	3	20	4

Table 7.7 indicates that FBLA improved the most in 75% of the subjects while VO_{2max} (ml/kg/min), maximum workload and VE_{max} improved in 70% of the participants following the HIIT intervention.

Table 7.9: The number (and %) of participants who responded positively, negatively or had no change in health indicators after the HIIT intervention

Variable	Improvement %	N	Worse %%	N	Same %	N
FBLA at 4mM/L	75	15	25	5	0	0
VO_{2max} (ml/min/kg)	70	14	30	6	0	0
WL max	70	14	5	1	25	5
VE max	70	14	30	6	0	0
Lactate Threshold mM/L	70	14	5	1	25	5
VO_{2max} (L/min)	45	9	45	9	10	2
Anaerobic Threshold	45	9	55	11	0	0

Chapter 8

DISCUSSION

8.1. INTRODUCTION

Faced with the global epidemic of obesity and overweightness, weight reduction seems to be the probable and integral answer in the prevention and treatment of the health diseases associated with obesity. Diseases like myocardial infarction, diabetes, hypertension, metabolic syndrome, varicose veins, angina pectoris and orthopaedic problems often coexist with obesity. However, regular exercise training has been shown to be a viable economic and non-pharmacological approach to improve health and assist in weight loss.

A number of studies have reported that the mitochondrial oxidative capacity in obese individuals is limited. This could be attributed to a reduction in mitochondrial size and alteration in mitochondrial structure (Houmard, 2007; Menshikova *et al.*, 2005; Kelly *et al.*, 2002; Bass *et al.*, 1975), as well as a reduced electron transport chain activity (Kim *et al.*, 2004; Ritov *et al.*, 2004). Moreover, a number of studies have also reported a reduction in enzymes involved in β -oxidation and the Krebs cycle. This has been linked to the pathogenesis of insulin resistance and subsequent metabolic and cardiovascular diseases. This reduction in enzymatic activity and mitochondrial size presents a challenge, which exercise physiologists have managed to address through the strategic implementation of exercise programs, such as endurance training and resistance training. HIIT has also emerged as a time efficient strategy which invokes the same physiological adaptations as that of endurance training and which enhance metabolic efficiency. These exercise strategies are paramount in our quest to combat the worldwide epidemic of obesity, insulin resistance and cardiovascular diseases.

This study envisioned investigating the effects of two weeks of HIIT training and detraining on health-related outcomes of young overweight/obese women. The main findings of this study were that two weeks of HIIT significantly improved the health-related outcomes of young women. This was shown by a decrease in body mass, resting glucose and cholesterol levels, resting blood pressure, as well as an increase in VO_{2max} . Although not the focus of this study, these improvements can probably be attributed to an increase in the muscle's oxidative capacity, circulatory function and rate of lactate removal. These results suggest that HIIT can be used as a vehicle of change in managing, maintaining and possibly reversing the health risk profiles of young women. Of paramount importance in this study is the effect of detraining on the health-related outcomes. The rapid gains accrued during HIIT were also rapidly lost following detraining which emphasises that exercise needs to be done continuously to prevent weight regain and maintain the improvements in VO_{2max} .

8.2. SUBJECT CHARACTERISTICS

The participants in this study were women and this choice was influenced by the fact that previous studies on health-related issues have only focused on men (Whyte *et al.*, 2010). According to the 2013 South African National Health and Nutrition Examination Survey (SANHANES-1) up to 40% of South African women are considered overweight or obese. This figure is alarming and rates among the highest in the world. For this reason more research should focus on women's health and the role of physical activity and exercise in the management of metabolic disease.

Furthermore, since most studies are limited to men, it cannot be assumed that these findings can be directly extrapolated to the female population. Some studies observed significant differences in the exercise responses of men and women (Astorino *et al.*, 2011), while others report significant differences. A case in point is Metcalfe *et al.* (2011) who reported a 28% change in insulin sensitivity in sedentary men and none in sedentary women after six weeks

of HIIT. In contrast, Richards *et al.* (2010) reported similar increases in insulin sensitivity (approximately 30%, $p = 0.04$) in men and women after two weeks HIIT (Wingate protocol). Astorino *et al.* (2011) also did not find any gender differences in the adaptations to $\text{VO}_{2\text{max}}$ between men and women after a training programme.

The age range for this study was 18 to 25 years. Most previous studies investigating the effects of HIIT have either focused on adolescents (Tjonna *et al.*, 2009, 2008), or adults older than 25 years (Trilk *et al.*, 2011; Whyte *et al.*, 2010). This age range (18 – 25 years) represents a transitional stage from adolescence to adulthood and is usually the time when children venture out on their own, as they go to universities or start employment. This greatly impacts on their dietary behaviors and physical activity levels. Some tend to consume more fast foods and are less involved in physical activity, as compared to being at school where physical education or participation in sport may still be compulsory. This results in a disturbance in the energy cycle whereby energy intake exceeds energy expenditure which results in a positive energy balance and which over time can lead to overweight and obesity. Moreover, university students often cite a lack of time as a barrier to their participation in physical activity, due to academic commitments like lectures and assignments.

Body mass index (BMI) and waist circumference were chosen as measures of adiposity for this study, because numerous studies have demonstrated its effectiveness in clinical diagnostics (Dudina *et al.*, 2011; Munk *et al.*, 2009; Klein-Platat *et al.*, 2005; Poirier and Despres, 2001). However, there are contradictory views on the use of BMI and waist circumference as diagnostic measures for adiposity (Wang *et al.*, 2005; Janssen *et al.*, 2004). Some are of the opinion that waist circumference is a better measure of indicating the risk profile for developing insulin resistance and hypertension as it measures the changes in intra-abdominal fat, while BMI only measures whole-body changes in weight. This is important because abdominal obesity is specifically associated with insulin resistance and metabolic

syndrome (Leung *et al.*, 2011; Wang *et al.*, 2005; Poirier and Despres, 2001; Lean *et al.*, 1995). However, Bray, (2004) contends that even though waist circumference is a better measure of adiposity, BMI has been recognized as the initial first step in assessing health risk by the American National Heart, Lung and Blood Institute (NHLBI, 1998). Moreover, BMI is also considered a good method of estimating the risk of cardiovascular disease and diabetes, whereas waist circumference is a good predictor of insulin resistance and the metabolic syndrome (Dudina *et al.*, 2011; Farin *et al.*, 2006; Bray, 2004; Hu *et al.*, 2004). As a result, both measures of adiposity should be used in estimating health risk profiles hence the use of both measures in this study (Dagan *et al.*, 2013; Farin *et al.*, 2006).

The BMI range for inclusion in this particular study was set at $> 25 \text{ kg/m}^2$. The rationale for this BMI range is its association with a higher risk of cardiovascular disease and chronic diseases such as diabetes (Kokkinos, 2012; Poirier *et al.*, 2006). Dudina *et al.* (2011) mentioned that a five –unit increase in BMI is associated with a 34% increase in cardiovascular disease mortality. A waist circumference $\geq 88\text{cm}$ was set as high risk for women (Kokkinos, 2012; Hans *et al.*, 1997). Waist circumference is of particular importance in that it is linearly related to insulin resistance and thus estimates the risk for insulin resistance better than other measures such as BMI (Janssen *et al.*, 2004). Moreover, waist circumference also shows greater improvements with exercise training compared to BMI and it was reasoned that it would be a better motivator for individuals looking to reduce weight, if they could observe obvious changes. Waist circumference is an easy method of indicating risk profiles and it can even be monitored by individuals themselves.

The outcomes of this study are compared to the findings by Racil *et al.* (2013), Hood *et al.* (2011), Whyte *et al.* (2010), Tjonna *et al.* (2009, 2008) and Buchan *et al.* (2010), who investigated the effects of HIIT interventions on health-related measures in different study populations.

Whyte *et al.* (2010) investigated the effect of HIIT on the health-related outcomes of overweight/obese men. Tjonna *et al.* (2008) and Tjonna *et al.* (2009) investigated the effect of HIIT in adolescents. Racil *et al.* (2013) investigated the effect of HIIT on the health parameters in obese young women over a 12 week period, while Tjonna *et al.* (2013) investigated the effect of 10 weeks of HIIT in sedentary overweight men. To the author's knowledge no study has investigated the effects of HIIT and detraining on the health-related outcomes of young overweight and obese women over a short period of two weeks.

8.3. BODY COMPOSITION

HIIT, an aerobic form of training, takes relatively shorter training time compared to continuous training; thus it is considered a time efficient strategy for inducing favorable adaptations in the body which reduces an individual's health risk profile. A reduction in the risk profile promotes good health and a better quality of life. The HIIT intervention in this study resulted in a mean weight loss of 1.6% ($p = 0.001$) which is considerable considering that this was obtained after only six sessions of training. This finding is in accordance with other HIIT studies on overweight and obese individuals, but that lasted longer (10 – 16 weeks) (Racil *et al.*, 2013; Tjonna *et al.*, 2013; Nybo *et al.*, 2010; Tjonna *et al.*, 2009; Trapp *et al.*, 2008). On the other hand, the results are in contrast to those of Whyte *et al.* (2010) who did not observe any changes in body weight after two weeks of HIIT training using the Wingate protocol. Perhaps the explanation for these inconsistent findings lies in the differences in exercise durations per session. In the study of Whyte *et al.* (2010) subjects performed all out sprints for a total time of two minutes in the first week and three minutes in the second week. In this study the high intensity bouts lasted 10 minutes in the first week and 15 min in the second week; thus five times more than that of Whyte *et al.* (2010).

Similarly, Burgomaster *et al.* (2006) reported no changes in fat oxidation following two weeks of Wingate tests (4-7 x 30 s all out sprint interspersed with 4min rest periods) which

had a total training time of 16 minutes of intense training. Talanian *et al.* (2007) reported a 36% increase in whole body fat oxidation following two weeks of HIIT (10x 4-min at 90% VO_{2max} interspersed by 2min of rest) in which seven sessions were done with a total of 40 minutes of intense exercise. Therefore the differences in exercise duration impact on the muscles' oxidative capacity, mainly fat oxidation capacity, which in turn contributes to weight loss. Moreover, differences can also be expected between running (this study) and cycling (Whyte *et al.*, 2010) protocols. Capastagno and Bosch (2010) reported that running elicits higher oxidation rates than that of cycling at the same intensities as it recruits more type 1 muscle fibers and involves larger muscle groups.

The weight loss observed after HIIT can also be attributed to the fact that exercise intensities greater than 90% of HR_{max} elicit greater energy expenditure after exercise (i.e. excess post-exercise oxygen consumption, EPOC) which create a negative energy balance and thus may have contributed to the weight loss observed in this study. This was demonstrated by King (2001) who reported an increase in 24 hour resting metabolic rate which accounts for 60 to 75% of total daily energy expenditure followings 8 weeks of interval training in obese women. Hazell *et al.* (2012) has shown that HIIT elicits 24 hr. EPOC equivalent to that of endurance training, despite much less training time. The HIIT protocol in this study consisted of 4 x 30-s all out sprints separated by 4-minute active recovery, while the endurance training consisted of 30 minute stationary cycling at 70% VO_{2max} . During EPOC there is a higher rate of fat oxidation which is also used to replenish muscle glycogen (Burns *et al.*, 2012; Boutcher, 2011; King, 2001). Higher total energy expenditure during training, as well as higher EPOC after training, will both contribute to higher fat oxidation rates and thus contribute to weight loss. It is also important to note that HIIT attenuates the feelings of hunger and expresses more effects of exhaustion (Imbeault *et al.*, 1997). This may have helped individuals to avoid overeating which is usually associated with light to moderate exercise when appetite can actually be stimulated after an exercise bout.

Another phenomenon that may explain the weight loss observed in this study is that of an increase in the resting energy expenditure (REE), which has been reported in the literature (Hunter *et al.*, 1998). The weight loss may occur because the increased REE observed after 24-48 hrs after an exercise bout is maintained if another exercise bout is repeated within the 24 to 48 hour period (Hunter *et al.*, 1998). Since the subjects in this study exercised every 48 hours during the week, it is reasonable to expect that their REE was increased for most of the week which would have helped to create a negative energy balance.

The reduction of fat mass of 3.7% and increase in lean mass of 1.9% observed in this study are explained by the fact that exercise (HIIT) induces weight loss through an increased EPOC and fat metabolism. This is contrary to popular belief that low intensity endurance training induces greater weight loss than any other type of exercise. Racil *et al.* (2013) reported an 8% decrease in percentage body fat in their HIIT group, while the endurance exercise group decreased percentage body fat by only 5%. Similarly, Macpherson *et al.* (2011) reported a 12.4% decrease in body fat and 1% increase in lean mass after 6 weeks of HIIT (Wingate protocol) in healthy recreational adults. The continuous training group showed a relatively smaller decrease in fat mass of 5.8% while the increase in lean mass was similar to the HIIT group (1%).

Tjonna *et al.* (2013) reported a 2% and 3% decrease in fat mass in groups who performed a single bout HIIT (1x 4 minute at 90% HR_{max}) and a four bout HIIT (4x4 minute at 90% HR_{max})(Table 3.4). King (2001) also reported a decrease in fat weight (-4.4%, $p<0.05$) in overweight women after interval training. The reduction of fat mass and increase in lean mass is explained by the concept that exercise (HIIT) induces weight loss through reduction of fat mass, and more specifically abdominal fat loss, and subsequently an increase in lean mass. The decrease in fat mass observed in this study was subsequently lower than that of Racil *et al.* (2013) and MacPherson *et al.* (2011). The difference can be explained by the different

exercise intervention periods which were 3 and 5 times longer than in the current study. The increase in lean mass observed in the current study and the abovementioned studies may also contribute towards an increase in energy expenditure as lean mass is more metabolically active than fat mass.

The results from the current and other HIIT studies show that HIIT is equally effective and even more superior in inducing whole body weight loss which is the main objective in the management of obesity and overweightness.

The most encouraging finding of the current study was that there were significant reductions in waist circumference in all participants, even though the intervention was very short. The reduction in waist circumference of 4.8% could have been a motivational factor for participants in the current study, as they could see greater changes compared to that of BMI (1.7%; $p = 0.001$) and total body weight loss (1.6%) (Lean *et al.*, 1995). This finding is concomitant with findings by Racil *et al.* (2013), Whyte *et al.* (2010) and Tjonna *et al.* (2008), who reported a 3.5%, 1.1% and 4.7% reduction in waist circumference in their studies, respectively. The reduction in abdominal fat after HIIT training was also confirmed by Trapp *et al.* (2008) who reported a 10.5% decrease in abdominal fat in women. The reduction in waist circumference, and therefore loss in abdominal fat mass, could be explained by the β adrenergic receptor found in the abdominal area. These β adrenergic receptors increase the lipolytic activity in the abdominal region resulting in the reduction of waist circumference.

The differences in the magnitude of adaptations in BMI and waist circumference suggest that HIIT is also more effective in reducing abdominal fat. This reduction in abdominal fat is of paramount importance in that waist circumference is linearly related to insulin resistance ($r = 0.78$ in men) (Tabata *et al.*, 2009). Moreover, large waist circumferences have also been associated with a 2.4 fold increased risk of developing hypertension (Leung *et al.*, 2011).

The waist circumference improved in 100% of sample in this study and this was also accompanied by a 35% increase in the number of people in the low risk category. Another important finding of the current study is that it showed weak negative correlations between changes in absolute VO_{2max} and changes in waist circumference from pre-training to post-training ($r = -0.11$). This then shows that the decrease in waist circumference occurs independently from changes in absolute VO_{2max} , as absolute VO_{2max} did not change statistically significantly following the HIIT intervention. The reduction in waist circumference, BMI and waist to hip ratio is related to a decrease in one's risk of developing insulin resistance, diabetes, dyslipidemia, hypertension, metabolic syndrome and cardiovascular disease (Goedecke *et al.*, 2006, Despres, 2001).

It is also important to note that HIIT attenuates the feelings of hunger and expresses more effects of exhaustion (Imbeault *et al.*, 1997). This may have helped individuals to avoid overeating which is usually associated with light to moderate exercise when appetite can actually be stimulated after an exercise bout. Some studies have showed that there was no significant change in post exercise food intake, and subjective levels of satiety and hunger (Imbeault *et al.*, 1997; Staten *et al.*, 1990; Kissilef *et al.*, 1990). King *et al.* (2007) reported that behavioural and metabolic compensatory responses may impede weight loss in weight loss programs. This could not have been the case in the current study as weight decreased following the intervention. This is also supported by findings by Sondike *et al.* (2003) who compared diet alone, exercise alone, and exercise without weight loss. The subjects in the exercise without weight loss group ate enough calories to equal the ones expended during the activity. The results from their study showed that the exercise without weight loss group did not lose weight while the exercise and diet groups lost weight. This then shows that if individuals do not compensate for food intake they can lose weight this is consistent with findings of the current study.

Despite the positive adaptations that were accrued during training, two weeks of detraining resulted in a total reversal of the loss in body mass and fat mass, and gain in lean mass. The reversal of these parameters following detraining has been observed in other studies as well and they attributed the deconditioning to a diminished oxidative capacity of the muscle (Orio *et al.*, 2008; Madsen *et al.*, 1993; Simoneau *et al.*, 1987). The enzyme 3 beta hydroxyacyl CoA dehydrogenase (HAD) which facilitates beta oxidation has been shown to decrease following a two to eight week detraining period (Amigo *et al.*, 1998; Bangsbo and Mizuno, 1988). In addition, the reversal of the maximal activities of the enzymes involved in the Krebs cycle and electron transport chain may also limit aerobic metabolism. Detraining decreases the enzymatic activities of lipoprotein lipase in the muscle thus limiting the availability of FFA for fat metabolism, while it increases this enzyme's activity in the adipose tissue. The increase in the activity of lipoprotein lipase in the adipose tissue results in more fat being stored which leads to a gain in weight.

8.4. RESTING GLUCOSE LEVELS

The reduction in glucose levels following training from a mean of 5.3 ± 0.43 to a mean of 4.7 ± 0.40 achieved in this study, can be attributed to the fact that HIIT increases the glucose transporter GLUT4 which increases the uptake of glucose into the cell and thus helping to lower glucose levels in the blood. The exercise independent insulin effect activates the translocation of GLUT4 by up to 10-fold, through signaling pathways. High levels of glucose in the blood increase one's risk of getting diabetes and cardiovascular disease and in extreme cases may cause organ failure (ADA, 2013).

Studies investigating the effect of HIIT on fasting glucose levels have reported contradictory results with some finding no decrease in fasting plasma glucose concentrations and others reporting changes in the fasting plasma glucose concentration. In contrast to the studies (Hood *et al.*, 2011; Whyte *et al.*, 2010; Babraj *et al.*, 2009) where lower resting glucose levels were

observed after HIIT, other HIIT interventions over two weeks did not observe changes in blood glucose levels (Hood *et al.*, 2011; Whyte *et al.*, 2010; Babraj *et al.*, 2009). Whyte *et al.* (2010) used the Wingate protocol in their study which consisted of a total of four to six minutes of very intense exercise over the two week period. However, in the current study the training duration for the high intensity work bouts was longer with a total exercise time of 10 to 15 minutes over the two week period. It is thus possible that the intensity and duration of exercise are critical factors that may determine whether glucose metabolism is altered or not.

The differences observed between the current study and those of Hood *et al.* (2011) and Babraj *et al.* (2009) could be attributed to differences in initial levels of fasting glucose. The participants in the current study had higher glucose levels at baseline compared to other studies (Hood *et al.*, 2011; Babraj *et al.*, 2009). In Hood *et al.* (2011) participants' baseline fasting glucose concentrations were 4.9 ± 0.3 mmol/l compared to the 5.3 ± 0.43 reported in this current study. It is important to note that Hood *et al.* (2011) reported a decrease in fasting glucose concentrations of 12.2% ($p = 0.09$), however, this was not statistically significant. Moreover, the protocol used by Hood *et al.* (2011) (10x1-minute cycling at 90% VO_{2max}) was similar to the one used in this study (10x1minute running at 90% VO_{2max}) in the first week. However the total training times in the second week was longer than in Hood *et al.* (2011) as the HIIT consisted of 15x 1minute runs at 90% VO_{2max} .

In contrast to the above studies where no changes in fasting blood glucose levels were found, the current study reported an 11% ($p = 0.001$) decrease in resting glucose levels. The decrease in fasting glucose observed in the current study is consistent with studies in which long intervention periods of 10- 16 weeks of HIIT training were used. Nybo *et al.* (2010) and Tjonna *et al.* (2008) reported a 9% and 4.3% decrease in fasting glucose after HIIT, respectively. Tjonna *et al.* (2013) found similar results using the two different HIIT protocols with the 1-AIT lasting one quarter of the time compared to that of the 4-AIT and resulting in

6% and 5% reduction in fasting blood glucose levels, respectively. The current study thus showed that HIIT done over a short period of time can also induce favorable changes in glucose profiles.

It is important to note that as little as one session of HIIT increases the activity of AMPK (1.5 fold) and PGC1- α (66%), which in turn increases GLUT4 activity (Bartlett *et al.*, 2012a; Little *et al.*, 2011; Gibala, 2009). Burgomaster *et al.* (2007) showed that 3 sessions of the Wingate Test protocol induced a 20% increase in GLUT4, while Hood *et al.*, (2011) demonstrated that two weeks of the Wingate intervention increased GLUT4 by 35% in sedentary men and women.

The increase in PGC1- α is consistent with findings by Hood *et al.* (2011) who reported a 35% and 56% increase in insulin sensitivity and PGC1- α , respectively, in overweight adults following two weeks of HIIT. Of particular interest is that Hood *et al.* (2011) reported a simultaneous increase of 260% in GLUT4 with only two weeks of training coupled with an increase in insulin sensitivity showing the potency of HIIT in increasing whole body glucose uptake and thus contributing towards the maintenance of glucose homeostasis. These findings are consistent with those of Babraj *et al.* (2009) who also reported a 37% increase in insulin sensitivity in healthy men following two weeks of HIIT (Wingate protocol).

All these results suggest that a two week HIIT intervention is adequate to stimulate important adaptations involved in glucose metabolism which will promote the uptake of glucose into the cells resulting in reduced fasting plasma concentrations (Racil *et al.*, 2013; Tjonna *et al.*, 2013; Nybo *et al.*, 2010). The reductions in glucose following training can be attributed to the fact that HIIT increases the glucose transporter GLUT4 which increases the uptake of glucose into the cell thus help lower glucose levels in the body. The increase in GLUT4 is also associated with an increase in insulin sensitivity.

Taylor *et al.* (1999) showed that binge eating in the evening can significantly increase fasting glucose levels in women. However this could not have been the case in the current study as glucose levels decreased by 11%. Moreover the fact that participants were asked to repeat the 24 hour diet record in the same order could have controlled for the effects of binge eating.

The two weeks of detraining resulted in a partial reversal of resting glucose levels, but it was still significantly lower than pre-training values. This could be explained in part by the retention in the activities of GLUT4 which have been shown to remain elevated even after one week (17.5%) and six weeks (13%) of training cessation (Burgomaster *et al.*, 2007). These findings suggests that the metabolic adaptations that are made in response to short-term training are relatively long-lasting and at least not completely reversed within two to six weeks of detraining.

8.5. RESTING CHOLESTEROL LEVELS

In this study HIIT improved the total cholesterol levels by 10.4% ($p = 0.01$). This reduction was consistent with findings by Racil *et al.* (2013), Tsekouras *et al.* (2008) and Tjonna *et al.* (2009) who reported a decrease in total cholesterol, as well as a decrease in LDL and an increase in HDLS. These changes were also consistent with findings from 7 – 16 weeks endurance interventions that have reported decreases in total cholesterol (Laaksonen *et al.*, 2000; Manson *et al.*, 1992; Weintraub, 1989).

The reduction in cholesterol levels in this study were due to exercise and not diet. The reason for this is that diet studies have stated that an improvement in lipid profile can be seen as early as 6 weeks with most using 12 weeks as a cutoff period (Sondike *et al.*, 2003; Noakes and Clifton, 2000; Flynn *et al.*, 1999; Warburg *et al.*, 1992). However the current study was undertaken within two weeks and according to the above mentioned studies this would be too little a time for diet to have an influence on lipid profile. This shows that the reduction in resting cholesterol observed in this study was due to exercise. To elaborate, on this the

detraining period even showed a reversal of the adaptations in cholesterol to pretraining values. The conclusion can then be made to say that the reduction in cholesterol levels is due to exercise and not diet.

Tjonna *et al* (2008) compared the effect of HIIT (4x4min at 90% HR_{max}) and endurance (47 minute runs at 70% HR_{max}) training in overweight adolescents after three months of training. They reported a 22% ($p < 0.05$) increase in HDL-C in the AIT group, while there was a 22% ($p < 0.05$) increase in HDL-C in the endurance training group. Racil *et al.*, (2013) investigated the effects of endurance training and HIIT over a 12 week period. They reported a 7% ($p < 0.05$) decrease in total cholesterol in the HIIT group, while no change was observed in the endurance training group. Moreover, LDL-C was decreased by 12.5% and 8% ($p < 0.05$) in the HIIT and endurance training group, while HDL-C were increased by 6.3% and 8.0% ($p < 0.05$) in the HIIT and endurance training group. The results from these studies demonstrate the viability of HIIT as a strategy for improving (high) lipid profiles which are responsible for the pathogenesis of cardiovascular disease and insulin resistance. These diseases are associated with a poor quality of life and high mortality rates. Of particular interest is the superiority of HIIT in improving total cholesterol levels despite HIIT involving much shorter training times.

The decrease in total cholesterol could be explained by the fact that aerobic training has been shown to increase HDL-C and lower the LDL-C (Mestek, 2009; Musa *et al.*, 2009). This increase in HDL-C is important in that HDLs have a scavenging role in the body of taking up LDL-C and transport it to the liver, where it is excreted from the body as bile. On the other hand, the fact that HDLs contain a substantially low amount of cholesterol (20% cholesterol) which is nearly half that of LDLs (50% of cholesterol) may result in the lowering of total cholesterol levels. It can therefore be assumed that the reduction in total cholesterol was a

result of an increase in HDLs which have less cholesterol content and also facilitate the excretion of LDL-C from the body (Wood and Haskell, 1979).

Brooks *et al.* (1995) also suggested that exercise reduces total cholesterol by increasing the maximal activity of LCAT and the increase in lipoprotein lipase activity. Furthermore exercise also reduces the amount of chylomicrons in the blood and which may also have contributed to a reduction in total cholesterol levels. It remains to be seen to what extent HIIT affects all the mechanisms involved in lipid metabolism and how it relates to fat metabolism dynamics with endurance and resistance exercise.

This study also showed weak positive correlations between changes in VO_{2max} and changes in cholesterol levels from pre-to post- training ($r = 0.36, p > 0.05$), and after detraining ($r = 0.17, p > 0.05$). This data then suggest that changes in cholesterol levels are not dependent on changes in aerobic fitness levels. Laaksonen *et al.* (2000) also reported a 4.9% decrease in total cholesterol following endurance training in type 1 diabetic men, with no concomitant changes in body mass. Laaksonen *et al.* (2000) then concluded that changes in cholesterol are also independent from changes in body mass. This was also consistent with the findings of the current study which showed weak positive correlations between changes in body weight and changes in cholesterol levels ($r = 0.31, p > 0.05$) and thus confirms that changes in lipid profile are not dependent on changes in body weight.

Total cholesterol levels remained lower than pre-training values after the two weeks of detraining. Previous studies have used longer periods of detraining (6.5 days to four months) which have shown to result in a loss of the training adaptations acquired after long endurance training (Ferguson *et al.*, 1999; Hardman *et al.*, 1998; Hardman *et al.*, 1994; Thompson *et al.*, 1984). The current study suggests that a shorter period of detraining retains the improvements in cholesterol observed after high intensity training. In addition, the favorable weight loss observed in participants could have prompted them to be more cautious with their diet and

thus may have contributed to the lower total cholesterol levels observed after the detraining period.

The decline in total cholesterol observed after two weeks of HIIT is remarkable considering that the time taken to complete the training periods was shorter than that of typical endurance training programmes. The reduction of total cholesterol in 80% of the participants shows the feasibility of HIIT as an intervention programme in the human population. This is particularly important as studies have shown that as slight a reduction of 1% in cholesterol levels reduces the risk of cardiovascular disease by 2-3% (Manson *et al.*, 1992).

The current study shows a 10% reduction in total cholesterol and on calculation this is associated with a 20-30% reduction in an individual's risk for developing cardiovascular diseases. Of particular interest is that detraining induces a partial loss of the adaptations acquired. Nevertheless it seems more viable that individuals continue to train so as to maintain and induce greater improvements in their health risk profile.

8.6. BLOOD PRESSURE

The improvements in SBP of 3.4% ($p = 0.001$) and DBP of 5.8% ($p = 0.001$) observed in the current study after the HIIT intervention are consistent with other studies which also reported decreases in resting BP (Liu *et al.*, 2012; Collier *et al.*, 2008; Hagberg *et al.*, 1989; Van Hoof *et al.*, 1989). Whyte *et al.* (2010) reported a 4% decrease in SBP following two weeks of HIIT (6 Wingate sessions). These findings were also similar to the current study as SBP decreased by 3.4%. This was also similar to the results of Tjonna *et al.* (2013) who reported a decrease of 7.1mmHg in SBP and 7.7 mmHg in DBP in the 1-AIT, respectively, whereas decrease of 2.1mmHg in SBP and 6.1mmHg in DBP were observed after the 4-AIT, respectively. This was consistent with the results from the current study as we reported 3mmHg and 4 mmHg decreases in SBP and DBP, respectively.

This is also similar to findings by Millen *et al.* (2013) who also reported a reduction of 5.6%, 8.5% and 7.2% in systolic, diastolic and mean arterial pressure respectively, following six weeks of training. Subjects were between the ages of 30 to 57 years, moderately active and were pre-hypertensive (overweight or obese). The training consisted of either continuous training (50 minutes at 60-75% VO_{2max}), or HIIT (4x4 min at 80-90% VO_{2max} interspersed by 3-min recovery at 50-60% VO_{2max}). They attributed the decrease in BP to a decrease in systemic vascular resistance. Nybo *et al.* (2010) conducted a study in untrained males and showed similar increases in SBP (8mmHg), after HIIT and endurance training, while differential results were observed in DBP (2mmHg and 5mmHg) after HIIT and endurance training, respectively. It is important to note that the HIIT protocol lasted 20 minutes, while the endurance protocol lasted 60 minutes; thus three times longer than the HIIT group. This then shows the effectiveness of HIIT in inducing arterial blood pressure improvements similar to that endurance training.

Whyte *et al.* (2010) attributed the reductions in BP to an increase in nitric oxide production which decreases vascular resistance. Fagard, (2006) suggested that the decrease in BP observed after exercise may be a result of a reduction in plasma renin and reduction in the activity of the autonomic nervous system activity and noradrenaline thus reduced systemic vascular resistance. The reduction in systemic vascular resistance results in a decrease in blood pressure. The reduction in blood pressure observed in this study shows the potency of HIIT in counteracting the pathogenesis of hypertension and cardiovascular disease. This is even supported Chobanian *et al.* (2003) who reported that a reduction as little as 2mmHg can significantly reduce the incidence of stroke (by as much as 6%), while coronary heart disease risk can be reduced by as much as 4%.

Rheaume *et al.* (2009) mentioned that there is a strong relationship between the amount of visceral adipose fat (VAT) and BP and he recommended that VAT becomes a clinical target

in the treatment and management of hypertension. The changes in BP observed in the current study of 3.4% and 5.8% in SBP and DBP can also be attributed to the decreases in visceral adipose tissue as measured by waist circumference (4.8%). This may then imply that the decrease in waist circumference induced by HIIT mediated a decrease in BP thus emphasising the effectiveness of HIIT in improving the risk profiles of hypertensive humans.

The two week cessation in training induced an increase in both SBP and DBP, although the values were still lower than pre-testing values. The findings of this study are similar to those of endurance training studies which employed longer endurance training interventions which were followed by long detraining periods (Motoyama *et al.*, 1995; Hagberg *et al.*, 1989; Martin *et al.*, 1986). The increase in DBP could be attributed to an increase in total peripheral resistance and a cessation of nitric oxide release which is vasodilator and results in a decrease in vascular resistance.

8.7. MAXIMAL AEROBIC CAPACITY

HIIT has been shown to elicit the same changes in VO_{2max} as that of endurance training, while some studies suggest that HIIT is even superior to endurance training in eliciting improvements in VO_{2max} (Racil *et al.*, 2013; Ciolac *et al.*, 2010; Moholdt *et al.*, 2009; Wallman *et al.*, 2009; Munk *et al.* 2009; Schjerve *et al.*, 2008; Wisloff *et al.*, 2007 Helgerud *et al.*, 2006).

The current study shows that two weeks of HIIT done by young overweight and obese women improved their aerobic capacity by 5.1% (relative VO_{2max}), while absolute VO_{2max} improved but the change was not statistically significant. The increase in relative VO_{2max} was similar to that observed by Astorino *et al.* (2012) who reported a 5.5% increase in VO_{2max} after two weeks of HIIT in young men and women. The change in relative VO_{2max} could not have been a learning as there were no changes in the absolute VO_{2max} . However, Gibala *et al.* (2006) did not observe any changes following two weeks of Wingate exercise bouts in healthy men.

Burgomaster *et al.* (2006) suggested that changes in $\text{VO}_{2\text{max}}$ is related to initial levels of fitness, meaning that subjects with low levels of fitness will have greater increases in $\text{VO}_{2\text{max}}$ following training. In this study, baseline $\text{VO}_{2\text{max}}$ was 27.8 ± 5.69 ml/kg/min, meaning participants had low levels of fitness. This may explain the change observed in relative $\text{VO}_{2\text{max}}$ following HIIT training in the current study.

The training induced an increase in absolute $\text{VO}_{2\text{max}}$ but this was not statistically significant. This can be attributed to the fact that the training period in the current study was relatively short with participants doing six sessions within two weeks. Talanian *et al.* (2007) observed a significant increase in $\text{VO}_{2\text{max}}$ in their study where they had seven sessions of HIIT and the work bouts consisted of 10x4-minute runs at 90% $\text{VO}_{2\text{max}}$. The total exercise durations between that of Talanian *et al.* (2007) and the current study could possibly explain the differences observed in absolute $\text{VO}_{2\text{max}}$. Endurance training has also been shown to increase $\text{VO}_{2\text{max}}$ as it takes a longer duration ranging from 40 to 90 minutes.

The current findings are also consistent with findings by Gibala *et al.* (2006) and by Burgomaster *et al.* (2006) who reported no change in absolute $\text{VO}_{2\text{max}}$ following two weeks of Wingate Tests. However, these studies reported an increase in the muscle's oxidative capacity as well as exercise performance. This is similar to the findings of this study as participants increased their maximum speed during the $\text{VO}_{2\text{max}}$ test. These findings indicate that HIIT did increase the participants' exercise tolerance. However, despite the changes in $\text{VO}_{2\text{max}}$ it is important to note that being physically active reduces one's risk of developing cardiovascular disease, diabetes and other chronic diseases. This is because physical inactivity has been shown to be part of the "big four" major risk factors for developing cardiovascular disease (Foss and Keteyian, 1998; Wilmore and Costill, 1988). This then implies that the participation of the people in the training program also helped reduced their health risk profiles as they increased their physical activity levels (So, 2013; Paffenbarger *et al.*, 1993).

An improvement in relative $\text{VO}_{2\text{max}}$ observed in this study is related to the weight loss observed in this study. The intensity of the HIIT induced a decrease in body mass through an increase in the muscle's oxidative capacity which has been shown to increase even after two weeks of HIIT training by changes in the maximal activities of both carbohydrate and fat metabolism (Hood *et al.*, 2011; Talanian *et al.*, 2007; Burgomaster *et al.*, 2007, 2005; Gibala *et al.*, 2006). This is particularly important as a number of studies have reported that in obese individuals the mitochondrial oxidative capacity is reduced. This could be attributed to a reduction in mitochondrial size and alteration in structure (Houmard, 2007; Kelly *et al.*, 2002; Bass *et al.*, 1975), as well as reduced electron transport chain activity (Kim *et al.*, 2004; Ritov *et al.*, 2004). It is thus suggested that the one minute work bouts in the current study, which was done at 90-95% HR_{max} , is a practical exercise model to use in sedentary overweight/obese individuals and that it would be sufficient to induce significant improvements in aerobic fitness as shown by an increase in exercise performance. It is important to note that the individuals in this study tolerated the exercise protocol very well and that no adverse events were experienced. HIIT then emerges as a solution to the shortcomings of endurance training as it takes a relatively short time thus proving to be a viable and time efficient strategy for improving people's cardiovascular risks profiles.

The total reversals in $\text{VO}_{2\text{max}}$ adaptations following detraining can be attributed to a decrease in stroke volume which has been shown to decrease rapidly within 12 days of detraining. The decrease in stroke volume can also be attributed to a decrease in blood volume which has also been shown to decrease rapidly following a detraining period. This is in accordance with other studies that have reported a reversal in $\text{VO}_{2\text{max}}$ after longer periods of detraining which are equal to the training period. The total reversal of the $\text{VO}_{2\text{max}}$ adaptations can also be explained in part by a decrease in the muscle's oxidative capacity, as illustrated in previous studies. This coupled with a reduction in blood flow to the active muscles result in a decrease

in the transport of oxygen and its extraction by the muscle and which would result in a decrease in maximal exercise capacity.

The maximal heart rate values of subjects in this study did not increase after HIIT training, despite the fact that individuals achieved a significantly higher workload at exhaustion compared to pre-training. This is consistent with the findings of Daussin *et al.* (2008) who also reported no changes in maximal heart rate values following HIIT training. This is consistent with the well-known fact that exercise does not necessarily change the maximal heart rate values during maximal exercise. Similarly detraining did not invoke any changes in maximal heart rate.

8.8. TIME TO REACH CRITICAL PERFORMANCE PARAMETERS

The HIIT programme induced an improvement in FBLA in 75% of the participants, while 70% of the participants showed an increase in relative $\text{VO}_{2\text{max}}$ (ml/kg/min), maximum workload (WL_{max}) and maximum minute ventilation. These changes confirm the improvements in aerobic fitness capacity that was induced by six sessions of HIIT. This is consistent with other studies that have reported an improvement in the time taken to reach the fixed blood lactate threshold after HIIT training (Millen *et al.*, 2003; Poole and Gaesser, 1985). The increase in maximum workload ($10.95 \pm 2.16 \text{ km/h}$ to $12.15 \pm 2.43 \text{ km/h}$, $p < 0.05$) shows that there was an improvement in exercise tolerance despite no changes observed in absolute $\text{VO}_{2\text{max}}$. This is similar to findings by Gibala *et al.* (2006) and Burgomaster *et al.* (2005) who reported an increase in time exercise capacity following 6 HIIT sessions.

Burgomaster *et al.* (2005) had recreationally active men and two women partake in a two week Wingate protocol HIIT training in which six sessions were done. They measured the participant's cycle endurance capacity before and after training by cycling to exhaustion. The results from their study showed that participants increased their endurance capacity by 100%. Gibala *et al.* (2006) reported a decrease in the time taken to complete a 750kJ and 50kJ trial

after HIIT of 10.1% and 4.1% respectively. The endurance training group also showed a decrease in the time taken to complete the 750kJ and 50kJ trial of 7.5% and 3.5% respectively. It is also interesting to note that the six HIIT sessions increased the time trial performance even better than endurance training.

The improvement in exercise tolerance could possibly be explained by changes in lactate dynamics, and primarily as a result of an increase in the lactate transporters (MCT 1 and MCT2) as suggested by Burgomaster *et al.* (2007). Perry *et al.* (2008) also reported an increase MCT2 and MCT4 of 14% and 16%, respectively, following six weeks of HIIT training. An increase in these transporters facilitates an increase in the lactate removal rate from the muscle, thus extending the time taken to reach FBLA.

Furthermore, time to exhaustion can also be improved through the increase in the muscle's oxidative capacity which has been reported to increase greatly even after only two weeks of HIIT (Hood *et al.*, 2011; Burgomaster *et al.*, 2007, 2005; Gibala *et al.*, 2006). The up regulation of mitochondrial enzymes of the Krebs cycle, β -oxidation, and electron transport chain following HIIT results in more energy being available for use thus limit the onset of anaerobic metabolism which yields lactate as a by-product. The increase in enzymes of aerobic metabolism therefore results in an increase in substrate availability and the muscle's reliance on aerobic metabolism (which does not yield large amounts of lactate as a by-product) and ultimately increases exercise capacity.

Moreover, the increase in blood flow associated with exercise training can be another factor which resulted in an increase in the time taken to reach FBLA. This is because an increase in blood flow facilitates a greater rate of lactate removal from the active muscle into the bloodstream where it can be taken up by different muscles and tissues (pancreas, kidneys, liver, brain) and where it is converted to glucose or used for energy production (Astrand *et al.*, 2003). Moreover, the greater blood flow facilitates a greater diffusion surface for the

exchange of gases, thus increasing oxygen availability to the active muscles and facilitating aerobic metabolism. This then limits the accumulation of lactate in the active muscle resulting in an increase in the time taken to reach FBLA and anaerobic threshold.

Another factor that may have contributed to an increase in the time taken to reach lactate thresholds could be an increase in the maximal activity of the enzyme lactate dehydrogenase (LDH) which has been shown to increase after HIIT (Kohn *et al.*, 2011). The increase in LDH facilitates glycolysis which helps in the overall contribution to substrate utilization and thus increase exercise capacity. LDH is used in the absence of oxygen to convert pyruvate into lactate which enables the re-oxidation of NADH to NAD, thus helping in the continuous production of energy by glycolysis during the high intensity work bouts. During the recovery periods it helps convert the lactate into pyruvate which can be used for aerobic metabolism. This plays a vital role in maintaining substrate availability and thus enhances both the exercise capacity and delay the onset of blood lactate accumulation.

Thus, a number of mechanisms, both central and peripheral, may explain the increase in the time taken to reach FBLA and VO_{2max} , as well as the increase in maximum workload observed during the VO_{2max} test. This was similar to the findings of Millen *et al.* (2013) who also reported an increase in the time taken to reach VO_{2max} after HIIT in overweight/obese individuals.

Detraining also resulted in a complete reversal in the time taken to reach FBLA which indicates that the possible peripheral changes that were made to enhance lactate removal, was rapidly lost. This assertion is supported by Burgomaster *et al.* (2007) who showed a decrease in MCT4 of approximately 43% and a decrease in MCT1 of approximately 80% after one week of detraining. This then shows that without the training stimulus there is a loss of the gains that were obtained during training. Moreover the decrease in oxidative enzymatic activity decreases the muscle's oxidative capacity and substrate utilization which also result in

a decrease in exercise capacity ($W_{L_{max}}$). This was also shown by Burgomaster *et al.* (2007) who reported a decline in COX4 following detraining.

8.9. CONCLUSIONS

The main research aim of this study was to investigate the training and detraining effects of a short-term HIIT programme on selected health-related measures in young overweight and obese women. The main finding of this study is that as short a period of two weeks of HIIT can elicit adaptations that can lower the risk profiles of developing cardiovascular diseases, hypertension, diabetics and insulin resistance in young overweight women. This then confirms the fact that physical activity is associated with a lower mortality risk. Moreover it is important to note that although the HIIT programme lasted a short period of time it resulted in a loss of weight which is a major target in lowering the prevalence of overweightness and obesity.

An interesting finding in this study is that two weeks of HIIT resulted in an improvement in health-related outcomes similar to those observed by Racil *et al.* (2013) in overweight adolescents after 12 weeks of HIIT training. This is important in that these results may indicate that adaptations occur at an early period of training and that higher and longer intensities elicit positive improvements in an individual's risk profile. Moreover this research provided an insight into the effects of training and detraining in young women, whereas most studies have focused on men, or middle aged individuals, or adolescents. The running HIIT does not require any specialised equipment and coupled with this is the fact that running is an inborn ability and has been reported to be more enjoyable than endurance training thus may help increase exercise adherence which is essential for the maintenance of low health risk profiles.

The current study provides an insight into the time course adaptations acquired after a short period of training seeing HIIT's emergence as a time efficient strategy in eliciting positive

adaptations in clinical populations and healthy people. On the one hand this study demonstrates that a short period of training elicits an improvement in health risk profiles of young overweight and obese women that are similar to that of endurance training, despite the differences in total exercise duration times. Of particular interest is the effect of HIIT on abdominal fat, as it induced a significant loss in waist circumference which is linearly associated with insulin resistance, metabolic syndrome and cardiovascular diseases and which often coexists in obese people. Moreover, the increase in the time taken to reach FBLA and VO_{2max} , as well as the increase in maximum workload observed during the VO_{2max} test involves both central and peripheral adaptations.

On the other hand, a short period of detraining can induce partial (glucose, diastolic blood pressure, waist circumference and cholesterol) and a total reversal in parameters of health risk markers (VO_{2max} , weight and systolic blood pressure). These findings suggest that the metabolic adaptations that are made in response to short-term training are relatively long-lasting and at least not completely reversed within two to six weeks detraining. This is of paramount importance because the increase in the health risk profiles also increases the risk of developing cardiovascular disease, diabetes, insulin resistance, metabolic syndrome, hypertension and other chronic diseases. Research has shown that these diseases are associated with higher rates of morbidity and mortality as well as increasing the financial burden on the overall health system and government.

To conclude, the findings suggest that the two weeks of HIIT induced a reduction of body mass and fat mass while increasing lean mass, as well as reducing resting glucose and cholesterol levels, blood pressure and improves exercise capacity in young overweight and obese women. These improvements were made in response to short-term training and are relatively long-lasting and at least not completely reversed within two to six weeks detraining.

From this study it can be concluded that 10 min and 15 min HIIT at near-maximal intensities are possibly the minimum amount of training that is needed to induce significant weight loss, although it would be worthwhile to determine the optimum duration of high intensity exercise sessions in future studies that are both effective and tolerable. This is important seeing that overweightness and obesity have become a global epidemic in which one of the solutions is a loss of fat mass. This would ultimately reduce risk profiles, improve quality of life and reduce the morbidity and mortality rates associated with obesity and also relieve the financial burden on the health systems and government. Moreover this study lays a foundation in which other studies can build on to determine how much HIIT is needed to maintain the improvements shown after a HIIT programme so as to prevent the loss of adaptations accrued during HIIT.

8.10. LIMITATIONS OF THE STUDY.

The limitation to this study is that a small sample size was used and was done in young female university students thus the results from this study cannot be generalised to a broader community of women between 18 and 25 years old. In addition HDL-C, LDL -C, triglycerides and insulin sensitivity were not measured. The measurement of the above mentioned variables could have helped in the explanation of the effects of HIIT on the metabolic profile. Moreover, in this study we did not record daily food intake which may have also changed as participants started to realise positive adaptations in the body, however no nutritional advice was given to participants during this period. However as a way of controlling food intake participants were asked to write down a one day dietary record during baseline measurements in the fasting state and they were asked to repeat this during the test times for post training and follow up training. This helped to control for the fluctuations in blood cholesterol and glucose levels that can be induced by diet.

Another limitation in this study is the lack of a control group. However, it must be remembered that the primary aim of this study was to quantify the detraining effects after

HIIT training so as to determine the duration of the adaptations elicited by HIIT. Participants then acted as their own control. The purpose was never to show that HIIT is more effective than another type of exercise programme, or better than a diet to induce health-related benefits.

8.11. RECOMMENDATIONS

Future studies should investigate the effects of the two week intervention and take dietary records of participants. Moreover the fact that this study showed an improvement in the health profiles of young women more studies need to be done on a larger sample size to show the extent of the adaptations to HIIT over a short period of time. In addition studies need to investigate the effect of HIIT on the lipid profile so as to get a better understanding of the mechanisms involved in the reduction of lipid profiles observed after HIIT.

It is recommended that an optimum intensity that can elicit an improvement in body weight be established for obese individuals. Future studies also need to combine diet and exercise seeing as a combination of aerobic exercise and dieting has shown to produce more beneficial effects than exercise alone. This could create an ultimate programme that is time efficient and is well tolerable by people.

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APPENDIX A

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	
<input type="checkbox"/>	<input type="checkbox"/>	1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
<input type="checkbox"/>	<input type="checkbox"/>	2. Do you feel pain in your chest when you do physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	3. In the past month, have you had chest pain when you were not doing physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
<input type="checkbox"/>	<input type="checkbox"/>	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?
<input type="checkbox"/>	<input type="checkbox"/>	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
<input type="checkbox"/>	<input type="checkbox"/>	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.



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APPENDIX B

EXERCISE RISK ASSESSMENT

Name _____ Gender _____ Age _____

Email address _____

Phone _____

Please provide the following as accurately and completely as possible so that it can be used to assess your cardiovascular exercise risk.

KNOWN CARDIOVASCULAR, PULMONARY, OR METABOLIC DISEASE

Have you been diagnosed with any of the following diseases/disorders/conditions or undergone any of the following procedures?

- | | |
|----------------------------------------------------------|--------------------------------------------------------------|
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Myocardial infarction (“heart attack”) _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Stroke or ischemic attack (“mini stroke”) _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Heart bypass surgery or other heart surgery _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Coronary catheterization and/or angioplasty _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Abnormal ECG (tachycardia, heart block, etc.) _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Other cardiovascular disease/disorder (aneurysm, etc.) _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Chronic obstructive lung disease (asthma, COPD, etc.) _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Diabetes (insulin dependent, non- insulin dependent) _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Hyperlipidaemia (high LDL, low HDL, etc.) _____ |

Comment: _____

SIGNS OR SYMPTOMS SUGGESTIVE OF CARDIOVASCULAR AND PULMONARY DISEASE

Have you experienced any of the following?

- | | |
|----------------------------------------------------------|------------------------------------------------------------|
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Pain/discomfort in your chest, jaw, or arms _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Shortness of breath at rest or mild exertion _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Dizziness or fainting spells _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Difficulty breathing while lying down _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Swelling of your ankles _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Skipped heartbeats or racing heartbeat _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Occasional leg pain, especially while walking _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Heart murmur _____ |
| <input type="checkbox"/> Yes <input type="checkbox"/> No | Fatigue or shortness of breath with usual activities _____ |

Comment: _____

RISK FACTORS OF CARDIOVASCULAR DISEASE

Do you have a personal history of the following?

- Yes No Cigarette smoking: packs/day _____ . years smoked _____
- Yes No Obese or highly overweight: body weight _____
- Yes No Physical activity _____
- Yes No High blood pressure (SBP >140, DBP > 90). BP _____ mmHg
- Yes No High cholesterol (total > 200, LDL > 130): total _____ . LDL _____ mg/dl
- Yes No Diabetes or high glucose (> 110): blood glucose _____ mg/dl
- Yes No Family history of heart attack/stroke at young age: _____

Comment: _____

DRUGS/MEDICATIONS

Please list any prescription or over-the-counter drugs/medications you are currently taking.

Drug/medication	Purpose/reason for taking
_____	_____
_____	_____
_____	_____

CLASSIFICATION OF EXERCISE RISK (ACSM GUIDELINES)

- Low Risk:** Free of cardiovascular, pulmonary, metabolic disease; and free of any signs or symptoms of cardiovascular disease; and possess no more than 1 major risk factor of cardiovascular disease; and male \leq 45 y, female \leq 55y
- Moderate Risk (age):** Free of cardiovascular, pulmonary, metabolic disease; and free of any signs or symptoms of cardiovascular disease; and possess no more than 1 major risk factor of cardiovascular disease; and male \leq 45 y, female \leq 55y
- Moderate Risk:** Free of cardiovascular, pulmonary, metabolic disease; and free of any signs or symptoms of cardiovascular disease; regardless of age; possess 2 or more major risk factor of cardiovascular disease
- High Risk:** Regardless of age; diagnosed of cardiovascular, pulmonary, metabolic disease; and free of any signs or symptoms of cardiovascular disease

Participants in **low risk** category can participate in maximal intensity exercise with little risk of cardiovascular problem (e.g., arrhythmia, etc.). It is not necessary that they get medical clearance before participating in exercise or any lab test.

Participants in the **moderate risk** category have somewhat higher risk of experiencing cardiovascular problems with vigorous (60% VO_2 max) maximal exercise intensity. ACSM recommends anyone in the moderate risk category get medical clearance before vigorous exercise. Lower intensity exercise (< 60% VO_2 max) poses less cardiovascular risk and can be done without prior medical clearance.

ACSM recommends that participants in the **high risk** category get medical clearance before participating in any type of exercise test or program.

Reference: American College of Sports Medicine. (2006). *ACSM's guidelines for exercise testing and prescription* (7th ed.): Philadelphia: Lippincott William & Wilkins.

IN CASE OF EMERGENCY

Name _____ Phone _____

APPENDIX C



UNIVERSITEIT • STELLENBOSCH • UNIVERSITY
jou kennisvennoot • your knowledge partner

STELLENBOSCH UNIVERSITY CONSENT TO PARTICIPATE IN RESEARCH

THE EFFECT OF HIGH INTENSITY INTERVAL TRAINING ON THE HEALTH-RELATED OUTCOMES IN YOUNG WOMEN.

You are asked to participate in a research study conducted by Privilege B. M. Ndlovu (BSc Honors in Sport Science and Coaching), from the Department of Sport Science at Stellenbosch University. This research project is part of Masters in sport science thesis. You were selected as a possible participant in this study because you are a young woman between the age of 18-25 years.

1. PURPOSE OF THE STUDY

To investigate the training and detraining effects of a short-term HIIT programme on selected health-related measures in young women

2. PROCEDURES

If you volunteer to participate in this study, we would ask you to do the following things:

2.1 PRETESTING -which includes the measurement of body composition (body mass height, percentage body fat, lean mass), waist and hip circumferences, glucose, cholesterol, blood pressure, VO_{2max} and blood lactate concentration.

2.2 HIIT INTERVENTION PROGRAM- which takes two weeks and includes six sessions of HIIT. The training sessions will consist of 10-15 repeated work bouts of sprint running on a treadmill. It commences with a five minute warm up at a running velocity corresponding to 70 % VO_{2max} followed by (10-15)one-minute work bouts at a running velocity corresponding to 90- 95% VO_{2max} . The bouts will be separated by one-minute active recovery periods at a running velocity corresponding to 50% VO_{2max} . The last recovery period is followed by a cool down period at a running velocity corresponding to 50% VO_{2max} . for five minutes at The number of bouts will be increased from 10 (sessions one, two and three) to fifteen (session four, five and six)

2.3 POST TESTING will be done within 48 hours of the last HIIT session, following the baseline protocols and procedure.

DETRAIN-stop the HIIT program and not exercise for two weeks.

2.1 FOLLOW UP TESTING will be done using the same protocols and procedure. All testing and HIIT program will be done in the Sport Physiology lab during August to September.

3 POTENTIAL RISKS AND DISCOMFORTS

HIIT on a treadmill can be uncomfortable and tiring. You may have muscle soreness the next day. However, it would not be anything more than what you would experience when you do some other exercise for the first time.

4 POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY

The training program will increase your fitness level and might help reduce weight. As well as reduce the risk of cardiovascular diseases, hypertension, type 2 diabetes, colon cancer, breast cancer, osteoporosis and several psychological disorders. It is also a time efficient strategy to induce adaptations associated with traditional endurance training, hence promotes health, quality of life and well being. To the society at large this study can be used by companies and organizations as an intervention during working hours to promote health.

5 PAYMENT FOR PARTICIPATION

There will be no payments to participants.

6 CONFIDENTIALITY

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission or as required by law. Confidentiality will be maintained by means of giving participants numbers to identify them so no one will be identified using their names. The data collected will be stored in a computer which is password protected and only the researcher will have access to it. This computer will be locked in an office.

Participants will be given individual reports after the intervention. Whilst the group results of the study will be published in a scientific journal and confidentiality will be maintained in that no participant or the university's name will be mentioned. Furthermore the department will keep the data for a period of five years.

7 PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may also refuse to answer any questions you don't want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so. This may happen if a participant gets ill, injured or if there are any adverse effects.

8 IDENTIFICATION OF INVESTIGATORS

If you have any questions or concerns about the research, please feel free to contact Privilege B. M. Ndlovu (Principal investigator), +27 74 1070865 . E-mail address: 17448263@sun.ac.za. Prof E. Terblanche (Supervisor) +27 21 808 4817. E-mail: et2@sun.ac.za.

9 RIGHTS OF RESEARCH SUBJECTS

You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. If you have questions regarding your rights as a research subject, contact Ms Maléne Fouché [mfouche@sun.ac.za; 021 808 4622] at the Division for Research Development.

SIGNATURE OF RESEARCH SUBJECT OR LEGAL REPRESENTATIVE

The information above was described to _____ by Privilege B.M Ndlovu in English and I am in command of this language. I was given the opportunity to ask questions and these questions were answered to my satisfaction.

I hereby consent voluntarily to participate in this study. I have been given a copy of this form.

3. Name of Subject/Participant

Name of Legal Representative (if applicable)

4. Signature of Subject/Participant or Legal Representative **Date**

SIGNATURE OF INVESTIGATOR

I declare that I explained the information given in this document to _____. She was encouraged and given ample time to ask me any questions. This conversation was conducted in English and no translator was used.

1. Signature of Investigator

Date

APPENDIX D**DATA SHEET**

Name		Surname	
Age		E-mail address	
Date of Birth		Mobile contact	

1	Blood Pressure (BP)			
---	---------------------	--	--	--

Anthropometric Measurements

2	Weight (kg)	
3	Height (cm)	
4	Waist Circumference (cm)	
5	Hip Circumference (cm)	
6	% Body Fat	
7	Fat Free Mass (FFM)	
8	Total Body Weight (TBW)	
9	BMR	
10	BFMI	
11	Body Mass Index (kg/m ²)	
12	Waist Hip Ratio	
13	FFMI	

13	Resting Glucose	
14	Resting Cholesterol	
15	VO ₂ max	
16	Speed(km/hr)	
17	Gradient %	
18	Time to exhaustion	
19	Lactate	

STAGE	TIME min	NCLINE %	SPEED Km/hr	LACTATE
Resting				
BP				
1	3	0	6	
2	3	0	7	
3	3	0	8	
4	3	0	9	
5	2	1	10	
6	2	1	11	
7	2	1	12	
8	1	2	13	
9	1	2	14	
10	1	2	15	