

THE EFFECT OF HIGH INTENSITY INTERVAL TRAINING ON
THE POST-EXERCISE HYPOTENSIVE RESPONSE IN
OVERWEIGHT/OBESE YOUNG WOMEN

by
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DECLARATION

By submitting this thesis, I declare that the entirety of the work contained therein is my own, original work, that I am the owner of the copyright thereof (unless to the extent explicitly otherwise stated) and that I have not previously in its entirety or in part submitted it for obtaining any qualification.

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SUMMARY

There are extensive literature on the PEH response after acute and chronic aerobic and resistance exercise, as well as a few studies on concurrent and water exercise. However, there is comparatively little evidence that high intensity interval training (HIIT) elicits similar post exercise blood pressure reductions (PEH) compared to other types of exercise. Furthermore, it is difficult to quantify the magnitude of the hypotensive response following these exercises, due to variations in exercise protocols in terms of intensity and duration. Both these training variables are considered important determinants of the magnitude and duration of the PEH response.

The current study determined the magnitude of the PEH response after an acute bout and six sessions of HIIT, and the effects after two weeks of detraining in overweight/obese young women.

Twenty young women (aged 21 ± 2 years) volunteered for the study. All the subjects were normotensive (SBP: 119.2 ± 5.6 mmHg and DBP: 78.8 ± 4.1 mmHg). Subjects performed six sessions of HIIT within two weeks and detrained for two weeks. SBP, DBP, MAP and HR were monitored during seated recovery after exercise for 60 min to determine the change from resting values.

The overall outcome showed that an acute HIIT session resulted in a reduction of 2.9 mmHg in SBP which approached near clinical significance, while six sessions of HIIT caused a clinically significant reduction of 5.3 mmHg; this response was almost totally reversed after detraining. There were no clinically significant reductions in DBP after the acute or six sessions of HIIT (1.7 and 2.7 mmHg, respectively). However, a clinically significant hypotensive response of 3.9 mmHg was sustained after detraining following the maximal exercise capacity test. MAP also reduced by a magnitude of 2.3 and 5.6 mmHg, respectively,

after the acute bout and six sessions of HIIT, and detraining values were still 2.9 mmHg lower than resting values and approached near clinical significance.

The results indicate that both an acute bout and six sessions of HIIT elicited a meaningful PEH response. However, the six sessions of HIIT caused a clinically significant reduction which was approximately twice the acute session. Likewise, detraining showed clinically significant effects in DBP and MAP, but SBP returned to near baseline values. This suggests that in only two weeks, the accumulated effects of six sessions of HIIT elicited a greater hypotensive response than after an acute session of HIIT.

OPSOMMING

Daar is omvattende literatuur oor die post-oefening hipotensie (POH) na afloop van akute en kroniese aërobiese en weerstandsoefeninge, asook enkele studies oor gelyktydige krag- en uithouvermoë- en wateroefeninge. Daar is egter relatief min bewyse dat hoë intensiteit interval oefening (HIIO) soortgelyke post-oefening afnames in bloeddruk (POH) in vergelyking met ander tipes oefening veroorsaak. Voorts is dit moeilik om die omvang van die hipotensiewe respons na afloop van oefening te kwantifiseer, hoofsaaklik as gevolg van die variasies in oefeningprotokolle in terme van intensiteit en tydsduur. Beide hierdie inoefeningveranderlikes word as belangrike determinante van die omvang en die tydsduur van die POH respons beskou.

Die huidige studie het die omvang van die POH respons na 'n akute sessie en ses sessies HIIO, en die gevolge na afloop van twee weke se nie-inoefening (“detraining”) by oorgewig/vetsugtige jong dames, bepaal.

Twintig jong dames (ouderdom 21 ± 2 jaar) het vrywillig ingestem om aan die studie deel te neem. Al die deelnemers was normotensief (SBD: 119.2 ± 5.6 mmHg en DBD: 78.8 ± 4.1 mmHg). Die deelnemers het ses sessies HIIO binne twee weke voltooi en het daarna vir twee weke geen inoefeningsessies gehad nie. SBD, DBD, GAD en HS is tydens 'n sittende herstelfase vir 60 minute gemonitor om die verandering vanaf rustende waardes te bepaal.

Die algehele uitkoms toon dat 'n akute HIIO sessie 'n afname van 2.9 mmHg in SBD tot gevolg gehad het wat aan kliniese betekenisvolheid grens, terwyl ses sessies van HIIO 'n klinies betekenisvolle afname van 5.3 mmHg veroorsaak het; hierdie respons wat bykans volledige omgekeerd na die twee weke met geen inoefening. DBD het geen kliniese betekenisvolle afname na afloop van die akute of ses sessies van HIIO getoon nie (1.7 en 2.7 mmHg, respektiewelik). 'n Klinies betekenisvolle hipotensiewe respons van 3.9 mmHg is

egter gevind na die geen inoefeningsperiodes. GAD het ook met 'n omvang van 2.3 en 5.6 mmHg, respektiewelik, verminder na afloop van die akute sessie en ses sessies van HIIO. Die geen inoefening waardes was steeds 2.9 mmHg laer as die rustende waardes en het aan kliniese betekenisvolheid gegrens.

Die resultate toon dat beide 'n akute sessie en ses sessies van HIIO 'n betekenisvolle POH respons ontlok het. Ses sessies van HIIO het egter 'n klinies betekenisvolle afname, wat ongeveer twee keer soveel as die afname van die akute sessie was, veroorsaak. In dieselde lig het 'n twee weke geen inoefeningsperiode steeds klinies betekenisvolle veranderinge in DBD en GAD getoon, maar SBD het tot naby aan die basislyn waardes teruggekeer. Hierdie resultate suggereer dat in slegs twee weke die geakkumuleerde effekte van ses sessies van HIIO 'n groter hipotensiewe respons as na 'n akute sessie van HIIO ontlok het.

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DEDICATION

I dedicate this thesis to my grandmother Grace Obeng.

You always wanted the best in education for me.

LIST OF ABBREVIATIONS AND ACRONYMS

°	:	Degree
°C	:	Degrees Celsius
~	:	About
≈	:	Approximately equal to
%	:	Percentage
%BF	:	Percentage body fat
>	:	Greater than
≥	:	Greater or equal to
<	:	Less than
≤	:	Less or equal to
±	:	Plus-minus
Δ	:	Change in
1RM	:	One repetition maximum
10RM	:	Ten repetition maximum
1-MET	:	One metabolic equivalent of task
ACE	:	Angiotensin converting enzyme
ACE DD	:	DD genotypes of the ACE gene
ACE II/ID	:	II/ID genotype of the ACE gene
ACSM	:	American College of Sports Medicine
ANOVA	:	Analysis of variance
ANP	:	Arterial natriuretic peptide
ATP	:	Adenosine triphosphate
BIA	:	Bio-electrical impedance analysis

b.min ⁻¹	:	Beats per minute
β - HAD	:	3-hydroxyacyl CoA dehydrogenase
BMI	:	Body mass index
BP	:	Blood pressure
CHO	:	Carbohydrate
cm	:	Centimeter
CO	:	Cardiac output
CO ₂	:	Carbon dioxide
COX	:	Cytochrome c oxidase
COX2	:	Cytochrome c oxidase subunit 2
COX4	:	Cytochrome c oxidase subunit 4
CoA	:	Coactivator
CPK	:	Creatine phosphokinase
CS	:	Citrate synthase
CVD	:	Cardiovascular diseases
DBP	:	Diastolic blood pressure
e.g.	:	For example
etc.	:	And so on
ET-1	:	Endothelial-1
EPOC	:	Excess post-exercise oxygen consumption
GLUT4	:	Glucose transporter isoform 4
g.min ⁻¹	:	Gram per minute
h	:	Hour(s)
HIIT	:	High intensity interval training
HR	:	Heart rate

HR_{max}	:	Maximum heart rate
HRR	:	Heart rate reserve
i.e.	:	That is
Kcal	:	Kilocalories
kg	:	Kilogram(s)
kHz	:	Kilo hertz
$kg.m^{-2}$:	Kilogram per square meter
$km.h^{-1}$:	Kilometers per hour
L	:	Liter
LSD	:	Least Significant Difference
MAP	:	Mean arterial pressure
ml	:	Milliliters
$ml.b^{-1}$:	Milliliters per beat
$ml.kg^{-1}.b^{-1}$:	Milliliters per kilogram per beat
$ml.kg^{-1}.min^{-1}$:	Milliliters per kilogram body weight per minute
$ml.min^{-1}$:	Milliliters per minute
mm	:	Millimeter
mmHg	:	Millimeters mercury
mm/MJ	:	Millimeter per mega joule
$mmol.L^{-1}$:	Millimol per liter
$mmol.min^{-1}.kg^{-1}$:	Millimol per minute per kilogram
$mmol.min^{-1}.kg^{-1}DW$:	Millimol per minute per kilogram dry weight
N_2	:	Nitrogen
Na^+	:	Sodium ion
NaCl	:	Sodium chloride

ng.ml ⁻¹ .h ⁻¹	:	Nanogram per milliliters per hour
NO	:	Nitric oxide
O ₂	:	Oxygen
PDH	:	Pyruvate dehydrogenase
PEH	:	Post exercise hypotension
PGC-1 α	:	Peroxisome proliferator-activated receptor γ coactivator -1 α
pg.m ⁻¹	:	Picogram per meter
PPO	:	Peak power output
PTS	:	Peak treadmill speed
R	:	Respiratory quotient
RMR	:	Resting metabolic rate
RPE	:	Rate of perceived exertion
SA	:	Sino atrial
SBP	:	Systolic blood pressure
SD	:	Standard deviation
SNA	:	Sympathetic nervous system activity
SSE	:	Steady-state exercise
SV	:	Stroke volume
TDEE	:	Total daily energy expenditure
TPR	:	Total peripheral resistance
Type I	:	Slow oxidative muscle fibbers
Type IIa	:	Fast oxidative muscle fibbers
Type IIb	:	Fast glycolytic muscle fibbers
μ A	:	Micro ampere
μ m ²	:	Micrometer square

$\mu\text{mol.kg}^{-1}.\text{m}^{-1}$:	Micromole per kilogram per meter
VO_2	:	Oxygen consumption
$\text{VO}_{2\text{peak}}$:	Peak oxygen consumption
$\text{VO}_{2\text{max}}$:	Maximum aerobic capacity
$V_{\text{VO}_{2\text{max}}}$:	Maximum velocity at $\text{VO}_{2\text{max}}$
V_{LT}	:	Speed at lactate threshold
V_{T}	:	Ventilatory threshold

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CHAPTER ONE

INTRODUCTION

The world is becoming a global village mainly as a result of technology. In the last few decades this has led to the adoption of urbanized lifestyles among many populations worldwide. This has also brought about modifications in dietary and lifestyle habits. Consumption of indigenous foods has gradually been replaced by the intake of refined carbohydrates and large quantities of saturated fat, while stress levels have soared and the levels of physical activity and exercise have declined. All these factors have contributed to an epidemic of obesity, hypertension, diabetes and various chronic diseases of which cardiovascular illnesses are prominent (Derman, 2008; Steyn *et al.*, 2008). Even though, the World Health Organization (WHO) defined health as: “a state of complete physical, mental and social well-being, not merely the absence of disease and infirmity” (Callahan, 1973).

Literature suggest that about 90% of healthy individuals who are 55 years and older are likely to develop hypertension which can eventually lead to the onset of all forms of cardiovascular disease, as well as renal and cerebral conditions (Taylor *et al.*, 2010). This explains the vast amount of hypertension-related research over many years. Attention has mainly focused on the regulation of arterial blood pressure following acute bouts of exercise and the inflated arterial blood pressure response to exercise in hypertensive patients. Secondly, the application of a non-pharmacological approach, such as regular exercise, to lower resting arterial blood pressure has also been investigated (Kenney and Seals, 1993).

Moreover, a third component of the blood pressure response to exercise has become of interest to clinical scientists and physiologists in recent years. This comprises the temporary decline in blood pressure (BP) observed in the minutes or hours following an acute bout of

exercise (Kenney and Seals, 1993), which has been termed post-exercise hypotension (PEH). Research suggest that PEH is observed in both hypertensive and non-hypertensive individuals (MacDonald *et al.*, 1999a), although the response is more pronounced in those with the highest resting BP (Forjaz *et al.*, 2000). PEH compared to rest is believed to be caused by a continuous decline in systemic vascular resistance which is not totally offset by cardiac output (CO) upsurges. This hemodynamic state is seen as a transition between that taking place at rest and during dynamic exercise. CO tends to drop more quickly from high values during exercise than systemic vascular resistance recovers at exercise cessation. The imbalance between these two determinants of arterial pressure causes the sustained hypotensive response observed for some hours after exercise (Halliwill, 2001). Uncertainty prevails on the exact duration and magnitude of the PEH response. However, the issue concerning PEH is more of the deliberation on its clinical significance or whether it is a mere physiological phenomenon. Hence to pronounce PEH as clinically significant, it should generate a significant drop in arterial pressure, should be long lasting and should be maintained during events of daily living. A clinically significant PEH response would thus be an effective alternative, or additional strategy to pharmacological treatment, in the management of hypertension (Kenney and Seals, 1993).

It is also known that hypertension is usually linked to numerous other cardiovascular risk factors which include overweight, obesity, inactivity, etc. Considering the global rise in the prevalence of obesity (Van Dam *et al.*, 2006), and the direct relationship between obesity and hypertension; as overweight individuals are more at risk for developing hypertension (Barrett-Connor and Khaw, 1985), it can be expected that the prevalence of cardiovascular disease, specifically hypertension, is also rising. Available report indicates that obese (body mass index, BMI > 30 kg.m⁻²) women have higher than 41% prevalence of hypertension whereas women with < 25 kg.m⁻² BMI have lower than 25% prevalence (Costa, 2002). More

importantly, evidence shows that a decrease in body mass by 1kg causes 1.6 and 1.3 mmHg reduction in SBP and DBP respectively. Thus an array of 4 - 8% reduction in body mass can result in 3 mmHg reduction in BP (Costa, 2002). PEH has also been investigated with various types of exercise which includes resistance exercise (Brown *et al.*, 1994), aerobic exercise (Bermudes *et al.*, 2003), water exercise (Terblanche and Millen, 2012) and concurrent exercise (Keese *et al.*, 2012).

Another concern is the problem of low levels of physical activity participation in the general population, regardless of the fact that physical inactivity is associated with increased risk of many diseases such as cardiovascular diseases, diabetes and other risk factors (Morrow *et al.*, 2004). According to Mosca *et al.* (2007) physical activity participation remains low, although the American College of Sports Medicine (ACSM) contends that a minimum of 30 minutes (min) of participation in moderate-intensity exercise on most days of the week helps to achieve health benefits (Whyte *et al.*, 2010; Pate *et al.*, 1995). It has recently been suggested that similar benefits could be achieved with shorter-duration (minimum of 20 min) higher intensity exercise to be done at least three times per a week (Haskell *et al.*, 2007). The introduction of shorter duration exercise sessions may be a significant step towards the promotion of physical activity, as lack of time is frequently mentioned as the main obstacle to engagement in regular activity (Whyte *et al.*, 2010; Reichart *et al.*, 2007; Trost *et al.*, 2002). Nonetheless the most favorable intensity, duration and volume of high-intensity exercise that is needed to attain optimal health benefits are still contentious. Some researchers are also wary about the safety of this type of exercise for at risk populations.

PEH is well known to occur after traditional endurance and resistance training and most researchers agree that an acute bout of exercise will lead to a clinically significant decrease in BP, especially in those with persistently high BP. However, information on the blood

pressure response to high intensity exercise is limited and little is known as to whether this type of exercise elicits a PEH response. Since the popularity of this type of exercise seems to be on the increase, it is worthwhile to investigate the effects of high intensity interval exercise on the BP response.

CHAPTER TWO

POST-EXERCISE HYPOTENSION

A. INTRODUCTION

About one billion individuals worldwide are affected by hypertension, which is considered the main risk factor for cardiovascular disease (Melo *et al.*, 2006; Chobanian *et al.*, 2003). This condition is associated with the development of coronary heart disease, congestive heart failure, stroke and renal dysfunction. Although pharmacologic interventions are effective in decreasing the risk for cardiovascular and renal disease, issues have been raised regarding the possible harmful side effects of antihypertensive drugs (The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure, 1997; National High Blood Pressure Education Program Working Group report on primary prevention of hypertension, 1993). For this reason, lifestyle changes, including exercise with particular attention to the PEH response, for the treatment and prevention of hypertension, are of great importance.

PEH is defined as the occurrence of a transient reduction in systolic and/or diastolic BP lower than resting levels after a single bout of exercise (Taylor *et al.*, 2010; Kenney and Seals, 1993). The recurrent occurrences of PEH may result in the sustained decline in arterial blood pressure and therefore can possibly be of clinical significance in the management of blood pressure (Lui *et al.*, 2012). Although Hill (1898) was the first to document PEH during 90 minutes after a 400 yard sprint in 1898, this phenomenon was only scientifically investigated after Fitzgerald (1981) subjectively reported the effect of jogging on his own labile hypertension. Because of the importance of the non-pharmacological effects of PEH as a less expensive treatment and prevention modality for arterial hypertension, the effects of exercise

on BP are frequently investigated (Pescatello *et al.*, 2004a). This PEH response observed after exercise compared to rest is believed to be caused by a continuous decline in systemic vascular resistance which is not totally offset by cardiac output upsurges (Halliwill, 2001). However, PEH ought to retain a significant magnitude over an extended period of ambulatory conditions to be clinically important (Cardoso *et al.*, 2010).

B. PEH IN HYPERTENSIVE AND NORMOTENSIVE INDIVIDUALS

PEH in people with borderline hypertension and diagnosed hypertension is well documented (Brownley *et al.*, 1996; Hara and Floras, 1994; Floras and Wesche, 1992; Cléroux *et al.*, 1992; Pescatello *et al.*, 1999; Floras *et al.*, 1989; Bennett *et al.*, 1984), as well as in normotensive individuals (Forjaz *et al.*, 1998; Franklin *et al.*, 1993; Somers *et al.*, 1991; Kaufman *et al.*, 1987), although this occurrence is inconsistent in studies, probably because the PEH response is of lesser magnitude in normotensive than in hypertensive patients. This phenomenon has been extensively reported to be of importance as persons with hypertension usually have systolic blood pressure (SBP) of ≥ 140 mmHg and diastolic blood pressure (DBP) ≥ 90 mmHg according to the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure (JNC) diagnostic criteria for hypertension. The post exercise decrease in arterial blood pressure is generally greater in hypertensive people compared to normotensive individuals (Pescatello *et al.*, 2004a). A review study has shown that the maximal exercise induced fall in SBP and DBP is on average 18 to 20 mmHg and 7 to 9 mmHg, respectively, in persons with high blood pressure and 8 to 10 mmHg and 3 to 5 mmHg, respectively, in normotensive individuals. In both cases, the reductions are considered clinically significant (Kenney and Seals, 1993).

C. PHYSIOLOGICAL MECHANISMS UNDERLYING PEH

Literature concerning the systemic and regional hemodynamics after bouts of exercise is conflicting as the specific physiological mechanism underlying PEH is not obvious. It is currently believed that a combination of factors contribute to the PEH response (Pescatello *et al.*, 2004a). The immediate reduction in BP is likely due to a decreased output from the sympathetic nervous system (SNS), reduced vascular responsiveness to α -adrenergic receptor activation and an increase in local vasodilatory substances (Halliwill, 2001). All of these responses may occur at different times or in varying sequences. Since mean arterial pressure is determined by cardiac output (CO) and total peripheral resistance (TPR), it implies that a decrease in arterial pressure after acute exercise might be arbitrated by reductions in either one or both variables (Pescatello *et al.*, 2004a; Kenny and Seals, 1993). However, reductions in TPR emerges as the likely principal mechanism by which BP decreases since resting CO usually increase after acute exercise because of increased stroke volume (SV) (Pescatello *et al.*, 2004a; Cl  roux *et al.*, 1992).

According to Poiseuille's law the resistance of a blood vessel is directly proportional to the blood viscosity and length of the blood vessel and inversely proportional to the fourth power of the radius of the blood vessel (Davies *et al.*, 2001). Thus changes in TPR is mainly influenced by the diameter of the blood vessel since blood viscosity and length usually do not change with exercise; as a small change in vessel diameter will have an immense effect on the vascular resistance (Pescatello *et al.*, 2004a). In addition, it has also been reported that blood plasma may be absorbed into the interstitial space, decreasing the blood volume and the subsequent venous return to the heart which would result in decreased SV and thus the CO; this is considered a likely underlying mechanism that contributes to PEH (MacDonald, 2002). However, a decrease in blood volume following exercise does not normally happen so

the reduction in blood plasma volume cannot be considered a major determinant of PEH (Cléroux *et al.*, 1992).

Cléroux *et al.* (1992) observed significant reductions in SBP and DBP (-11 mmHg and -4 mmHg, respectively) in hypertensive subjects after leg exercise on the cycle ergometer at an intensity of 50% VO_{2peak} which was significantly lower compared with the control values. However, in their normotensive counterparts there were no significant reductions in BP. These authors attributed the reduction in BP to the observed drop in TPR as the primary hemodynamic mechanism in hypertensive subjects. This conclusion was made even though both groups experienced reduced TPR after exercise. However, the reduction in TPR for the hypertensive group was significantly more than in the normotensive group (-27% and -15%, respectively). CO on the other hand, was increased in both groups, and also significantly more in hypertensives than the normotensives (+31% and +15%, respectively). Cléroux *et al.* (1992) indicated that the CO increment was related to the differences in SV which increased significantly in the hypertensive individuals (+22%) following exercise, compared to controls (+3%). These authors therefore suggested that the increased stroke volume was probably associated with the decreased afterload due to an unchanged left ventricular internal diameter in diastole in hypertensive individuals following exercise. Additionally, SV was not significantly affected in the normotensives due to increased left ventricular internal diameter in diastole.

Brandão Randon *et al.* (2002) investigated the impact of hemodynamic left ventricular function on post exercise BP in elderly hypertensive. The participants were 23 hypertensive and 18 normotensives who performed 45 min of bicycle exercise at low-intensity (50% VO_{2max}). They showed a significant decrease in BP and CO as early as 15 and 30 minutes post-exercise, which was significantly more in the hypertensive than the normotensive

subjects. The drop in BP was associated with a reduction in left ventricular end-diastolic volume and a consistent decrease in SV and HR. CO and SV remained unchanged in normotensive subjects and thus no PEH was noticed. Additionally, Brandão Randon *et al.* (2002) observed no changes in TPR. Therefore, this study suggests that the PEH response in the elderly may be more related to a drop in CO rather than changes in peripheral vascular resistance as is typically observed in younger individuals.

Moreover, Teixeira *et al.* (2011) examined the hemodynamics and autonomic modulation after a single session of aerobic, resistance, and concurrent exercises in normotensive individuals. All participants performed four experimental sessions in random order: 30 min without exercise (Control, C); 30 min cycling on an ergometer at 75% of VO_{2max} (Aerobic, A); 6 exercises, (bench press, leg press, latissimus pull-down, knee flexion, arm curl, and squat; 3 sets, 20 repetitions, 50% of 1 RM) (Resistance, R); and a combination of A and R (Concurrent, AR). These authors observed significant decreases in both SBP and DBP (SBP: -13 mmHg, -8 mmHg, and -11 mmHg; DBP: -2 mmHg, -3 mmHg and -3 mmHg) respectively after A, R and AR that were associated with reductions in CO. This finding was consistent with previous research (Dujic *et al.*, 2006; Forjaz *et al.*, 2004; Brandão Rondon *et al.*, 2002; Senitko *et al.*, 2002; Takahashi *et al.*, 2000). Teixeira *et al.* (2011) explained that the decline in BP after exercise was accompanied by a reduction in CO as a result of a reduction in SV which may have caused an upsurge in afterload. They further reported on a reduction in CO following the control session which was as a result of a reduction in HR and a slight decrease in SV. However, the reduction in CO was followed by a rise in systemic vascular resistance (SVR) that inhibited the decrease in BP after the C session.

Concerning CO post-exercise, most studies support the hypothesis that the reduction in CO is due to a decrease in SV, which is probably mediated by the decline in the end-diastolic

volume. The reduction in venous return to the heart may explain the change in end-diastolic volume. However, a sustained reduction in SV and a progressive decline in HR beyond 30 min appear to result in the observed PEH.

According to Pescatello *et al.* (2004a) reductions in TPR after exercise is mostly arbitrated by the changes in vascular resistance as a result of small changes in vessel diameter. This change is linked with neurohumoral and structural adaptations, vascular responsiveness and possibly vasoactive stimuli, or any combination of these factors.

1. Neurohumoral adaptations

1.1. Sympathetic nervous activity (SNA)

Muscle SNA is regulated by the arterial baroreflex and cardiopulmonary receptor reflex during rest. Though, arterial baroreflex remains the major mechanism that controls short-term arterial BP (Ichinose *et al.*, 2008). This mechanism is responsible for the alteration of arterial BP and the reciprocal alteration in HR and sympathetic activity. Sympathetic activity increases as a result of an increased demand for oxygen and the baroreflex is set to operate at higher BP and HR during exercise (Ichinose *et al.*, 2008). However, after exercise there is a decreased sympathetic vasoconstrictor nerve activity outflow to skeletal muscle vascular beds and then the arterial and baroreflex are reset to a lower BP than initial levels before exercise (Floras *et al.*, 1989).

Floras *et al.* (1989) examined whether PEH is accompanied by a decrease in SNA to leg muscles in nine hypertensive subjects. They showed that SBP was significantly lower at 60 minutes post exercise (pre: 135 mmHg vs. post: 125 mmHg), while no significant change was observed for DBP (pre: 83 mmHg vs. post: 83 mmHg). SNA was significantly reduced (pre:

28 bursts/100 heart beats vs. post: 18 bursts/100 heart beats) 60 min post exercise. This study therefore suggests that PEH may be mediated at least in part, by the inhibition of SNA to muscle. Thus the authors suggested that the possible neural mechanism for the decline in SNA to the muscles after exercise could be attributed to suppression of the efferent SNA as a result of a prolonged increase in BP; inhibitory cardiopulmonary reflexes enabled after exercise and the inhibition of sympathetic outflow by the central baroreceptor reflexes as a result of the activation of opioid and serotonergic systems. This observation was confirmed by Somers *et al.* (1991) who demonstrated that an increase in baroreflex sensitivity was accompanied by lower BP, as well as a reduction in the baroreceptor set point with increased physical fitness in hypertensive individuals after endurance training.

Likewise, Hua *et al.* (2009) investigated the effect of low intensity exercise on BP and autonomic modulations of HR in middle aged hypertensive patients who were on medication. Participants were grouped into exercise and control groups. The exercise group performed low intensity exercises (walking at 35 - 40% HR reserve, HRR) 4 days per week for 12 weeks, while their control counterparts continued with normal daily activities. At the end of the training program, a significant reduction in mean SBP/DBP (11.1/5.2 mmHg) was observed for the exercise group compared to the control (6.1/1.0 mmHg). The baroreflex sensitivity increased in the exercise group while it decreased in the control group. The authors explained that the observed reduction in BP was mediated by the resetting of baroreflex sensitivity to higher operating levels due to the exercise training.

Hart *et al.* (2010) investigated the carotid baroreflex (CBR) control of HR and MAP in young normotensive men. The participants performed ergometer rowing at 10 –15% of workload below the lactate threshold for four hours. Results indicated a significant reduction in MAP (98 mmHg vs. 86 mmHg) and a significance increase in HR (60 b.min⁻¹ vs. 81 b.min⁻¹) after

exercise. It showed that CBR operated around a lower arterial pressure which the researchers explained was associated with resetting of the baroreflex after exercise and they concluded that resetting of CBR to a lower arterial pressure after exercise contributed to the lower MAP. Also, Halliwill *et al.* (2013) contends that the decline in BP after exercise could be attributed to a combination of centrally mediated decline in sympathetic nerve activity, together with a decrease in sympathetic signal transduction to local blood vessels.

From the literature, it is clear that the SNA plays an important role in the underlying mechanism of PEH. This is confirmed by the resetting of baroreflex sensitivity to higher operating levels observed after exercise.

1.2. Atrial natriuretic peptide (ANP)

ANP is released from heart muscle cells, specifically in the atria, in response to high blood pressure (Vollmar, 1990). It has both natriuretic and vasodilatory properties and plays a role in fluid balance, thus controlling BP (MacDonald *et al.*, 1999a). It has been shown that ANP levels rise in the circulation in response to high intensity exercise as well as endurance exercise (Perrault *et al.*, 1994; Perrault *et al.*, 1991). MacDonald *et al.* (1995) also detected higher ANP concentrations in the circulation (pre: 11.5 pg.ml⁻¹ vs. post: 18.6 pg.ml⁻¹) following heavy resistance exercise (80% 1RM) in young healthy individuals. According to Espiner and Nicholls (1987), the hemodynamic effect of ANP is sustained for several hours after exercise, even though it has a short half-life (2 min).

MacDonald *et al.* (1999a) investigated the effect of exercise on ANP concentrations in plasma and also assessed the possible correlation between ANP and post exercise BP in young normotensive men. The subjects underwent 15 min of unilateral leg press exercise (65% 1RM) and 15 min of cycle ergometer exercise (65% VO_{2max}) within a one week

interval. They observed significant reductions after exercise in both trials. After 10 min SBP was ≈ 20 mmHg less than pre-exercise, whereas after 30 min the mean pressure was ≈ 7 mmHg less. However, there was only a small non-significant increase in ANP concentration immediately after exercise and this returned to resting levels after 5 min into recovery. Thus they concluded that PEH after acute sessions of either resistance or submaximal cycle exercise was not directly related to the activation of ANP.

Generally, from the literature it is known that increased ANP concentration is associated with a decrease in BP. However, the possible reason for the non-significant increase in ANP concentration in the study of Macdonald *et al.* (1999a) could be due to the fact that exercise duration was insufficient to provoke the release of ANP. They also reported faulty preservative reagents which confounded the ANP analysis for some of the subjects.

1.3. Renin-Angiotensin system

The renin-angiotensin system is the most influential hormonal system in the human body. It plays a key role in the regulation of vessel diameter and volumes of fluid in the body (Cornelissen *et al.*, 2011). In the juxtaglomerular apparatus, granular cells of the afferent and efferent arterioles of the glomerulus secrete the enzyme renin. The secretion of renin is stimulated by three factors which includes: increased renal SNA, decreased renal perfusion pressure (decrease in pressure within the afferent arteriole resulting in reduced tension in the arteriole wall) and reduction in sodium chloride (NaCl) delivery to the macula densa (e.g. when blood volume is reduced sodium ions (Na^+) are reabsorbed into the proximal convoluted tubule). Macula densa cells release prostacyclin and act on renin-secreting cells to cleave decapeptide angiotensin I from angiotensinogen. Angiotensin converting enzyme (ACE) then converts angiotensin I to octapeptide angiotensin II which is a powerful

vasoconstrictor (Davies *et al.*, 2001). Since this system plays an important role in blood volume homeostasis, reductions in the resting levels of plasma renin and angiotensin II may possibly affect the BP response after exercise.

Kiyonaga *et al.* (1985) examined the hormonal responses of middle aged, hypertensive patients after 10 to 20 weeks of aerobic training at 50% VO_{2max} . These authors found significant reductions in mean BP after the 10-weeks (109 mmHg) and the 20-weeks (105 mmHg) of training compared to initial values (120 mmHg). 50% of the patients responded with an effective reduction in BP after 10-weeks training and 78% of the patients achieved effective reduction in BP after 20-weeks training. In comparison to the non-responders, the responders showed significantly reduced initial plasma renin-angiotensin activity ($0.6 \text{ ng.ml}^{-1} \cdot \text{h}^{-1}$) than the non-responders ($1.95 \text{ ng.ml}^{-1} \cdot \text{h}^{-1}$). This correlated significantly with the reduction in BP after aerobic training, indicating that plasma renin-angiotensin also contribute to the changes in BP. Thus the change in BP in the responders was possibly mediated by the reduced plasma renin activity at rest.

Cononie *et al.* (1991) evaluated the effect of six months of resistance and endurance exercise training on BP, hemodynamic parameters, and pressor hormone levels in normal to moderately hypertensive men and women (aged 70 – 79 years). Participants were grouped into resistance training (one set of 8 – 12 repetitions, three sessions a week) and endurance training (20 – 30 min at 50% VO_{2max} for the first four months of exercise and progressed in the last two months for 35 - 45 min at 75 – 85% VO_{2max} , three sessions a week). Results indicated that resting BP decreased significantly in the endurance group (pre: 109 mmHg vs. post: 101 mmHg, but not the resistance group ($105 \pm 12 \text{ mmHg}$ vs. $105 \pm 5 \text{ mmHg}$; $p > 0.05$). Further analysis of the pressor hormonal levels also indicated statistically significant increases in plasma angiotensin I and II (pre: 14.5 pg.m^{-1} and 10.8 pg.m^{-1} vs. post: 19.9 pg.m^{-1}

and 14.0 pg.m^{-1}) compared to their respective initial levels. Furthermore, they reported a statistically significant relationship between the reduction in BP and initial levels of plasma angiotensin I after three and six months of training in the endurance training group.

Considering the limited literature, one can only speculate whether the renin-angiotensin system may contribute to the post-exercise BP response. Further studies are warranted on this topic.

2. Vascular adaptations

At tissue level, circulating hormones and/or metabolic factors might contribute to PEH as several vasodilator substances (e.g. nitric oxide, histamine and prostaglandins) have been associated with this phenomenon (Lockwood *et al.*, 2005). Nitric oxide (NO) is an important vasodilator which is released from the endothelium of arterioles. NO promotes the relaxation of smooth muscles in the arterioles which results in vasodilation and lowering of vascular resistance and thus causing blood flow to increase (Powers and Howley, 2009). Endothelial-1 (ET-1), on the other hand, is a strong vasoconstrictor peptide also generated by the vascular endothelial cell and has strong proliferation activity on vascular smooth muscle cells. The NO production pathway seems to cross talk with the generation of the ET-1 pathway, such that the two endothelial secretions counter inhibit the other. Thus exercise training is believed to reduce ET-1 in the endothelium, while NO generation is increased which will probably result in a lower BP (Maeda *et al.*, 2001).

Nyberg *et al.* (2012) investigated the role of NO and prostanoids in the regulation of BP before and after eight weeks of high intensity cycling training in essential hypertensives matched with normotensive controls. The authors failed to describe the training protocol in terms of intensity and duration. After training mean BP was significantly reduced (pre: 126.6

mmHg vs. post: 117.5 mmHg) in hypertensive subjects but not in normotensive subjects (pre: 98.8 mmHg vs. post: 98.6 mmHg) compared to pre-exercise. But when these authors later inhibited NO and prostanoid systems by the infusion of N^G -mono-methyl-L-arginine (L-NMMA; inhibition of NO formation) and indomethacin (inhibition of prostanoid formation) together after exercise this training-induced lowering effect in BP was abolished. The skeletal muscle vascular endothelial NO synthase uncoupling expression and phosphorylation status were similar in both groups pre and post training. This indicates that the observed reduction in BP was associated with the training induced change in the tonic effect of NO and/or prostanoids on vascular tone. They further explained that the influence of L-NMMA and indomethacin on BP could have accounted for the activation of the arterial baroreflex and subsequent inhibition of central sympathetic outflow. However, the absence of change in heart rate makes this improbable.

Maeda *et al.* (2003) demonstrated significant reductions in resting BP in both SBP (pre: 127 mmHg vs. post: 112 mmHg) and DBP (pre: 79 mmHg vs. post: 65 ± 3 mmHg) following 3 months of aerobic training (30 min cycling at 80% of V_T) performed 5 days per week by older normotensive women. This reduction in BP is believed to be mediated by the reduction ET-1. Since ET-1 was significantly decreased (pre: 2.90 pg.ml^{-1} vs. post: 2.22 pg.ml^{-1}) after the training. ET-1 correlated positively with changes in SBP and DBP after training; confirming that reduction in ET-1 mediates PEH. Even though these authors did not measure NO, it can be suggested that the suppression of plasma ET-1, would permit an increased action of NO which would cause the blood vessel to dilate to allow increased blood flow at a reduced pressure.

Similarly, Maeda *et al.* (2001) investigated the effects of eight weeks of training and detraining on plasma levels of endothelium-derived factors, ET-1 and NO in young

normotensives. Participants exercised at 70% $\text{VO}_{2\text{max}}$ on the cycle ergometer for an hour, 3 - 4 days per week. At the end of the eight weeks of training, even though there was no significant change in BP compared to pre-exercise values, there was a significant increase in plasma NO concentration ($30.69 \mu\text{mol.L}^{-1}$ vs. $48.64 \mu\text{mol.L}^{-1}$) and a concomitant and significant decrease in plasma ET-1 (1.65pg.mL^{-1} vs. 1.23pg.mL^{-1}). In both cases the lower levels were sustained for four weeks after training and returned to pre-exercise values after eight weeks of detraining. However, the reciprocal change in NO and ET-1 concentration in the plasma showed a significantly negative correlation between the two endothelium-derived factors.

It can be deduced from the literature that an increase in the production of NO induced by exercise training would partly contribute to the suppression of ET-1 production in the endothelium; which would reduce the resistance to blood flow and thus reducing the blood pressure in the vessels. However, it is not clear what the exact physiological interactions of these endothelium-derived factors are and whether these substances independently cause lower BP after exercise. One can suggest that increases in NO and the reciprocal decrease in ET-1 may cause decreased vascular resistance and a subsequent decline in BP.

3. Structural adaptations

When an individual undergoes exercise training, there are also changes to the vascular structure of the muscle tissue. These adaptations include increased length, cross-sectional area and diameter of already existing arteries, as well as the development of new blood vessels (i.e. angiogenesis) that cause the capillary bed to expand, greater distensibility of the vessels and a larger lumen diameter. All these structural adaptations may possibly contribute to the lower TPR after training (Pescatello *et al.*, 2004a). Not only do the changes occur in

the vasculature surrounding the muscle, but the muscle fiber itself also adapt to training. Cross-sectional data showed that endurance trained athletes have larger diameters of the arterial lumen in conduit arteries than untrained control subjects (Huonker *et al.*, 1996; Wijnen *et al.*, 1991; Shenberger *et al.*, 1990).

Furthermore, reduced femoral artery intima-media thickness (IMT) has also been detected in endurance-trained men and women compared to sedentary peers (Dinenno *et al.*, 2001). Moreau *et al.* (2002) contrasted femoral and carotid artery IMT between endurance-trained athletes and sedentary controls and reported decreased femoral artery IMT in athletes, though no significant difference was found between the groups for carotid IMT. These results indicate that remodeling of the arterial wall in peripheral arteries may be more heavily related to training than that seen in the carotid arteries (Thijssen *et al.*, 2012). Thus it can be concluded that arterial wall thickness is reduced in well trained athletes, compared to untrained individuals. A thickened arterial wall will have increased vascular tone and sympathetic nerve activity, and thus result in an increased mean BP; the opposite may contribute to lower BP after exercise training.

Even though the adaptations in the cardiovascular and musculoskeletal system following exercise training may contribute to the reduction of BP, these adaptations only occur with prolonged training by the accumulation of the acute effects of exercise.

4. Genetic variation

Even though the phenomenon of PEH is well documented in patients with hypertension, its magnitude (-2 to -12 mmHg) and duration (4 to 16 hours) differ significantly, indicating that it might be influenced by several factors such as subject and/or exercise specificity (Cardoso *et al.*, 2010). However, some studies have also reported that genetic variation could be a

contributing factor that affects the magnitude and duration of PEH responses in individual subjects.

Blanchard *et al.* (2006) reported the presence of the angiotensin converting enzyme gene (ACE DD) among hypertensive persons who demonstrated ambulatory PEH, whereas PEH was not observed in hypertensive subjects who had other polymorphic variants (ACE II/ID). Moreover, they also found that hypertensive persons who had more than three polymorphisms associated with the renin-angiotensin-aldosterone system demonstrated greater reductions in ambulatory BP after exercise. Additionally, Pescatello *et al.* (2007) also found that hypertensive individuals with low calcium intakes and the ACE DD polymorphism exhibited greater hypotension effects after low-intensity aerobic exercise, whereas subjects who had low calcium intakes but without the polymorphism, or those who had high calcium intakes and the polymorphism, responded better to moderate intensity exercise.

Considering the literature it is clear that the genetic variation of individuals seem to play a part in the underlying mechanism of PEH, since individuals who have the angiotensin converting enzyme gene (ACE DD) seem to be better responders of reduced BP than those who possess the polymorphic variants (ACE II/ID) type.

It is impossible to select one unique mechanism that actually mediates PEH. However, the mechanisms underlying reductions in BP after acute exercise may be linked to the acquired physiological adaptations accumulated after prolonged exercise training (Tsai *et al.*, 2004). Nevertheless, some of the acquired physiological adaptations due to exercise may not directly relate to the reduction in BP, but rather it might indirectly influence other mechanisms that contribute to PEH in one way or the other. Moreover a single mechanism does not account for PEH in isolation, rather a complex interaction of several mechanisms contribute to the observed PEH response.

D. SUMMARY OF FACTORS THAT AFFECT THE MAGNITUDE OF PEH

Although a session of submaximal exercise is sufficient in causing important cardiovascular changes which may minimize cardiovascular risk and maximize the hypotension effects through exercise, the interaction between the different characteristics (such as intensity, duration, volume of work load and repetition per set, recovery in between sets or sessions, etc.) must be carefully considered, as a certain blend of various variables may have the most beneficial effect on exercise training. For this reason extensive investigations have been done on the different exercise variables in an attempt to find the most effective mode, intensity, volume and duration of exercise to elicit a significant decrease in BP. Nevertheless, more research is required on the different characteristics of exercise to elicit the greatest PEH response possible, especially with regard to the intensity of the exercise bouts (Boroujerdi *et al.*, 2009). The majority of studies concerning post-exercise BP responses have shown that most types of exercises decrease BP in the course of the recovery period (Forjaz *et al.*, 1998). However, the magnitude and the time duration of the BP responses subsequent to the various types of exercise are contradictory.

1. Intensity of exercise

It is suggested that the magnitude and duration of the PEH response might be affected by the intensity as some studies that involved higher intensity exercise showed longer durations of PEH (Forjaz *et al.*, 2004; Quinn, 2000). However, it is not clear from the literature whether there is truly any relationship between the magnitude and duration of PEH, and the intensity of exercise. For example, Pescatello *et al.* (1991) did not find any difference in the lowering of ambulatory BP after exercise with different intensities within the aerobic range (40% and 70% $\text{VO}_{2\text{max}}$) in the normotensive group of their study, but PEH observed in hypertensive

counterparts indicated that exercise intensity did not matter as there was no difference in the magnitude of PEH associated with intensity of exercise. These authors found that exercise intensity at 40% $\text{VO}_{2\text{max}}$ elicited similar magnitude of PEH in SBP as exercise intensity at 70% $\text{VO}_{2\text{max}}$ (6 mmHg vs. 5 mmHg, respectively) for 12.7 hours post-exercise in duration. On the other hand, Quinn *et al.* (2000) observed greater hypotensive effects in both hypertensive and normotensive individuals after heavy exercise (75% $\text{VO}_{2\text{max}}$) in comparison to light exercise (50% $\text{VO}_{2\text{max}}$), while Blanchard *et al.* (2006) observed greater PEH responses in hypertensive subjects after low-intensity exercise. Their results indicated significant reductions in mean SBP (control: 133.2 mmHg vs. 131.2 mmHg and 130.9 mmHg, respectively) after moderate (60% $\text{VO}_{2\text{max}}$) and low (40% $\text{VO}_{2\text{max}}$) exercise compared to control sessions. However, average DBP was lower only after low intensity exercise (81.1 mmHg) versus control (82.7 mmHg) for 14 hours.

Moreover, studies that made the effort to directly compare exercise intensity and post-exercise BP have reported conflicting results. High-intensity aerobic exercise (70 to 75% $\text{VO}_{2\text{max}}$) has been shown to evoke greater PEH than lower intensity (50% $\text{VO}_{2\text{max}}$) exercise (Forjaz *et al.*, 2004; Kenny *et al.*, 2003; Quinn, 2000; Piepoli *et al.*, 1994). Conversely, other studies (Blanchard *et al.*, 2006; Syme *et al.*, 2006; Pescatello *et al.*, 2004b) found greater reductions in PEH after low intensity exercise (40% $\text{VO}_{2\text{max}}$) than moderate intensity exercise (60% $\text{VO}_{2\text{max}}$) and others (Pescatello *et al.*, 2007; Guidry *et al.*, 2006; Cornelissen *et al.*, 2010; Pescatello *et al.*, 1991) observed no significant influence of exercise intensity on PEH. In the cases, there were similar magnitudes of PEH after exercise at intensities ranging from 40 to 70% $\text{VO}_{2\text{max}}$ or HRR.

Another study by Teixeira *et al.* (2011) examining the hemodynamics and autonomic modulation after a single session of aerobic, resistance, and concurrent exercises, conjectured

that the absence of an additive effect when aerobic and resistance exercises are combined may be associated with the exercise intensity (75% of VO_{2max}) employed in their investigation. These authors observed that the greatest hypotensive effects had already occurred with the aerobic exercise at 75% VO_{2max} , thus adding resistance exercise did not result in any additional effect.

Syme *et al.* (2006) investigated the relationship between peak SBP attained during a maximal graded exercise test and PEH response in adult hypertensive men. Subjects performed graded exercise test and two cycling exercise sessions at 40% (light) and 60% (moderate) VO_{2max} SBP which was monitored for 10 hours post exercise. Subjects' peak SBP on a graded exercise test was grouped into low, medium and high. The group with high SBP during the maximal exercise test had SBP decreased by 7.3 mmHg and 5 mmHg after light exercise and moderate exercise respectively. While the group with low SBP 6.3 mmHg reduction in SBP after the moderate exercise session. Thus only light exercise is required to cause hypotensive response in the group with high peak SBP, while in the group with a low peak SBP, moderate exercise intensity is required to cause hypotensive response. The investigators suggested that the light exercise intensity interacted positively with the hormonal ambiance of the men with high peak SBP which might probably be as a result endothelial dysfunction due to lesser vasoconstrictor effect of the sympathetic nervous system. On the other hand, the group with the low peak SBP could offset the greater neural vasoconstriction exerted by sympathetic nervous system on the vascular system probably because of their efficient endothelium which required higher intensity exercise for a more continued decline in peripheral vascular resistance. Therefore, it is important to know the tolerable stimulus for a particular population in order to control the factors that might affect PEH to avoid over training.

Keese *et al.* (2012) also reported on the influence of different intensities of the aerobic segment of concurrent exercise sessions, together with the same amount of resistance exercise, on PEH among individuals with normal BP. Subjects performed four sessions of exercise: control (CTL) 60 min seated rest, and concurrent exercise 1; CE1 which represented 2 sets of 6 exercises at 80 % 1RM followed by 30 min of cycle ergometer exercise at 50 % VO_{2max} , concurrent exercise 2 and 3; CE2 and CE3 also consisted of 2 sets of 6 exercises at 80 % 1RM each followed by 30 min of cycle ergometer exercise at 65 % and 80% VO_{2max} , respectively. The magnitude of the reduction in SBP was similar after all CE sessions (CE1: 4.2 mmHg; CE2: 4.8 mmHg; CE3: 6.0 mmHg), but the hypotension response lasted about 1 hour longer after CE2 and CE3 (2 hours) compared to CE1 (60–70 min) ($p < 0.05$.) There was no significant difference in the magnitude of the DBP decrease between CE2 and CE3 (1.5 ± 0.6 mmHg and 1.8 ± 1.2 mmHg, respectively; $p = 0.1$), but reductions in DBP following CE3 was greater than after CE1 (1.2 ± 0.4 mmHg; $p < 0.05$) and lasted longer after CE3 (60 min) compared to CE2 and CE1 (40 min). It was therefore concluded that CE sessions (combining resistance and aerobic sessions) elicited a meaningful PEH, especially when the intensity of the aerobic exercise was higher than 65 % VO_{2max} .

Liu *et al.* (2012) compared the magnitude of PEH following two intensities of prolonged exercise in both middle-aged (52 years) and young (28 years) adult endurance athletes. Both groups performed prolonged (120 min) treadmill running at either moderate intensity (60% VO_{2max}) or high intensity (80% VO_{2max}) in random order over a four week period. During an hour recovery, there were significant reductions in SBP and DBP following high intensity exercise in both groups. However, the magnitude of PEH was greater in the middle aged group (SBP/DBP: 15.1/9.8 mmHg) than the younger group (SBP/ DBP: 5.7/4.0 mmHg). Compared to high intensity, the magnitude of PEH in SBP and DBP following moderate intensity was lower even though it was significant in the middle-age (SBP/DBP: 12.3/6.6

mmHg) and younger group (SBP/DBP: 1.2/2.5 mmHg). The findings of these authors confirm that high intensity exercise have a greater effect on the magnitude of PEH. Furthermore, the magnitude of PEH is also affected by age and baseline BP (middle-age: 124/79 mmHg; young: 111/69 mmHg).

Eicher *et al.* (2010) was one of the first to examine the influence of a range of exercise intensities on PEH. Their study participants comprised overweight/obese individuals who were hypertensive (SBP/DBP; $144 \pm 1.5/85.4 \pm 1.2$ mmHg) and borderline dyslipidemia. The subjects completed four experimental sessions; control without exercise, and three cycle exercises on an upright ergometer performed at 40% VO_{2max} (low), 60% VO_{2max} (moderate) and 100% VO_{2max} (vigorous or high). The average post exercise reduction in SBP over a nine hour recovery seated rest period compared to the control session were 2.7 mmHg after low intensity, 5.4 mmHg after moderate and 11.7 mmHg after vigorous intensity, respectively. The difference in the PEH response was statistically significantly greater after vigorous exercise compared to moderate and low intensity exercise. The decrease in DBP after exercise followed a similar pattern of 1.5 mmHg, 2.0 mmHg and 4.9 mmHg after low, moderate and vigorous intensity, respectively. The authors concluded that there is a dose response relationship between exercise intensity and the magnitude of the PEH response, e.g. more vigorous bouts elicit greater reductions in BP. Thus to optimize the magnitude of the BP lowering effects of aerobic exercise, a more individualized approach to exercise intensity prescription may be warranted for those with stage 1 hypertension and individuals who are willing and able to tolerate intense exercise.

Considering the literature, conclusions could be made that the intensity of exercise has a major effect on the hypotensive response following exercise. Differences in results between studies can be attributed to the relative intensity at which subjects performed the exercise,

such that what seemed high intensity for populations in one study was probably considered low or moderate intensity in another study. Since intensity varied relatively from one study to the other there would be conflicting results. Nevertheless, the majority of studies indicate that it is very important to consider intensity as a key component in a training program for BP reductions.

Moreover, it is reasonable to expect a relationship between exercise intensity and the BP response, since SNS activity, norepinephrine release and the changes in the hemodynamic response during exercise are directly related to exercise intensity. Therefore it is possible that the intensity of exercise also has an effect on the cardiovascular responses after exercise (Forjaz *et al.*, 2004; Forjaz *et al.*, 1998; Rueckert *et al.*, 1996; Cl  roux *et al.*, 1992).

2. Duration of exercise

The duration of the exercise bouts is another variable that can influence the magnitude of PEH as it is the expectation that longer exercise sessions may cause greater PEH responses. It is difficult to evaluate this aspect in the literature due to the differences in terms of intensity, modality of exercise and the duration of the post-exercise BP measurements in various studies (MacDonald, 2002). There is evidence that exercise as short as 20 min is enough to decrease BP, but that longer duration exercise cause a greater magnitude of hypotensive response (Guidry *et al.* 2006).

Bennett *et al.* (1984) investigated the BP response to intermittent exercise in both normotensive and hypertensive subjects. The individuals exercised for 10 min alternately with 3 min rest periods and this was repeated five times. Their results showed significant reductions in mean SBP/DBP (108/73 mmHg) after the fifth bout of exercise compared to the

mean SBP/DBP (117/78 mmHg) after the first bout of exercise compared to the pre-exercise mean SBP/DBP (120/80 mmHg) values in normotensive subjects. However, in the hypertensive subjects mean BP decreased significantly after all five bouts of exercise (SBP/DBP: 146/92 mmHg; 134/85 mmHg, for the first and fifth bout of exercise, respectively) compared to the pre exercise values (SBP/DBP: 163/108 mmHg). But the greatest magnitude of PEH was observed only at the fifth bout of exercise (29 mmHg vs. 17 mmHg) compared to the first bout of exercise. This showed that the duration of the exercise influenced the decline in BP levels post exercise.

Guidry *et al.* (2006) examined the influence of short and long duration exercise on the BP response of hypertensive individuals to an acute bout of dynamic exercise. The short duration consisted of 15 min of cycling at 40% or 60% VO_{2max} . The long duration consisted of 30 min of cycling at 40% or 60% VO_{2max} . The researchers observed a significant reduction in SBP by 5.6 mmHg and 4.3 mmHg after short and long duration exercise at 40% VO_{2max} compared to 4.1 ± 1.6 and 4.9 ± 1.9 mmHg ($p < 0.05$) after the short and long duration exercise at 60% VO_{2max} . DBP also decreased significantly by 3.4 mmHg and 4.6 mmHg after short and long duration exercise at 40% VO_{2max} compared to 2.1 mmHg and 3.6 mmHg after the short and long duration exercise at 60% VO_{2max} . It was concluded that the differences in PEH response was less dependent on the duration of exercise, but rather on the intensity of exercise.

Mach *et al.* (2005) reported greater and significant reductions in mean SBP (-16 mmHg; -19 mmHg) after longer duration exercise of 40 and 80 min in mild hypertensive individuals who performed moderate intensity cycling at 80% VO_{2max} , compared to shorter duration exercise of 10 and 20 min (mean SBP: -7 mmHg; -4 mmHg, respectively) during 90 min post exercise. The magnitude and duration of PEH was significantly greater after the longer exercise sessions (40 and 80 min) and it was lower throughout the recovery period compared

to the control period (control SBP: 131 mmHg). However, the duration of the exercise sessions did not have different effects on the DBP responses after exercise.

MacDonald *et al.* (2000) investigated the effect of exercise duration on the magnitude of PEH in normotensive and borderline hypertensive individuals. The normotensive subjects performed cycling at 70% VO_{2max} for 15, 30 and 45 min, while the hypertensive subjects did similar exercise but only for 10 and 30 min. Their results showed 12 mmHg and 4.6 mmHg significant reductions in SBP from pre-exercise values between 5 and 45 min post-exercise in all the exercise bouts in the normotensive subjects, but reductions in DBP was not affected by exercise duration. In the hypertensive subjects, SBP was decreased significantly by 14 mmHg and DBP by 8 mmHg for 60 min post-exercise. The magnitude of hypotension at all measured time points was lower following the 30 min trial, but this was not significantly different from the 10 min trial. The authors concluded that the duration of exercise had no significant effect on the magnitude of PEH in normotensive or hypertensive subjects.

The differences in results between studies may be related to the actual duration of the exercise bouts, as well as the total work done during exercise bouts. For instance, perhaps the exercise in the study of MacDonald *et al.* (2000) was not long enough to elicit a hypotensive response, whereas the 40 and 80 min bouts in the study of Mach *et al.* (2005) may indicate the optimal duration of exercise bouts. Results in different studies are further confounded by varying intensities of exercise bouts, which have been shown to be an important determinant of the PEH response.

Although it is not clear from the literature how much the duration of exercise affects the magnitude of PEH, this exercise variable should be considered together with the intensity of exercise. However, there is still no conclusive evidence on the optimal duration of exercise which will elicit a significant and long lasting PEH response.

3. Volume of exercise

In resistance training, volume is usually the total number of sets and repetitions performed during a training session (Scher *et al.*, 2011), while in aerobic exercise the volume of exercise may refer to exercise 'dose' in terms of the total energy expended representing the total work done. This is achieved by the combination of intensity and duration of exercise in terms of exercising at high intensity for shorter duration or at lower intensity for longer durations (Jones *et al.*, 2007).

Jones *et al.* (2007) investigated the magnitude of the acute PEH response by controlling the total work done during exercise at different intensity/duration in normotensive young men. Participants performed four exercise sessions which consisted of intense, INT (70% VO_{2max}) cycling for 30-min short duration, moderate (40% VO_{2max}) longer duration for 50-min (LMOD) and moderate (40% VO_{2max}) shorter duration for 30-min (SMOD), and control 30-min seated rest on cycle ergometer. Their results indicated similar post-exercise responses in BP between INT and LMOD which was greater than in SMOD exercise which in turn was significantly greater than control values. The minimum values of SBP after INT, LMOD and SMOD were -15 mmHg, -12 mmHg and -6 mmHg and these were significantly different from control values. However, mean DBP was not significantly different from control values. Thus, similar PEH responses were observed for high intensity short duration exercise and moderate intensity longer duration sessions, which were matched for total work done. The authors therefore concluded that the magnitude of the PEH response is dependent on the total work done during an exercise bout.

Polito and Farinatti (2009) investigated the effects of muscle mass and number of sets during RE on PEH in young healthy male university students. Participants were randomly assigned to three experimental sessions consisting of arm, leg and control sessions. The arm group did

biceps curls (6 and 10 sets of 10 repetitions at a 12RM workload), leg group did leg extension with 6 and 10 sets of 10 repetitions at a 12RM workload) and the control group did no exercise. Their results indicated that arm exercise, irrespective of the number of sets, did not result in significant reductions in BP compared to control session. However, SBP (120.6 mmHg vs. 107.1 mmHg to 113.4 mmHg) significantly decreased at all-time points taking during the 60 min recovery for 10 sets of leg extension exercises group, but not for 6 sets. Reductions in DBP were not significant for either group, while significant reductions in MAP (90.3 mmHg vs. 85.1 mmHg) were only observed at 30 min in the leg group but not the arm group. These results are in line with those by Jones *et al.* (2007) that higher volume exercises, i.e. with higher total work done, are more likely to cause a significant PEH response.

Scher *et al.* (2011) assessed the effect of different volumes of acute low-intensity resistance exercise on the magnitude of BP changes in older hypertensive persons. Sixteen subjects (mean age of 68 ± 5 years) completed three sessions in random order: Control (40 min of rest), Exercise 1, E1 (20 min of one complete circuit at 40% of 1RM) and Exercise 2, E2 (40 min of two complete circuits also at 40% of 1RM). These authors reported significant decreases in 24 hours ambulatory BP after all exercise sessions compared to baseline BP (baseline SBP: 130 mmHg; DBP: 76 mmHg; SBP (E1): 121 mmHg; SBP (E2): 119 mmHg, DBP (E1): 75 mmHg; DBP (E2): 73 mmHg). However, the exercise session with the highest volume, (E2), provoked the greatest magnitude of reduction in mean SBP (E2: 11 mmHg vs. E1: 9 mmHg) and DBP (E2: 3 mmHg vs. E1: 1 mmHg) 24 hours after exercise, with even greater reductions in BP during sleep (SBP (E2): 19 mmHg vs. SBP (E1): 16 mmHg and DBP (E2): 8 mmHg vs. DBP (E1): 6mmHg). They concluded that different volumes of acute resistive exercise sessions significantly reduced BP, but the greatest magnitude of effect was observed for the highest volume of exercise.

On the contrary, Simão *et al.* (2005) investigated the effect of resistance training volume and session format on the PEH response in young (22 years) normotensive men. Subjects were randomly allocated to four different resistance exercise groups (RE). G1 performed 5 resistance exercises at 6RM for all three days, while G2 performed 6 resistance exercises at 6RM. On the second day the two groups performed 3 sets of 6RM of their respective number of exercises in set repetition format. On the third day, G1 performed 3 sets of 12 repetitions at 50% 6RM in a circuit format, while G2 performed 3 sets of 12 repetitions at 50% 6RM in a set repetition format. There was no statistically significant difference between the two groups in terms of the magnitude of the PEH response, however, the lower BP lasted slightly longer in G2 than in G1 (60 min vs. 50 min). This study therefore suggests that the PEH response is not dependent on the volume of exercise, but that it may affect the duration of the PEH response. However, one can consider that even though the total work done in both exercise were not equal, the difference in the number of exercises performed between groups was not large enough (G1: 5 resistance exercises vs. G2: 6 resistance exercises) to elicit a significant difference in magnitude of PEH. Probably there could be significant difference between the two groups in terms of magnitude of PEH, if the difference in the number of exercises performed per set was larger (e.g. ≥ 3 exercise, instead of a difference of one exercise).

It can be concluded from the literature that the majority of studies suggest that the volume of exercise has an effect on the magnitude of the hypotensive response following exercise. Exercise at higher volume elicits greater magnitude of hypotensive response compared to low volume of exercise.

E. THE EFFECT OF DIFFERENT TYPES OF EXERCISE ON CHANGES IN BLOOD PRESSURE

1. Effect of resistance exercise (RE) on changes in blood pressure

Resistance training is utilized mainly for the improvement in muscle strength, power and muscle endurance for the maintenance of good health. In general, resistance exercise causes significant reductions in BP after exercise (Cardoso *at al.*, 2010). For example, Kelly and Kelly (2000) conducted a meta-analysis on the effects of progressive resistance exercise on resting BP of both hypertensive and normotensive adults. They found that in a series of training sessions from 6 to 30 weeks and at a frequency of 2 to 5 times weekly at varying intensities from 30 to 90% of 1RM over a time period of 20 to 60 min per session resulted in decreases of $\approx 2\%$ and $\approx 4\%$ in resting SBP and DBP, respectively (SBP: -3 mmHg; DBP: -3 mm Hg). They suggested that progressive resistance exercise is effective for reductions in resting BP. Such reductions in BP are of importance in reducing cardiovascular risks for stroke and coronary heart diseases morbidity and mortality.

Table 2.1: A summary of the effect of resistance exercise on changes in blood pressure

Study	Population H/NH	Exercise protocol	Duration of exercise	Method of recovery BP monitoring	Significant Results
Mota <i>et al.</i> (2013)	H elderly women	3 sets (8 -12 rep. of 10 exercises at 60 - 80 % 1 RM) at incremental intensities each four weeks.	40 min for 3 times per week in 16 weeks	60 min ambulatory	Decreased SBP Decreased DBP
Boroujerdi <i>et al.</i> (2009)	NH healthy young males	4 sets (6 &12 rep. of 4 exercises at 85% & 42.5% 1RM resp. for HIWT & LIWT	~40 – 60 min	60 min auscultatory	SBP decreased at all point 60 min but DBP decreased to only 30 min at HIWT SBP & DBP only decreased to 30 min at LIWT
Mota <i>et al.</i> (2009)	H middle aged men & women	A circuit of 20 rep. of 13 exercises at 40% 1RM	20 min	60 min ambulatory	Decreased SBP Decreased DBP
Melo <i>et al.</i> (2006)	H middle aged women	3 sets (20 rep. of 6 exercises at 1RM)		90 min auscultatory & 21 h ambulatory	Decreased SBP & DBP during 90 min but SBP & DBP only decreased to 10 h during 21 h ambulatory
DeVan <i>et al.</i> (2005)	NH young men & women	1 set of 9 exercise at 75% 1RM	30 – 45 min	150 min ambulatory	No change in SBP and DBP
Fisher (2001)	NH & H adult women	3 sets (15 rep. of 5 exercises at 50% 1RM)	~30 min	60 min auscultatory	Decreased SBP Decreased DBP
Roltsch <i>et al.</i> (2001)	NH sedentary young men & women	2 sets of 8 RM or 12 RM of 12 upper & lower body exercises	45 – 60 min	24 h ambulatory	No change in SBP and DBP

H, hypertensive; NH, non-hypertensive; rep, repetition(s); RM, repetition maximum; min; minute; SBP; systolic blood pressure; DBP, diastolic blood pressure; ~ about; HIWT, high intensity weight training; LIWT, low intensity weight training; &, and; h, hour

From the summary (Table 2.1) above the available literature suggests that resistance exercise is effective in causing hypotensive responses in both young and old hypertensive and normotensive individuals, even though studies have varying results. The majority of studies are done on hypertensive individuals and in women. The absence of a reduction in BP after exercise in some studies (DeVan *et al.*, 2005; Roltsch *et al.*, 2001), as well as the varied findings in studies cannot be explained, however, this may be attributed to the different study populations in terms of age or the differences in the exercise protocols including the intensity at which the exercises were performed. Also one can observe the methodological issues adopted in monitoring recovery blood pressure. For instance, auscultatory and ambulatory methods do not always yield the same results. Furthermore, the literature suggests that if resistance exercise is properly executed and well supervised, it holds no risks for hypertensive individuals. The benefits gained from resistance exercise form a major part in the maintenance of healthy metabolic rate as well as muscular strength and endurance.

2. Effect of acute and chronic aerobic exercise (AR) on changes in blood pressure

Aerobic exercise is described as the performance of repeated sessions of dynamic exercise, using large muscle groups, such that there is a considerable rise in heart rate as well as energy expenditure over an extended period of time (Howley, 2001). Acute effects of aerobic exercise refer to changes that occur following a single session while chronic (training) effects is associated with changes that occur over time as a result of adaptations in the various body systems (Howley, 2001). It is well known that BP decreases after aerobic exercise and that the response is most pronounced in hypertensive patients. Some studies have also reported positive responses in normotensive subjects (Carvalho *et al.*, 2009; Ciolac *et al.*, 2008; Cornelissen and Fagard, 2005; Pescatello and Kulikowich, 2001), while Gilders *et al.* (1989)

reported no change in resting or ambulatory BP when hypertensive adults performed 16 weeks of aerobic exercise training at 80% HR_{max}.

Whelton *et al.* (2002) limited their meta-analysis to randomized controlled trials. 54 clinical trials were included which involved 2419 participants across a wide range of ethnic and geographic regions. Their results revealed that the acute response of aerobic exercise significantly reduced SBP by 3.8 mmHg and DBP by 2.6 mmHg.

Forjaz *et al.* (2000) investigated the effect of a single bout of aerobic exercise on ambulatory BP, as well as possible factors that influence the PEH response in normotensive and hypertensive humans. Subjects performed a control session (45 min of rest) and exercise session (45 min of bicycling at 50% VO_{2max}) in random order. Their results showed a significant decrease in SBP (113 mmHg vs. 115 ± 2 mmHg) and DBP (69 mmHg vs. 70 mmHg) over 24 hours after the exercise session in normotensive subjects, but no significant decrease in BP in their hypertensive counterparts. However, individual response data revealed that 65% of the hypertensive subjects showed lower BP during recovery. Furthermore, younger individuals who had higher VO_{2max} and resting BP values, as well as lower BMI responded positively to the exercise, i.e. showed a PEH response. The authors concluded that the observed post-exercise ambulatory fall in BP depended on individual characteristics in both normotensive and hypertensive humans. Moreover, the post-exercise ambulatory hypotension was greater in those normotensive individuals with a higher initial blood pressure level and lower BMI.

A meta-analysis by Cornelissen and Fagard (2005) concluded that SBP and DBP are reduced by 6.9 and 4.9 mmHg, respectively, through aerobic exercise training in hypertensive patients, compared to 2.4 and 1.6 mmHg, respectively, in non-hypertensive individuals.

Additionally, Jessup *et al.* (1998) investigated the effects of 16 weeks endurance training in normotensive subjects aged between 61 to 77 years. The subjects performed exercise three times per week on a treadmill and stair climbing at an intensity of 50% HR_{max} for 25 min and progressed gradually to 85% HR_{max} for 45 min during the final four weeks. Although there were no statistically significant decrease in resting BP after training, both SBP and DBP decreased significantly during 24 hours recovery period after training (SBP: 7.9 mmHg and DBP: 3.6 mmHg). At the same time, there were no changes in the BP responses of the control group. This study makes the important conclusion that aerobic exercise reduces the day to day BP in healthy older adults which could prevent the steady rise in BP associated with ageing.

Tsai *et al.* (2002) examined the effect of a 12 week aerobic exercise training programme on resting BP in hypertensive, middle-aged subjects who performed 30 min of walking/jogging on the treadmill (60 – 70% HR_{max}) three times per week. They showed a significant reduction in SBP of 18 mmHg (pre: 139.1 mmHg vs. post: 121.1 mmHg) and DBP of 10 mmHg (pre: 99.5 mmHg vs. post: 89.5 ± 8.2 mmHg). The results indicated that the high BP in hypertensives can decrease to normal levels after an endurance exercise which can serve as an antihypertensive effect. Similarly, Kiyonaga *et al.* (1985) showed significant reductions of 14 mmHg in SBP (pre: 153 mmHg vs. post: 139 mmHg) and 9 mmHg in DBP (pre: 103 mmHg vs. post: 94 mmHg) after 10 weeks of moderate intensity (50% HR_{max}) aerobic exercise in essential hypertensive adults. After 20 weeks of exercise, a further decrease of 3 mmHg and 4 mmHg was observed in the SBP and DBP respectively, indicating that aerobic exercise training promotes the hypotensive effect even more with longer periods of training. Even though there was significant reductions in BP, not all subjects responded positively to the exercise. The observed reduction in BP indicates that the responders showed lower plasma renin values, which could have contributed to the larger decrease in BP.

It can be concluded that aerobic exercise training induces PEH responses in resting and ambulatory BP in both normotensive and hypotensive subjects. Even though there are some studies that did not observe significant reductions in BP after exercise, the majority of the literature are consistent with the fact that BP decreases after aerobic exercise training. Moreover, the differences in the BP changes after exercise training can be ascribed to disparities in study design and methodological issues specially the exercise protocol. Also reason for unresponsiveness to BP reduction could be attributed to the genetic makeup of the individual subject as observed in the study of Kiyonaga *et al.* (1985).

3. Effect of water exercise on changes in blood pressure

Water exercise is one approach of exercise that has in recent years been recognized as a treatment therapy for individuals with chronic heart disease (Terblanche and Millen, 2012). An individual's body weight is decreased by the buoyancy of the water by about 90%. This minimizes the mechanical stress imposed on the limbs and makes it possible for those with injuries or weakness of the lower limbs, as well as those who have low levels of fitness, to train safely. Furthermore, temperature regulation during exercise may also be improved due to heat exchange between surrounding water and the body (Barbosa *et al.*, 2007). Muscle strength may be improved by moving against the water's resistance, while SV and CO are increased due to the redistribution of the blood to the thoracic cavities as a result of the hydrostatic pressure (Cider *et al.*, 2005; Park *et al.*, 1999). Previous research in normotensive and hypertensive individuals have shown an increase in BP (Park *et al.*, 1999), a reduction (Terblanche and Millen, 2012) or unchanged values (Cider *et al.*, 2005) following exercise by immersion in thermo-neutral water. Thus the true effect of water exercise on the post-exercise BP response is still unclear.

Terblanche and Millen (2012) compared the magnitude and duration of PEH during free-living conditions after acute sessions of concurrent water and land exercise in pre-hypertensive and hypertensive patients. Participants completed exercise sessions in thermo-neutral water, on land, as well as a no exercise control session, in random order as their BP was monitored 24 hours after all three sessions. The land exercise consisted of concurrent aerobic (30 min at 60 - 80% VO_{2max}) and resistance (25 min of 2 sets of 10 repetitions) exercises. Water exercise included mainly aerobic exercise, combined with movements against the resistance of the water. They observed a significant reduction in SBP (land: 12.7 and water: 11.3 mmHg) compared to the control session (2.3 mmHg) during daytime. The duration of the hypotensive response lasted 9 hours after water exercise, compared to nearly 24 hours after the land exercise. There was no difference in the daytime DBP for the three treatments. Although all three groups showed significant reductions during nighttime, both exercise treatments showed greater nocturnal falls in BP compared to the control treatment.

Cider *et al.* (2005) investigated the acute cardiorespiratory effect of immersion in warm water in elderly patients with chronic heart failure (CHF) matched with healthy subjects as a control. The subjects completed both land and water exercise in random order within 10 days. The land session consisted of a 5 min rest followed by 4 min of seated submaximal leg extensions without external weight applied, with a cadence of 60 extensions per minute. The water exercise followed a similar pattern, but participants had to walk 12 m in the water before they sat in a chair. The authors observed no significant difference in BP after both land and water exercise compared to their respective baseline values. Both exercise sessions had no effect on the BP response. One must consider that, although the intensity of the exercise protocol was not indicated in this study, it is possible that the exercise program was not enough to stimulate hypotensive response.

The available literature is limited on the effect of water exercise on the PEH response and existing studies report contrasting results. The different results could be explained by the type of exercise protocols used, especially with regards to the intensity of exercise, total work done, as well as the duration of the exercise session. For instance, Terblanche and Millen (2012) considered all these variables, but Cider *et al.* (2005) did not. However, it is promising that water exercise might be an equally effective alternate to land based exercise in healthy and diseased individuals. More research is needed in this area.

4. Effect of concurrent exercise on changes in blood pressure

It is known that PEH is evident immediately after aerobic exercise (AE) and may persist for up to 24 hours after exercise (Keese *et al.*, 2011; Cornelissen *et al.*, 2010; Dujic *et al.*, 2006; Pescatello *et al.*, 2004b; MacDonald, 2002; MacDonald *et al.*, 2000). This is also evident after resistance exercise (RE), even though the magnitude and duration of the BP lowering effects are perhaps less after resistance exercise compared to aerobic exercise (Keese *et al.*, 2011; Ruiz *et al.*, 2011; Forjaz *et al.*, 2004). Concurrent exercise is a combination of both AE and RE (Keese *et al.*, 2012). In view of the fact that concurrent exercise may improve muscle strength and cardio-respiratory fitness at the same time, there is ample reason to promote this type of exercise (Keese *et al.*, 2012), as less time is spent exercising and the physiological benefits may also be greater compared to aerobic or resistance training only. Although there are few studies on the effect of concurrent training on the BP response, the findings so far are in agreement (Keese *et al.*, 2011; Ruiz *et al.*, 2011; Teixeira *et al.*, 2011).

Keese *et al.* (2011) compared the immediate effects of resistance (RE), aerobic (AE) and concurrent exercise on PEH among normotensive individuals. They showed a significant decrease in SBP (5.1 mmHg) and DBP (1.6 mmHg) which lasted 120 min and 40 min,

respectively after bouts of concurrent exercise. The magnitude of the hypotensive effect in concurrent exercise was similar to aerobic exercise (SBP: 6.3 mmHg and DBP: 1.8 mmHg) and resistance exercise (SBP: 4.1 mmHg and DBP: 1.8 mmHg), but the duration of the PEH in DBP was longer than in RE (SBP/DBP: 80/20 min) and slightly shorter than following the AE (SBP/DBP: 120/50 min). It was concluded that exercise sessions combining aerobic and resistance activities are as effective as AE only and more effective than RE only to promote PEH.

Keese *et al.* (2012) further reported on the influence of different intensities of the aerobic segment of concurrent exercise sessions together with the same amount of RE on the PEH among subjects with normal BP. Subjects performed four sessions of exercise: control session (CTL: rest for 60 min), and three exercise sessions consisting of 2 sets of 6 exercises at 80% 1RM followed by 30 min of cycle ergometer exercise at 50% (CE1), 65% (CE2), and 80% (CE3) VO_{2max} . The magnitude of reduction in SBP was similar after all CE sessions (CE1: 4.2 mmHg; CE2: 4.8 mmHg; CE3: 6.0 mmHg), but the hypotension response lasted significantly longer (about 1 hour) after CE2 and CE3 (2 hours) compared to CE1 (60–70 min). There were no differences in the magnitude of the DBP responses between CE2 and CE3 (1.5 mmHg and 1.8 mmHg, respectively), but reductions in DBP following CE3 was greater than after CE1 (1.2 mmHg) and lasted for a longer period of time after CE3 (60 min) compared to CE2 and CE1 (40 min). It can be concluded from this study that concurrent training elicits a significant PEH response, especially when the intensity of the aerobic exercise component is higher than 65% VO_{2max} . These findings are consistent with previous results indicating that the higher the exercise intensity, the greater the PEH response.

Ruiz *et al.* (2011) investigated the effect of different training modalities using separate and combined aerobic and resistance exercise on the PEH response in normotensive men.

Participants performed three training sessions with 2 to 3 days in between training sessions in a counterbalance crossover design. Training sessions comprised a strength training session (STS) in which subjects completed 3 sets of 12RM for each exercise, an aerobic training session (ATS) in which participants did 40 min cycling (60 – 70% HR_{max}) and combined aerobic and strength training (ASTS) in which subjects did similar exercise in ATS and STS in a sequence. Their results showed significant reductions in SBP during recovery for all training modalities, however, the magnitude of reduction at the 60th min was greater after ASTS (8.0 mmHg) than after ATS (4.8 mmHg) and STS (4.5 mmHg) compared to the baseline. Reduction in DBP was not significant. This indicated that concurrent exercise induced a PEH response in SBP that may even be greater compared to resistance or aerobic exercise in isolation.

Teixeira *et al.* (2011) evaluated the PEH response after a single session of aerobic (A), resistance (R), and concurrent (AR) exercises in young normotensive subjects. Subjects performed three exercises (A, R and AR sessions) and a control session in random order over 5 days. Their results showed significant reductions in SBP/DBP after all the exercises, but the higher reductions occurred after the aerobic and concurrent exercise (-13/-3 mmHg, -8/-2 mmHg and -11/-3 mmHg, respectively after A, R and AR compared to rest values) with no change in SBP after control sessions and an increase in DBP compared to rest values (+4 mmHg; $p < 0.05$). In a similar study, Shaw *et al.* (2010) investigated the effect of concurrent training in sedentary young normotensive adult men on BP. Subjects either completed aerobic exercise training or concurrent training for 60 min a day, 3 days per week for 16 weeks. Subjects were divided into three groups in the following order; control group, aerobic exercise at 60% HR_{max} , or an aerobic (60% HR_{max}) and resistance training (8 exercises; 2 sets of 15 repetitions at 60% 1RM) combined group. Both aerobic and concurrent exercise showed significant decreases in SBP. However, the observed reductions in BP following the

concurrent exercise was greater in magnitude (9.8 mmHg), while the aerobic group demonstrated smaller decreases in BP (3.9 mmHg). These findings are thus in agreement with other studies that concurrent exercise produces a hypotensive response, similar in magnitude, but potentially even with greater magnitude to aerobic and resistance exercise alone.

Deducing from the literature, it is certain that the combination of aerobic and resistance exercise lower BP similarly to aerobic or resistance training in normotensive and hypertensive individuals. Furthermore, some studies suggest that concurrent training can even be more effective to lower BP after exercise than separate aerobic or resistance exercise and can thus be successfully used as antihypertensive treatment.

5. Effect of high intensity interval training (HIIT) on changes in blood pressure

HIIT has been investigated to show its effectiveness in the improvement of health and the related benefits including fat loss (Trilk *et al.*, 2011; Whyte *et al.*, 2010; Tjønnå *et al.*, 2009; Warburton *et al.*, 2005) and improved performance (Esfarjani and Laursen, 2007; Laursen *et al.*, 2002), as well as improved insulin sensitivity (Hood *et al.*, 2011). However, it is not known whether the same is true for PEH following HIIT.

Rossow *et al.* (2010) compared the PEH response following acute steady state (SS) and HIIT in endurance trained men and women. SS comprised 60 min of continuous cycling at 60% HRR and HIIT comprised four 30 sec “all-out” cycling bouts interspersed by 4.5 min light active recovery. The authors found significant decreases in mean SBP/DBP by magnitude of 5.6/5.1 mmHg after HIIT and 5.1/3.9 mmHg after SS following a single exercise session from pre-exercise values. Similarly, MAP significantly decreased by 4.4 mmHg after HIIT

and 3.8 mmHg after SS from pre exercise values. However, the difference between modes of exercise was not statistically significant. These authors upon assessing other hemodynamics after the exercise sessions explained that the observed PEH following HIIT was due to an increased CO that was offset by a greater reduction in TPR following HIIT. Therefore the finding of this study is an indication that an acute session of HIIT is capable of provoking PEH similarly to steady state exercise.

Previously discussed under the summary of factors that affect the magnitude of PEH (page 20), Eicher *et al.* (2010) reported a reduction in SBP and DBP in overweight/obese individuals, following exercise at different intensities ranging from low (40% VO_{2max}), moderate (60% VO_{2max}) and high (100% VO_{2max}). The results showed that SBP slightly decreased by 2.7 mmHg following low intensity exercise, but the reduction in SBP was statistically significant following moderate and high intensity exercise (5.4 mmHg and 11.7 mmHg respectively, compared to control levels). Likewise, DBP declined by 1.5 mmHg and 2.0 mmHg respectively after low and moderate intensity exercise. However, only the high intensity exercise resulted in a statistically significant reduction by 4.9 mmHg. The results indicate that PEH after high intensity was greater than moderate and low intensity exercises. This suggests that high intensity exercise may have beneficial effects on BP.

Likewise Nyberg *et al.* (2012) reported an 8.8 mmHg reduction in SBP (pre: 182.8 mmHg vs. post: 174 mmHg) and 12 mmHg reduction in DBP (pre: 101.1 mmHg vs. post: 89.0 mmHg), as well as 9.1 mmHg in MAP (117.5 mmHg vs. 126.6 mmHg) in subjects with essential hypertension following high intensity aerobic training. These authors did not describe the type of training protocol in terms of intensity, duration and volume even though they reported that participants performed exercise on the cycle ergometer for two to three times per week.

Ciolac *et al.* (2010) compared the effects of high intensity aerobic interval training (AIT) and continuous moderate intensity exercise (CMT) in young normotensive women. The subjects were randomly assigned to AIT, CMT or 2 control groups. The AIT group performed 1 min running bouts at intensity of 80 – 90% VO_{2max} alternated with 2 min active recovery at an intensity of 50 – 60% VO_{2max} for 40 min. CMT completed 40 min of walking at an intensity of 60 – 70% VO_{2max} . Control groups continued their normal daily activities. The exercise groups trained three times a week for 16 weeks with 15 min of additional calisthenics to every exercise session, thus a total of 55 min per session. At the end of the intervention the exercise groups improved their BP response to exercise. SBP significantly decreased (mean SBP pre/post AIT: 113.3/111.3 mmHg and pre/post CMT: 112.6/109.9 mmHg), as well as DBP (mean DBP pre/post AIT: 71.1/69.1 mmHg and pre/post CMT: 69.4/67.3 mmHg) compared to baseline values. The control groups showed no significant reduction in BP compared to the pre-exercise values. Upon comparing the improvement in other hemodynamic variables that were assessed in addition to BP, these authors concluded that high intensity aerobic interval exercise was superior to CMT.

Whyte *et al.* (2010) employed the Wingate anaerobic test protocol which consisted of 15 min of 6 x 30 sec bouts “all-out” efforts of sprinting on a cycle ergometer interspersed by 4.5 min of active recovery for two weeks in sedentary overweight/obese men. Their results showed a significant reduction in SBP by a magnitude of 6 mmHg post-exercise compared to baseline level following 24 hours post-exercise. However, the reduction in DBP only approached statistical significance ($p < 0.06$) compared to baseline. Since the reduction in BP was sustained for 24 hours, it can be concluded that HIIT poses a hypotensive response even with such short duration activity.

Chan and Burns (2013) also investigated among ten healthy males, post exercise blood pressure following sprint interval exercise (SIE). Subjects undertook two trials in a randomly balanced order (SIE and the control (C) without exercise). The SIE session was completed by 4 x 30 sec bouts on the cycle ergometer interspersed by 4.5 min active recovery (Wingate test protocol). During 2 hours post BP measurements, the authors observed a significant reduction of 8 mmHg in SBP (C: 117 mmHg vs. SIE: 109 mmHg) and DBP was lowered by 6 mmHg (C: 84 mmHg vs. SIE: 77 mmHg) following SIE upon comparison to the control period. The significant reduction in BP sustained for 2 hours is an indication that HIIT is capable of causing a hypotensive response.

Moreover, Nybo *et al.* (2010) determined the effectiveness of brief intense interval training as an exercise intervention for promoting health and evaluated the potential benefits of prolonged exercise and strength training. The subjects were grouped into four groups. These included: 1) intense interval running (INT) group in which subjects performed 15 min of five 2 min bouts of running at intensity above 95% of HR_{max} separated by 2 min of rest; 2) strength-training (STR) group who completed an hour of progressive heavy resistance strength training with one minute rest interval (12–16 repetition maximum (RM) during the first four weeks and 6–10 RM during the remaining eight weeks) of the training period, with the absolute loads gradually adjusted to match the individual progressions in muscle strength; 3) moderately intense running (MOD) group consisting of an hour of continuous running at 80% HR_{max} and 4) a control (CON) group performing no physical training but continued their daily life activities during the period. The intervention lasted for 12 weeks as training sessions were scheduled for three sessions per week. Their results showed that SBP was significantly decreased by a magnitude of 8 mmHg in all three training sessions performed. DBP was only significantly reduced following MOD (5 mmHg), but to a lesser extent in INT (2 mmHg). MAP significantly decreased by 3 mmHg following INT and 6 mmHg following

MOD and 5 mmHg following STR. It was revealed that short but intense training possibly decreased arterial BP. Thus, the group that completed the high intensity training intervention had a significant reduction in SBP and as a result reduced their MAP. The 8 mmHg reduction in SBP was the same as the changes in the moderate and strength training sessions, while the effect on DBP appeared to be less after the high intensity interval training session compared to the moderate training session. They explained from a statistical perspective that due to relatively few subjects involved in the study, they could not conclude whether HIIT is less or equally effective compared to prolonged moderate intensity exercise interventions.

Burns *et al.* (2012) investigated the effects of HIIT on post exercise metabolism and BP in adolescents (15 – 18 years). All subjects performed two tests on separate days; (1) Wingate anaerobic tests which consisted of 15 min of two 30 sec “all-out” efforts of sprinting on a cycle ergometer interspersed by 4 min rest and (2) a control session where participants rested in the laboratory for 15 min. Their results showed a significant reduction in SBP (pre exercise: 104 mmHg, post exercise: 100 mmHg) compared to control level (pre exercise: 101 mmHg, post exercise: 102 mmHg) after 90 min of post exercise. However, the reduction in DBP was not different from control values. Even though this was an acute session, the reduction in SBP suggests that HIIT is capable of causing a hypotensive response since the decrease was sustained for 90 min. Moreover, the hypotensive response concurs with adults studies. Similarly, Tjønnå *et al.* (2009) also reported a 9.4 mmHg reduction in SBP and 5.5 mmHg reductions in DBP in overweight adolescents compared to baseline after the completion of HIIT. In this case, the HIIT protocol comprised 4 x 4 min intervals at 90% of HR_{max} and separated by 3 min active recovery at 70% of HR_{max} two times per week for a period of three months. The findings of these two studies (Burns *et al.* 2012; Tjønnå *et al.* 2009) indicate significant effects on BP which led to a hypotensive response and also showing that HIIT is tolerable in adolescents.

Astorino *et al.* (2012) on the other hand, observed no reduction in resting blood pressure in healthy normal weight recreationally active men and women who performed Wingate tests consisting of 4 to 6 sessions of 30 sec “all-out” efforts on the cycle ergometer with active recovery of 5 min twice a week for three weeks. The number of training sessions increased by one bout every two sessions: on day 1 and 2, participants performed 4 x 30 sec sprints; day 3 and 4, participants performed 5 bouts and lastly 6 bouts for day 5 and 6. Their lack of a positive response in BP could be as a result of the training protocol. Even though they adopted similar high intensity training protocol than other researchers, the frequency of sessions per week could explain the difference, as the number of rest days between sessions outnumbered the training days. As a result any reduction in BP would have vanished. It is therefore important that exercise be performed on most days of the week for effective results.

Moreover, Tjønnå *et al.* (2008) compared the effect of training mode based on intensity in treating metabolic syndrome patients. Metabolic syndrome patients are more than three times likely to die of heart disease than healthy individuals. These authors randomly divided patients in three groups; continuous moderate intensity exercise group (CME; completed 47 min uphill running on the treadmill at 70% of HR_{max}), aerobic interval training group (AIT; performed 4 bouts of 4 min at 90% of HR_{max} interspersed by 3 min active recovery at 70% of HR_{max} on the treadmill) and the third control group who only followed their physician’s advice with no specific exercise training. The exercise intervention was 3 days per week for 16 weeks. At the end of the training intervention and compared to baseline, patients in the AIT group improved their body composition significantly by 2.4 kg (body mass) and 0.7 $kg \cdot m^{-2}$ (BMI). SBP and DBP were also significantly reduced by 9 and 6 mmHg respectively with a 6 mmHg reduction in MAP. VO_{2max} also increased significantly by 35%. Considering the results of a meta-analysis of one million adults (Lewington *et al.*, 2002), a BP reduction

of such magnitude would, in the long-term, be associated with 40% and 30% reductions in the risks of deaths resulting from stroke and heart conditions.

However, literature supporting the hypotensive effects of HIIT is not as much as compared to studies done on other types of exercise. Of the eleven studies reviewed only one study (Astorino *et al.*, 2012) observed no change in BP which could be explained by the small number of session days performed in a week compared to the number of rest days between sessions. However, this explains why exercise training on most days of the week is advised for the accumulation of long-term adaptations. Two studies (Burns *et al.* 2012; Tjønnå *et al.* 2009) reported significant reductions in BP in adolescents even though this population typically has lower resting BP compared to adults. One study (Tjønnå *et al.*, 2008) reported a PEH response in coronary artery bypass patients and two studies (Nyberg *et al.*, 2012; Eicher *et al.*, 2010) reported PEH in hypertensive individuals, which also indicate that HIIT is tolerable in the patient population. It can be assumed that the varied HIIT protocols used in these studies (in terms of duration, intensity and volume), which may have been selected with the study population in mind, may explain the different reported magnitudes of PEH. On the whole it can be concluded that HIIT stimulate physiological adaptations in the human body, similarly to continuous endurance training, but with little time spent.

F. CONCLUSION

From the available literature it is not certain as to the complete mechanism that accounts for PEH, however, it is likely that a series of physiological mechanisms work together to promote PEH. Moreover, it is certain that BP is positively affected by exercise training, despite the fact that there are several issues regarding PEH that still remains unclear, for example the exact mechanisms underlying PEH. PEH is well documented in both

normotensive and hypertensive individuals and have been demonstrated to have positive effects that cannot be denied. Different types of exercise have also been shown to invoke PEH, even though there are some contrasting results in the literature. Inconsistent results can probably be attributed to the population being studied, or the differences in the methodological protocol adopted, i.e. variations in the intensity, frequency and duration of exercise sessions and training programs. It can be concluded that exercise training is a vital non-pharmacological approach in lowering BP specifically in treating hypertension.

CHAPTER THREE

HIGH INTENSITY INTERVAL TRAINING

A. INTRODUCTION

It has been clinically shown that exercise training is a vital interventional approach that most often prevents or at least delay the onset of health problems related to many chronic diseases. However, the precise type and dose of exercise needed to accumulate health benefits is still a contentious issue, with no clear agreement regarding recommendations for the prevention of diseases of lifestyle. To add to the confusion, a growing body of evidence reveals that HIIT is an effective alternative to load-matched traditional endurance-based training, which provides similar or even better physiological adaptations, as well as performance and health-related indicators in both healthy individuals and diseased populations (Hwang *et al.*, 2011; Tjønnha *et al.*, 2009; Wisløff *et al.*, 2007).

HIIT depicts an exercise training method that is characterized by short (10 sec to 5 min), repeated bouts of vigorous activity, executed at or near peak oxygen uptake, and interspersed by periods of rest or low-intensity exercise. Most interventions in the literature lasted between two to six weeks. The main idea behind HIIT is to allow for completion of maximal work load in a session by separating the work done in rest intervals (Kemi, and Wisløff, 2010). This is to allow the physiological systems to be overloaded with exercise intensities greater than those achieved during a progressive maximal aerobic capacity test (Stuckey *et al.*, 2011). Literature suggests that HIIT stimulates physiological remodeling similar to moderate intensity continuous training, despite the considerable shorter time commitment and reduced exercise volume (Gibala and McGee, 2008). Outcomes of this nature of exercise are of significance due to the fact that 'lack of time' is usually claimed as a barrier to regular exercise participation (Kimm *et al.*, 2006; Stutts, 2002; Trost *et al.*, 2002).

According to the American College of Sports Medicine moderate physical activity of 150 to 250 min per week is sufficient and effective to avoid weight gain (Donnelly *et al.*, 2009). These recommendations have been endorsed and supported by the Centers for Disease Control and Prevention as well as the Committee on Exercise and Cardiac Rehabilitation of the American Heart Association (Haskell *et al.*, 2007). Nevertheless, people fail to accomplish these traditional exercise recommendations, possibly because these types of training programs require at least 45 to 60 min exercise sessions. There is evidence however, that short intense exercise training may provoke similar improvements in weight control (Whyte *et al.*, 2010) and cardiorespiratory fitness as prolonged training (Burgomaster *et al.*, 2008; Gibala, 2007).

B. TYPES OF HIGH INTENSITY INTERVAL TRAINING

Various types of HIIT programs have been developed to suit the different population groups and the majority was able to demonstrate potential for enhanced physical activity participation and greater feelings of perceived enjoyment. Such positive affective feelings are induced by HIIT compared with the continuous endurance training irrespective of matched average intensity, duration, and work done for both training protocols (Bartlett *et al.*, 2011), especially for individuals who do not participate in physical activity for various reasons. There are two major types of HIIT protocols which include cycling and running.

1. Cycling protocol

In this type of HIIT protocol, the Wingate test is typically used. It consists of a 30 sec “all-out” cycling effort against a supramaximal workload. Four to six bouts are completed

interspersed by 4 min of recovery at 70% HR_{max} , giving a total of 2 to 3 min of intense exercise during a training session that lasts 20 min. During the 30 sec bouts, the oxygen independent energy systems are mainly used whilst the 4 min recovery periods engage mainly the oxygen dependent energy system (Gibala, 2007). Several studies (Chan and Burns, 2013; Burns *et al.*, 2012; Little *et al.*, 2011; Psilander *et al.*, 2010; Whyte *et al.*, 2010; Burgomaster *et al.*, 2005) utilizing this type of HIIT have been shown to promote metabolic adaptations as well as enhance aerobic capacity and performance (Hazell *et al.*, 2012; Hazell *et al.*, 2010).

Little *et al.* (2011) examined the mechanisms controlling the adaptive response to HIIT in normally active men through the determination of levels of peroxisome proliferator-activated receptor γ coactivator (PGC-1 α) (a key regulator of mitochondrial biogenesis) in response to an acute bout of HIIT (four 30 sec maximal cycling interspersed with 4 min rest periods). They showed significant increases in enzyme activities of citrate synthase (CS) (quantitative enzyme marker for presence of intact mitochondria) by 14% and cytochrome c oxidase (COX) (facilitates cellular energy transfer in the synthesis of energy in mitochondrial electron transport chain) increased by 19%, but this could not reach statistical significance during 24 hours recovery following exercise. PGC-1 α increased significantly by 66% after 3 hours recovery, but decreased to baseline values after 24 hours. The results suggested that HIIT is effective in provoking the activities of enzymes and the key regulators in mitochondrial biogenesis involved in aerobic metabolism, thus maximizing the individual's aerobic capacity. However, these adaptive responses are short lived and therefore the accumulation of several bouts of training is needed.

Psilander *et al.* (2010) also showed that a single session of low-volume HIIT (7 bouts x 30 sec 'all-out' efforts interspersed by 4 min of active rest at 50 W) provoked significant

increases in genetic markers for mitochondrial biogenesis that were comparable to the changes after more prolonged endurance sessions (3 x 20 min bouts at ~87% of VO_{2max}) in well-trained cyclists. Considering the low-volume of work and the fact that mitochondrial genetic markers of mitochondrial biogenesis increased the downstream target of PGC-1 α , the authors concluded that brief intense interval training might be a time-efficient strategy for highly trained individuals.

Burgomaster *et al.* (2005) examined the effect of sprint interval training on muscle oxidative potential, VO_{2max} and time to fatigue during cycling among 16 healthy recreationally active individuals. They assigned participants into a sprint interval training, SIT group (6 sessions of 7 x 30 sec “all-out” efforts of cycling separated by 4 min passive recovery, 3 days per week for two weeks) or control group. After the intervention, the maximal activity of CS increased significantly by 38% (pre: 4.0 mmol.kg protein⁻¹.h⁻¹ vs. post: 5.5 mmol.kg protein⁻¹.h⁻¹) in the SIT group, and resting muscle glycogen content increased by 26% (pre: 489 mmol.kg⁻¹ DW vs. post: 614 mmol.kg⁻¹ DW). Although VO_{2max} did not change, expired minute ventilation was significantly lower (pre: 104 L.min⁻¹ vs. post: 91 L.min⁻¹) along with the respiratory exchange ratio (pre: 1.24 vs. post: 1.18). Time to fatigue also increased significantly by 100% (pre: 26 min vs. post: 51 min) in the SIT group. The control group showed no significant changes in any of the measured parameters. The increased activity of CS indicates the potential of SIT to improve the endurance capacity of the individuals.

Hazell *et al.* (2010) assigned 48 young adults into four groups based on their VO_{2max} , gender and 5 km time trial performance following two weeks of either one of the following “all-out” efforts sessions: 1) 4 – 6 x 30 sec x 4 min active recovery, 2) 4 – 6 x 10 sec x 4 min active recovery and 3) 4-6 x 10 sec x 2 min active recovery, and lastly a control without exercise group. All exercise sessions were performed at a cycling resistance of 100 g.kg⁻¹ body mass

and active recovery of no resistance (i.e. unloaded cycling). Time trial for 5 km cycling was ~9.5 min for all groups at baseline. At the end of the two weeks intervention, participants in the exercise groups significantly improved their performance in the time trial; 5.2% (-28.9 sec; $p < 0.00$) in the 30 sec x 4 min group, 3.5% (-19.8 sec) in 10 sec x 4 min group, and 3.0% (-15.7 sec) in the 10 sec x 2 min group. The control group showed no change in performance. Likewise, VO_{2max} was significantly increased by 9.3% ($4.3 \text{ ml.kg}^{-1}.\text{min}^{-1}$) in the 30 sec x 4 min group and 9.2% ($4.5 \text{ ml.kg}^{-1}.\text{min}^{-1}$) in the 10 sec x 4 min group. In the 10 sec x 2 min group, the increase in VO_{2max} approached statistical significance (3.8%; $1.8 \text{ ml.kg}^{-1}.\text{min}^{-1}$; $p = 0.06$), while the control group showed no significant change in VO_{2max} (from 45.2 to 45.3 $\text{ml.kg}^{-1}.\text{min}^{-1}$). The results suggest that even though all exercise groups showed significant improvements in both VO_{2max} and time trial performance, the 30 sec x 4 min group showed the greatest improvements. It is clear that the volume of the work load (i.e. in terms of the duration of the exercise bout) is a determining factor in the magnitude of the performance changes. These findings support the notion that adaptations and improvement to HIIT is dependent on the intensity, duration of bout per session and volume of the work load.

Hazell *et al.* (2012) compared the oxygen consumption of healthy young active men during and after 24 hour sprint-interval exercise (SIE; 4 bouts of 30 sec maximal cycling efforts at a resistance of 10% of body mass separated by 4 min active recovery at 0% resistance) against 30 min of continuous endurance cycling exercise (CEE) at 70% VO_{2max} . The intervention lasted six weeks; participants trained three times per week and the aim of the programme was to promote weight loss. Total oxygen (O_2) inspired over 8 hours of recovery significantly increased after both exercises (CEE: 263.3 L and SIE: 224.2 L) compared to baseline (CTRL: 163.5 L). But CEE was 17% higher than SIE and 61% higher than CTRL, while SIE was 37% higher than CTRL. At a 24 hour recovery, VO_2 remained significantly higher after both exercise sessions, however CEE did not differ significantly from SIE. Even though total O_2

consumed during the exercise session was greater in CEE than SIE (87.6 L vs. 35.1 L, respectively), the post-exercise metabolism during the rest of the day resulted in similar total O₂ uptakes over 24 hours after both exercise sessions. However, the exercise RER was greater in SIE than during CEE (CEE: 0.90 vs. SIE: 1.20) despite a non-significant difference in exercise HR (CEE: 157 b.m⁻¹ vs. SIE: 149 b.m⁻¹). This indicated that SIE significantly helped participants to lose body fat due to the rise in metabolism after exercise. The authors speculated that SIE stimulated oxidative activities in the mitochondria more than CEE, which would mediate the oxidation of fat.

1.1. Positive and negative effects of cycling HIIT

It is evident that this type of HIIT protocol is time efficient as less time is spent to acquire adaptive responses compared to the continuous endurance training (Ball *et al.*, 2006). Moreover, this cycling protocol is well tolerated by participants as they do not experience feelings of dizziness, light headedness or nausea after exercise (Little *et al.*, 2011). However, the negative side of this type of training is that, it is performed on a specialized cycle ergometer and the protocol requires high resistance which is very demanding. It would therefore not be suitable for all sick individuals. Having said this, some studies have successfully utilized this protocol in hypertensive patients (Nyberg *et al.*, 2012; Eicher *et al.*, 2010).

2. Running protocol

This type of HIIT is usually performed on the treadmill and has also been shown to be well tolerated by patient populations (Wharburton *et al.*, 2005) such as those with hypertension

(Molmen-Hansen *et al.*, 2011), metabolic syndrome (Tjønnna *et al.*, 2008) and coronary artery disease (Moholdt *et al.*, 2009). All these studies reported positive results in terms of reducing cardiovascular risk factors and improvements in fitness levels. This protocol consists of 1 to 4 min work bouts at a running velocity of 90 - 100% VO_{2max} , interspersed by 1 to 4.5 min recovery periods at 50 - 70% VO_{2max} for a total of 20 - 30 min.

Helgerud *et al.* (2007) showed that high intensity aerobic exercise is superior to moderate intensity training in improving VO_{2max} in healthy young trained men. Subjects were randomly assigned to either one of the following groups: 1) long slow distance, LSD (45 min continuous run at 70% HR_{max}); 2) Lactate threshold running, LT (24.25 min continuous run at lactate threshold; 85% HR_{max}) 3) 15/15 interval running, (4 x 15 sec intervals at 90 – 95% HR_{max} separated by 15 sec active recovery at 70% HR_{max}) and 4) 4 x 4 min interval running, (90 – 95% HR_{max} interspersed by 3 min active recovery at 70% HR_{max}). All sessions were performed on the treadmill, three times per week for eight weeks. There were statistically significant improvements in VO_{2max} for the 15/15 group (pre: 60.5 $ml.kg^{-1}.min^{-1}$ vs. post: 64.4 $ml.kg^{-1}.min^{-1}$), as well as the 4 x 4 min group (pre: 55.5 $ml.kg^{-1}.min^{-1}$ vs. post: 60.4 $ml.kg^{-1}.min^{-1}$), while no significant changes for VO_{2max} were observed for the LSD (pre: 55.8 ± 6.6 vs. post: 56.8 ± 6.3 $ml.kg^{-1}.min^{-1}$) and LT groups (pre: 59.8 ± 6.6 $ml.kg^{-1}.min^{-1}$ vs. post: 60.8 ± 7.1 $ml.kg^{-1}.min^{-1}$). There was no significant difference in the improvement of VO_{2max} between the interval groups (15/15 and 4 x 4 min). The increases in VO_{2max} were probably facilitated by increases in SV, as both the 15/15 group (pre: 1.8 $ml.kg^{-1}.b^{-1}$ vs. post: 1.99 $ml.kg^{-1}.b^{-1}$) and the 4 x 4 group (pre: 1.8 $ml.kg^{-1}.b^{-1}$ vs. post: 1.98 ± 0.5 $ml.kg^{-1}.b^{-1}$) experienced statistically significant increases in SV. On the other hand, there were no changes in SV recorded for the LSD and LT groups. The results suggest that intensity of training determines the training response. It can be concluded that HIIT is significantly more effective than performing the same total work at moderate intensity in improving VO_{2max} .

However, the results of this study contrast with the results of Hazell *et al.* (2012) in terms of VO_{2max} , despite both studies involving healthy active men. The difference could be because of the different modes of exercise (running vs. cycling), or perhaps due to the longer duration of active recovery interval (4 x 30 sec cycling separated by 4 min rest compared to 4 x 15 sec running separated by 15 sec rest and 4 x 4 min separated by 3 min rest). Furthermore, the intensity of the recovery interval could also be a factor as subjects in Helgerud *et al.* (2007) exercised at 70% VO_{2max} , while subjects in Hazell *et al.* (2012) exercised at 0% resistance. The latter may be considered too low for such active young men to cause a significant change in VO_{2max} .

Moholdt *et al.* (2009) showed in their study that cardiovascular fitness of coronary artery bypass patients can be significantly improved through HIIT after surgery. These authors subjected patients to either aerobic interval training (4 x 4 min interval runs on the treadmill at 90% HR_{max} with an active recovery at 70% HR_{max}), or 46 min moderate continuous walking at 70% HR_{max} to ensure isoenergetic training protocols. Patients completed the training over four weeks for 5 days per week. The VO_{2max} of patients in the HIIT group increased significantly from baseline values (27.1 $ml.kg^{-1}.min^{-1}$ to 30.4 $ml.kg^{-1}.min^{-1}$), while resting HR decreased significantly (68.6 $b.min^{-1}$ to 66.4 $b.min^{-1}$), compared to those in the moderate continuous training group (pre: 26.2 $ml.kg^{-1}.min^{-1}$ vs. post: 28.5 $ml.kg^{-1}.min^{-1}$) and HR (pre: 102 $b.min^{-1}$ vs. post: 86 $b.min^{-1}$). A six months follow up revealed that the HIIT group still had significantly higher VO_{2max} values (four weeks: 30.4 $ml.kg^{-1}.min^{-1}$ vs. six months: 32.2 $ml.kg^{-1}.min^{-1}$), however, VO_{2max} values were lower in the moderate continuous training group (four weeks: 28.5 $ml.kg^{-1}.min^{-1}$ vs. 6 months: 29.5 $ml.kg^{-1}.min^{-1}$). The increase in VO_{2max} is an indication of improved cardiovascular fitness which will protect the patient against cardiovascular risk factors as VO_{2max} is one of the predictors of cardiovascular

diseases (Myers *et al.*, 2002). Moreover, aerobic interval training showed superior benefits by increasing VO_{2max} which was also longer lasting compared to moderate continuous training.

2.1. Positive and negative effects of running HIIT

The positive effect of this type of HIIT protocol is that it is tolerable by the patient population. It is also less demanding compared to the cycling protocol (Wingate type) where one has to cycle against a given resistance proportional to the body weight. It is important to note that this type of running protocol is still time efficient and induces the same physiological changes as that of the Wingate type, even though the duration of exercise per session is slightly longer.

C. PHYSIOLOGICAL ADAPTATIONS TO HIIT

There are enormously intricate factors that contribute to the training-induced improvements in exercise capacity. Various physiological (e.g. cardiovascular, muscle metabolic, respiratory, neural) and psychological (e.g. mood, motivation, perception of effort) factors operate in unison to constitute the adaptations that come with training. Physiological adaptations to exercise can be categorized into central and peripheral. This section attempts to discuss the general physiological adaptations that take place during HIIT.

1. Central adaptations to HIIT

The physiological changes that affect the cardiovascular system as a result of training are generally described as central adaptations. The central adaptations to training facilitate the

delivery of oxygen to the contracting muscle and are manifested by changes in cardiac output, heart rate, stroke volume and blood pressure (Foss and Keteyian, 1998).

1.1. Cardiac output

Cardiac output (CO) is the product of heart rate and stroke volume and adaptations to these variables are key to enhanced cardiovascular function. Increases in maximal CO facilitate proportional changes in maximal aerobic capacity (VO_{2max}). The change in CO is mainly determined by increases in stroke volume, as minimal changes in maximal HR is evident with training (McArdle *et al.*, 2010).

1.2. Stroke volume

In high intensity training, the improvement in oxygen delivery could be ascribed to increased SV. This may be the result of an increased left ventricular contractility and/or increased end-diastolic volume (Laursen and Jenkins, 2002). This is supported by the results of Helgerud *et al.* (2007) who found significant increases in SV following HIIT performed at four bouts of 15 sec exercise at 90 – 95% HR_{max} interspersed by 15 sec of active recovery at 70% HR_{max} and four bouts of 4 min exercise at 90 – 95% HR_{max} interspersed by 3 min of active recovery at 70% HR_{max} by health young males.

1.3. Heart rate

With exercise training, CO is mainly improved through the increase in SV, as submaximal and maximal HR usually decreases with training as a result of greater parasympathetic dominance. A decrease in maximal HR is mostly evident in highly trained persons, but short

term training in sedentary individuals also causes reduction in maximal HR. Moreover, highly trained individuals can maintain a higher workload and reach higher oxygen consumption before even reaching maximum HR compared to an untrained individual (McArdle *et al.*, 2010).

Trilk *et al.* (2011) showed that HIIT improves circulatory function in overweight/obese sedentary women who completed sprint interval training over four weeks (SIT: 4 – 7 x 30 sec cycle sprints at 5% body mass as resistance with 4 min active recovery). HR was significantly lowered (pre: 135 b.min⁻¹ and post: 124 b.min⁻¹), SV was significantly increased (pre: 86.9 mL.b⁻¹ and post: 96.6 mL.b⁻¹) and VO_{2max} was significant increased (pre: 21.6 mL.kg⁻¹.min⁻¹ and post: 24.5 mL.kg⁻¹.min⁻¹). These results indicate that high intensity training at low volume is effective in improving circulatory function and VO_{2max}, thus increasing functional capacity and decreasing risk for all-cause mortality in sedentary, overweight/obese women.

1.4. Blood pressure

The mean arterial blood pressure increases proportionally in relation to exercise intensity, generally due to a matching increase in systolic BP while diastolic BP stays at near resting levels (Rowell, 1993). In this manner, given that the mean arterial pressure is equal to cardiac output multiplied by total peripheral resistance, the observed increase in mean arterial pressure emanates from an increase in cardiac output that counterbalances a simultaneous decrease in total peripheral resistance. In essence, the increase in mean arterial pressure is deemed normal and desirable as it facilitates the result of the resetting of the arterial baroreflex to a higher pressure. In the absence of such resetting, the body would experience severe arterial hypotension during intense exercise training (Rowell, 1993).

Both resting systolic and diastolic BP has been shown to decrease after high intensity training. After two weeks of sprint interval training among sedentary overweight and obese adults, Whyte *et al.* (2010) demonstrated significant reductions in both resting SBP and DBP. Utilizing only two bouts of 30 sec all-out cycle sprinting interspersed by 4.5 min rest, Burns *et al.* (2012) showed similar and significant reductions in resting SBP and DBP in normal weight adolescents. Rossow *et al.* (2010) also compared the effect of an acute session of high intensity training and steady-state aerobic exercise and found both resting SBP and DBP to lower significantly to a similar extent as steady-state aerobic exercise in endurance trained men and women.

The acute and chronic effects of exercise and HIIT in particular, on PEH are discussed in chapter two.

2. Peripheral adaptations to HIIT

Peripheral adaptations to exercise training denote the enhanced ability of the contracting muscles to generate and utilize ATP. During high intensity exercise, the demand for energy is high and thus plenty of substrates are needed to synthesize ATP, while enzymes involved in the various metabolic pathways must be up regulated to increase the rate of ATP production. (Spriet, 1995). The peripheral adaptations refer to all the adaptations at cellular level within skeletal muscles, and most of these adaptations to training take place in the mitochondria.

For instance, it has been shown in a number of studies (Chan and Burns, 2013; Burns *et al.*, 2012; Little *et al.*, 2011; Psilander *et al.*, 2010; Whyte *et al.*, 2010; Burgomaster *et al.*, 2005) that the activity of the mitochondrial enzymes such as PGC-1 α , citrate synthase (CS), 3-hydroxyacyl CoA dehydrogenase (β -HAD), pyruvate dehydrogenase (PDH), and cytochrome oxidase (COX) are significantly enhanced with high intensity training. PGC-1 α is a

transcriptional coactivator that coordinates expression of both nuclear and mitochondrial genomes leading to mitochondrial biogenesis in muscle (recognized as the key regulator of mitochondrial biogenesis) (Hargreaves, 2006; Hood and Irrcher, 2006). CS plays a major role in fatty acid synthesis that takes place in the cytoplasm and acts as a carrier for acetyl-CoA. It is also a quantitative enzyme marker for the presence of intact mitochondria. β -HAD is also a mitochondrial enzyme marker that reflects maximal capacity of skeletal muscle for CHO and fat oxidation. Total protein content of PDH plays a major role in limiting the extent of lactate accumulation, while COX is one of the nuclear-coded polypeptide chains of cytochrome c oxidase that facilitates cellular energy transfer in the synthesis of energy in mitochondrial electron transport chain (Hargreaves, 2006; Hood and Irrcher, 2006).

2.1. Carbohydrate and fat oxidation

Training increases skeletal muscles' mitochondrial ability to oxidize CHO through 1) increases in the number, size and membrane surface area of the mitochondria and 2) increases in the concentration of oxidative enzymes involved in the Krebs cycle and the electron transport system (Foss and Keteyian, 1998). However, endurance training also enhances fat oxidation and in well trained individuals adaptations to fat utilization happen to such an extent that it results in the sparing of muscle glycogen stores, less lactic acid accumulation and less fatigue in the exercising muscles (Foss and Keteyian, 1998). More importantly, the increase in fat oxidative capacity following training is enhanced by four factors which include; 1) the increased blood flow to trained muscles, 2) increased fat-mobilizing and fat metabolizing enzymes, 3) enhanced mitochondrial respiratory capacity and 4) decreased catecholamine release for the same absolute power output (McArdle *et al.*, 2010).

Whyte *et al.* (2010) investigated the 24 hour post exercise substrate utilization following 15 min of six 30 sec “all-out” efforts of cycle sprinting interspersed by 4.5 min of active recovery for two weeks compared to pre training levels in sedentary overweight/obese men. They reported a significant reduction in the rate of CHO oxidation compared to baseline (pre: 0.08 g.min⁻¹ vs. post: 0.03 g.min⁻¹), while fat oxidation increased significantly compared to baseline (pre: 0.11 g.min⁻¹ vs. post: 0.13 g.min⁻¹). Similarly, Chan and Burns (2013) compared the substrate oxidation among healthy men, following 4 x 30 sec bouts cycle sprinting interspersed by 4.5 min active recovery to a control (resting) session. These authors observed a significantly higher rate of fat oxidation during the interval exercise which was 75% higher than the control session. At the same time, CHO oxidation rate decreased and was lower compared to the control session (pre: 0.17 g.min⁻¹ vs. post: 0.07 g.min⁻¹).

Likewise Talanian *et al.* (2010) demonstrated a ~20% decrease in whole body CHO oxidation, a total change of 22.7 g decrease of CHO content from pre training (109.7 g) and a concomitant increase of ~68% (10.4 g) in fat oxidation from pre training (15.2 g) in previously untrained healthy young women after 10 x 4 min cycling bouts at 90% VO_{2max} with 2 min of rest at 65% VO_{2max} for 3 days per week in six weeks. The changes in substrate utilization was accompanied by a significant increase in CS (pre: 18.4 mmol.min⁻¹.kg⁻¹ and post: 28.5 mmol.min⁻¹.kg⁻¹), β-HAD (pre: 18.5 mmol.min⁻¹.kg⁻¹ and post: 11.8 mmol.min⁻¹.kg⁻¹), COX4 (pre: 1.00 mmol.min⁻¹.kg⁻¹ and post: 1.57 mmol.min⁻¹.kg⁻¹), as well as significant increase in sarcolemmal GLUT4 content (pre: 1.0 g and post: 1.3 g in six weeks; normalized arbitrary units).

Similar results as above were reported for recreationally active women, following a two week HIIT intervention (4 – 6 x 4 min cycling at 90% VO_{2max} interspersed by 2 min of rest) (Talanian *et al.*, 2007). This training resulted in significant increases in VO_{2max} by 13% (pre:

36.3 ml.kg⁻¹.min⁻¹ and post: 40.9 ml.kg⁻¹.min⁻¹), whole body fat oxidation by 36% (pre: 15.0 ± 2.4 and 20.4 ± 2.5 g) during exercise, CS increased significantly (pre: 24.45 mmol.min⁻¹.kg⁻¹ and post: 29.31 mmol.min⁻¹ kg⁻¹) and β-HAD also increased significantly (pre: 15.44 mmol.min⁻¹ kg⁻¹ and post: 20.35 mmol.min⁻¹ kg⁻¹). All these findings suggest that HIIT improves whole body fat oxidation and the capacity for skeletal muscle to oxidize fat which is mediated by the increased activities of the mitochondrial enzymes.

Stephano *et al.* (2001) observed the same tendencies in highly trained cyclists following a single session of HIIT (8 x 5 min at 86% VO_{2max}, interspersed by 1 min recovery). The results showed a slightly decreased rate of CHO oxidation (346 μmol.kg⁻¹.min⁻¹ to 312 μmol.kg⁻¹.min⁻¹), from the first to the last bout of exercise but this was not statistically significant. The rate of fat oxidation increased significantly (16 μmol.kg⁻¹.min⁻¹ to 25 μmol.kg⁻¹.min⁻¹) measured throughout the HIIT session. The authors suggested that the decrease in CHO oxidation is mediated by the increased activity of β-HAD, while the increase in fat oxidation is mediated by the increased concentrations of CS enzymes in the skeletal muscles. Furthermore, changes in the maximal activities of “marker” enzymes such as CS also suggest that muscles’ oxidative capacity is greatly enhanced by HIIT.

Conversely, Burns *et al.* (2012) compared substrate metabolism in normal weight adolescents who performed 2 x 30 sec “all-out” cycle sprints with 4 min recovery over 15 min, and resting control session. A significant increase in CHO oxidation was reported ($p < 0.01$), but there was no change in fat oxidation ($p = 0.25$) over 90 min after exercise compared to the control session. This is despite the fact that Burns *et al.* (2012) used a similar HIIT protocol than Whyte *et al.* (2010) and Chan and Burns (2013). The difference in these findings could be explained by the difference in the body mass of the participants, or perhaps that the exercise was not enough to increase fat oxidation rates.

2.2. Insulin action

Skeletal muscle glucose uptake increases in response to the intensity of exercise. This is accomplished through increased blood flow and capillary recruitment to the active muscles, as well as increased glucose extraction by the sarcolemmal glucose transporters and activation of the glycolytic pathways responsible for glucose metabolism. Sarcolemmal glucose transport is enhanced by the translocation of the glucose transporter isoform 4 (GLUT4) from intracellular sites to the plasma membrane with exercise (Hargreaves, 2006). Insulin stimulates the rate of transport of glucose (i.e. the rate-limiting step for the uptake of glucose by skeletal muscles across cell membranes) into muscle cells and the most important glucose transporter for insulin action is GLUT4 (Hood and Irrcher, 2006).

Hood *et al.* (2011) examined the skeletal muscle oxidative capacity and insulin sensitivity effects following a HIIT protocol in middle-aged sedentary adults. Subjects performed six sessions of 10 x 1 min bouts of cycling (~95% HR_{max}) and 1 min recovery intervals, 3 days per week for two weeks. GLUT4 content in the skeletal muscle significantly increased by ~260% after the training sessions, compared to the pre training session, and protein content of CS and COX4 (also used as a marker of oxidative capacity) increased significantly by 31% and 39%, respectively, as well as PGC-1 α (56%) increase compared to pre-training values. In only two weeks of HIIT these authors showed that insulin sensitivity action was significantly improved, and markers of skeletal muscle mitochondrial content and glucose transport increased in spite of low total exercise volume with minimal time commitment.

Similarly, Burgomaster *et al.* (2007) reported significant increases in the concentration of GLUT4 by 20% and COX4 by 35% in skeletal muscle compared to baseline after 4 – 6 bouts of 30 sec “all-out” efforts and 4 min recovery (3 days per week for six weeks) by healthy men. Increased concentration of COX4 indicates high muscle oxidative capacity. The

increased activity of GLUT4 protein content could facilitate higher glucose uptake during recovery thereby improving insulin action.

3. Skeletal muscle adaptations

Muscles adapt to demands imposed on it in order to provide effective and efficient force for contraction. In response to the demand, it changes its ability to extract oxygen, choose energy sources, and rid itself of waste products. Skeletal muscles can change in terms of size and fiber type distribution, as well as storage capacity for fuel substrates. Thus this cause the muscle fiber type I (slow twitch fiber) and type II (fast twitch fiber) to change as a result of training (Foss and Keteyian, 1998).

Simoneau *et al.* (1985) compared the changes in muscle fiber type proportions and size following 15 weeks of high intensity intermittent training in sedentary individuals (10 - 15 x 15 - 30 sec intervals and long duration sessions of 4 - 5 x 60 - 90 sec bouts at 80 - 90 % $\text{VO}_{2\text{max}}$). Using a muscle biopsy of the vastus lateralis, the researchers found a significant increase in type I muscle fibers (pre: 41% vs. post: 47%), with a simultaneous increase in cross sectional area (size) (pre: $4089 \mu\text{m}^2$ vs. post: $4933 \mu\text{m}^2$) compared to baseline. There was a significant decrease in type IIb fibers compared to pre training (pre: 17 % vs. post: 11%) and a significant decrease in size (pre: $3593 \mu\text{m}^2$ vs. post: $2899 \mu\text{m}^2$). The proportion of the type IIa fibers remained unchanged (pre: 42% vs. post: 42%), even though the size increased slightly but not significantly (pre: $3794 \mu\text{m}^2$ vs. post: $4266 \mu\text{m}^2$). The results of Simoneau *et al.* (1985) therefore supports that HIIT may alter the proportion and size of type I and type IIb muscle fibers. Kohn *et al.* (2011), on the other hand, subjected eighteen endurance athletes to six weeks of HIIT sessions performed at 94% of individual's peak treadmill speed (PTS) twice a week and reported unchanged proportions of type IIa and slight

decreases in the cross-sectional area of the fibers (pre: 5532 μm^2 vs. post: 4603 μm^2). There were no changes in type I fibers. Although the reasons for the different findings between the two studies are not apparent, it may be related to the difference in training durations (six weeks vs. 15 weeks).

4. Body composition changes

The human body is made up of fat and fat free mass. Fat free mass primarily comprises muscles, bones, water and remainder elements. Excess fat mass is associated with cardiovascular health risk factors (Turocy *et al.*, 2011) and steady state, submaximal exercise has often been used to promote fat loss. For many reasons (discussed in chapter four) long term and permanent changes in body composition are not always achieved through traditional training programmes (Boutcher, 2011). The question therefore arises whether the employment of high intensity exercise would lead to better results.

Tjønnå *et al.* (2009) reported significant improvements in body composition in adolescents following 4 x 4 min aerobic interval training at 90 – 95% HR_{max} over three months compared to a multidisciplinary approach, MT (exercise, dietary and psychological advice, twice a month for 12 months). Individuals in the high intensity exercise group showed a significant reduction in total fat percentage of 1.3% and 2 % at the 12 month follow up. Lean body mass increased by 2% in three month and 4.1% at follow up. BMI also significantly reduced by 0.7 after training and 1.8 $\text{kg}\cdot\text{m}^{-2}$ at follow up. Body mass did not change, probably because lean body mass had increased. The MT group on the other hand showed no significant improvement in any of these variables of body composition after the 3 and 12 months intervention. It was encouraging to note that the individuals maintained the improvement in

their body composition parameters after 12 months, even though they only participated for three months in a training intervention.

Trapp *et al.* (2008) investigated the effects of a 15-week high intensity intermittent exercise (HIIE) program on subcutaneous and trunk fat and insulin resistance of young healthy women. Subjects were randomly assigned to HIIE group, steady-state exercise (SSE) group or control. The HIIE and SSE groups underwent a 15-week exercise intervention. Both groups showed significant improvements in cardiovascular fitness (increased VO_{2max}), however, only the HIIE group had a significant reduction in total body mass, fat mass and abdominal fat (9.5%) and fasting plasma insulin levels was significantly lower after HIIE compared to control. Fat loss through HIIE was attained with half the time commitment, but with similar energy expenditure to that of SSE. The subjects in HIIE lost 11.2% of total fat mass with SSE subjects experiencing no fat loss. Collectively, these results demonstrate that intermittent running compared to SEE is a more effective and efficient way of changing body composition. Thus, it is likely that HIIT would have an even greater fat-reducing effect on individuals possessing larger abdominal fat stores.

5. Performance adaptations

Performance variables like ventilatory threshold (T_{vent}), peak power output (PPO), and VO_{2max} have been associated with high intensity exercise training.

Laursen *et al.* (2002) using highly trained cyclists ($VO_{2max} = 68.7 \text{ ml.kg}^{-1}.\text{min}^{-1}$) showed adaptations in cardiorespiratory and performance variables after four sessions of HIIT (20 × 60 seconds at PPO 2 min recovery) completed in two weeks. The cyclists were able to perform a greater number of HIIT bouts and completed more work following training. The improved performance was accompanied by reductions in both the respiratory exchange ratio

and 1 min recovery HR from the first to the last HIIT session. T_{vent} and PPO obtained during the progressive exercise test also improved significantly as a result of only 4 bouts of HIIT sessions.

Esfarjani and Laursen (2007) compared the influence of HIIT on VO_{2max} and 3000 m running performance in moderately trained men. Depending on the participant's performance during an initial 3000 m running performance time, they were grouped into two HIIT groups, namely G1 and G2 and a control group, G_{con} . The HIIT groups completed two sessions twice per week and two 60 min recovery run sessions ($11.5 \text{ km}\cdot\text{h}^{-1}$; $75\% VO_{2max}$), while the control group completed four 60 min recovery run sessions ($75\% VO_{2max}$) each week at $11.5 \text{ km}\cdot\text{h}^{-1}$. During the HIIT sessions G1 performed 5 to 8 intervals at $V_{vo_{2max}}$ (minimal speed at which VO_{2max} occurred) for a duration equal to $60\% T_{max}$ with 1 min at $15.7 \text{ km}\cdot\text{h}^{-1}$, and a 1 min recovery run at $7.8 \text{ km}\cdot\text{h}^{-1}$ for 3.5 min. G2 performed 7 to 12 bouts of 30 sec at $19.9 \text{ km}\cdot\text{h}^{-1}$ ($130\% V_{vo_{2max}}$) interspersed by 4.5 min recovery at $7.8 \text{ km}\cdot\text{h}^{-1}$. It was shown that time trial performance improved significantly by 50 sec in G1 (-7.3%) and 23 sec in G2 (-3.4%) and a non-significant 1 sec in G_{con} (-0.1%). Also, VO_{2max} increased significantly by 9.1 and 6.4 $\text{ml}\cdot\text{kg}\cdot\text{min}^{-1}$ in G1 and G2, respectively but not G_{con} (+2.1 $\text{ml}\cdot\text{kg}\cdot\text{min}^{-1}$). Likewise T_{max} significantly increased in both HIIT groups (G1 = +35 sec and G1 = +32 sec), but not G_{con} (+3.5 sec). Speed at lactate threshold (V_{LT}) also increased significantly to $11.7 \text{ km}\cdot\text{h}^{-1}$ in G1 and $4.7 \text{ km}\cdot\text{h}^{-1}$ in G2, but not in control (+1.9 $\text{km}\cdot\text{h}^{-1}$). Running speed at VO_{2max} ($V_{vo_{2max}}$) significantly increased by $6.4 \text{ km}\cdot\text{h}^{-1}$ in G1 and $7.8 \text{ km}\cdot\text{h}^{-1}$ in G2, but not in control (+1.3 $\text{km}\cdot\text{h}^{-1}$). It was clear that improvements in performance were significantly greater for G1 than G2. It can be explained that the different intensity and volume of interval training in these two groups may be the reason for the difference in results. This suggests that HIIT actually improves performance even though the magnitude of performance may differ with the varying HIIT protocols in terms of intensity and volume of work load per interval.

D. CONCLUSION

From the literature it is clear that HIIT provoke adaptive responses which may be better than that of prolonged exercise training despite the minimum time spent in the execution of HIIT. Most studies deal with the effects of HIIT on health related outcomes (obesity, body composition, cardiovascular fitness and metabolism) and only a few addresses the PEH responses to HIIT. However, variations in the duration of the intervention, duration of exercise bouts (short vs. long), as well as different recovery periods in the reported studies may account for the varied results.

CHAPTER FOUR

OBESITY AND EXERCISE

A. INTRODUCTION

The human body has its own mechanism of regulating energy balance which is made up of three components; energy intake, energy expenditure and energy storage. However, the consequence of how these three components are controlled may result in obesity (Hill, 2006). Energy balance is ascertained by dividing the change in energy stores per day by the energy intake needed for maintaining weight and expressed as a percentage of baseline energy intake.

$$\text{Energy balance} = \left[\frac{\Delta \text{Energy store/day}}{\text{Energy intake for weight maintenance}} \right] \times 100$$

In the above equation, a positive number shows a positive energy balance or weight gain, while a negative number signifies a negative energy balance or weight loss (Redman *et al.*, 2007). Thus, a simplistic explanation of the current worldwide obesity epidemic is that most individuals are constantly in a state of positive energy balance, although it is acknowledged that many other factors can also contribute to overweight and obese states. Whether the positive energy balance is the result of overeating or very low levels of physical activity is a matter of debate. It is recognized that many factors, among other, biological, behavioral and environmental work together to affect energy balance and body weight (Hill, 2006). Thus a plethora of research focusses on a variety of approaches in which the issue of overweight and obesity can be addressed. A significant amount of research investigates the role of exercise in weight management and the prevention and treatment of overweightness and obesity.

B. THE TRADITIONAL VIEW OF THE EFFECTS OF EXERCISE ON WEIGHT LOSS

Concerning the effects of exercise on weight loss, it was traditionally believed that low intensity exercise is more beneficial for fat loss than high intensity exercise. It was thought that the amount of calories of fat burnt was indicative of the amount of fat loss, such that large amount of fat calories burnt at a lower intensity exercise meant large amounts of fat loss (McArdle *et al.*, 2010). However, it wasn't taken into consideration that the amount of total calories burnt during high intensity exercise means that the absolute number of fat calories burnt would be more, despite the fact that this type of exercise might burn a relatively lower percentage of fat calories in a set period. Moreover, high intensity exercise (especially when performed for longer durations) evokes excess post-exercise oxygen consumption (EPOC) more than with low intensity exercise, as an increase in calories consumed is related to greater EPOC at termination of exercise (McArdle *et al.*, 2010). Considering the effect of high intensity exercise in weight reduction as well as fat loss, it does not automatically take place during the exercise; rather it is the resting metabolic rate (RMR) which will increase caloric expenditure. High intensity exercise can cause an acute increase in RMR throughout the day for up to 24 hours following exercise (Treuth *et al.*, 1996; Bielinski *et al.*, 1985), as a result more calories will be expended through the day. Also, more fat is burnt after exercise to replace the glycogen stores depleted during the high intensity exercise. It has been shown that high intensity exercise promotes fat mass loss and preserves fat-free mass at a greater rate than low intensity exercise (Grediagin *et al.*, 1995; Racette, *et al.*, 1995; Tremblay *et al.*, 1994). This increase in fat-free mass serves to increase an individual's absolute RMR (Grediagin *et al.* 1995), which, in turn, causes an increase in total daily energy expenditure (TDEE) (Treuth *et al.*, 1996) and, therefore, increases overall caloric expenditure.

Moreover, there is the need for an effective weight loss program as researchers have conducted various studies to assess the effectiveness of a number of exercise program of varying intensity and duration on weight and fat loss. The following review will evaluate the effects of exercise and caloric restriction on weight loss and cardiovascular fitness following the traditional approach and high intensity exercise.

1. Traditional low to moderate intensity exercise and weight management

Keim *et al.* (1990) assessed the energy expenditure and physical performance in overweight women. The subjects were divided into an exercise and caloric restriction group, and an exercise only group. Both groups exercised six days per week at 65 - 80% VO_{2max} . Constant caloric expenditure was maintained by setting exercise energy expenditure at 15% of each individual's caloric intake necessary to maintain weight. As a result exercise duration differed among individuals. The results showed a statistically significant difference in weight loss between the diet and exercise group and the exercise group (13.08 kg vs. 5.61 kg). In the diet and exercise group, 33% of the weight loss was lean body mass, while in the exercise group 14% of the weight loss was lean body mass. This difference in lean body mass depicts the propensity of exercise in maintaining lean body mass. The intensity of exercise (65 - 80% VO_{2max}) had no significant effect on weight loss due to the constant caloric expenditure maintained for each individual although it promoted weight loss to a certain extent.

Kempen *et al.* (1995) also examined the energy balance in obese women during an eight week intervention. These researchers found statistically significant differences in substrate utilization for the diet group (D) compared to the diet/exercise group (DX). In the caloric restriction group, pre exercise carbohydrates and fat usage was 55% and 45%, respectively, while post exercise it was 60% carbohydrates and 40% fats. On the other hand, subjects in the

caloric restriction/moderate exercise group showed significantly different substrate utilization post exercise (55% carbohydrates and 45% fats), compared to pre exercise (70% carbohydrates and 30% fats). Therefore fat loss is enhanced due to the increase in fat substrate utilization which shows the tendency for exercise to increase the overall expenditure of fat as energy.

Racette *et al.* (1995) investigated the effect of exercise and dietary carbohydrate on energy expenditure in obese women. They divided subjects into four groups: low fat diet/exercise, low fat diet/no exercises, low carbohydrate diet/exercise, and low carbohydrate diet/no exercise. Low fat diet consisted of 18% fat and 59% carbohydrates and the low carbohydrate diet consisted of 49% fat and 27% carbohydrates. All the diets contained one-fourth of the components as protein. The exercise sessions consisted of 45 min at 60 - 65% VO_{2max} , 3 times per week for 12 weeks. The two groups who performed exercise as well showed a trend to lose more weight (-10.5 kg) than the two groups who did not exercise (-8.3 kg), however, the difference in weight loss was not statistically significant. More importantly, considering the fat loss on the other hand, the exercise groups had 89.4% of the weight loss made up of fat mass which was significantly more compared to 71.3% fat loss in the groups that did not exercise. The observed difference in fat loss indicates that exercise is important in a weight loss program in that its addition to diet promotes a higher percentage of fat-free mass.

1.1. Advantage and disadvantage of traditional approach to weight loss

Dietary changes and exercise in isolation or in combination have traditionally been used as a way to lose weight. Weight loss is accomplished by the reduction of total calories consumed including fat intake. However, exercise promotes weight loss by increasing caloric and fat expenditure (Keim *et al.*, 1990). This has been beneficial and less costly to some extent,

however, individuals' goals and expectations are usually not effectively met by the outcomes as these have mostly resulted in minimal or no fat loss (Shaw *et al.*, 2006). The traditional methods of weight loss take a long time to affect changes. Thus long-term devotion is required (Grediagin *et al.*, 1995) which most individuals fail to comply with as lack of time and interest emerges as a common barrier (Ball *et al.*, 2006).

2. High intensity exercise and weight management

A more effective exercise program that promotes fat loss and can be performed by overweight, inactive individuals is HIIT. A few studies have shown that HIIT improves fat oxidation, mitochondrial enzyme activity and energy expenditure in normal-weight and overweight adults (Burns *et al.*, 2012; Whyte *et al.*, 2010; Tjønnna *et al.*, 2009; Burgomaster *et al.*, 2008; Trapp *et al.*, 2008; Talanian *et al.*, 2007; Burgomaster *et al.*, 2005; Tremblay *et al.*, 1994).

Tremblay *et al.* (1994) investigated the impact of exercise intensity on body fatness in young normal weight adults. They compared 20 weeks of endurance training (ET) at moderate intensity cycling (started at 60% HR_{max} and progressed to 85% HR_{max} for 30 min and progressed to 45 min, four times a week and increased to five times a week) and 15 weeks of HIIT which was made up of short and long duration intervals. The short duration exercise consisted of 10 and later 15 sessions of 15 to 30 sec at 60% of maximal output per session (intensity was increased by 5% every three weeks). The long duration exercise consisted of 4 to 5 sessions of 60 to 90 sec at 70% of maximal work output per session. All training sessions were interspersed by recovery at a HR of 120 to 130 $b.m^{-1}$. The subjects were either assigned to ET or HIIT. The researchers found no significant change in body weight for either ET (pre: 60.6 kg; post: 60.1 kg) or HIIT (pre: 63.9 kg; post: 63.8 kg). The reason could be because the

subjects were not overweight or obese, as no large amount of weight could be lost to effect a significant change. However, a statistically significant difference in energy expenditure was found between ET (120.4 MJ) and HIIT (57.9 MJ). In spite of the fact that ET energy expenditure was twice greater than HIIT, there was a significant reduction in skinfold thickness (94.2 mm to 80.3 mm) after HIIT, but no significant reduction after ET (79.2 mm to 74.7 mm). Moreover, the change in skinfold thickness as per energy expenditure showed a significant reduction in HIIT (~0.25 mm/MJ) but not ET (~0.04 mm/MJ). The greater reduction in skinfold thickness in the HIIT group is an indication that higher intensity exercise is more effective in fat loss than lower or moderate intensity exercise of longer duration.

Whyte *et al.* (2010) studied sedentary overweight and obese adults, who performed two weeks of six sprint interval training sessions (4 – 6 bouts of 30 sec “all-out” efforts on the cycle ergometer interspersed by a fixed 4.5 min recovery between bouts). These researchers demonstrated that HIIT led to a significantly increased fat oxidation (pre: 0.11 g.min⁻¹ vs. post: 0.13 g.min⁻¹) and decreased carbohydrate oxidation (pre: 0.08 g.min⁻¹ vs. post: 0.03 g.min⁻¹), and decreased waist circumference (pre: 101.3 g.min⁻¹ vs. post: 98.9 g.min⁻¹) and hip circumference (pre: 110.9 g.min⁻¹ vs. post: 109.8 g.min⁻¹). Looking at the results, it can be suggested that high intensity training is effective in reducing fatness in overweight and obese individuals as a result of improved metabolism. Thus, indicating an enhanced metabolic health in this population.

Tjønnå *et al.* (2009) compared the effects of a multi-treatment approach (MTG) and aerobic interval training (AIT) on cardiovascular risk factors in overweight adolescents. The authors randomly divided the subjects into either AIT in which the subjects performed walking/running ‘uphill’ on the treadmill (4 x 4 min intervals at 90 - 95% HR_{max} and

separated by active rest interval of 3 min at 70%, twice a week for three months), or to MTG which consisted of exercise, dietary and psychological advice, twice a month for 12 months. Their results showed reduced BMI by 0.7 and 1.8 kgm⁻² respectively after three and 12 months follow up (33.2; 32.5; and 31.4 for baseline; three months; and 12 months, respectively) in AIT. However, subjects in the MTG group had no significant change in BMI (33.3; 33.1; and 32.9 for baseline; three months; and 12 months, respectively). Subjects in both groups increased in height over the 12 months period because they were still in a growing stage during the experimental period, but there was no significant change in body weight for both groups. However, subjects in the MTG group had a greater increase in weight than the AIT group (MTG vs. AIT: 94.3; 95.4; 96.1 vs. 94.1; 94.4; 94.4, respectively for baseline; three months; and 12 months). Reductions in percentage of body fat (%BF) of 1.3 and 2% at three months and 12 months, respectively, was observed following AIT, while there were no changes observed in the MTG group. Lean body mass increased by 2.0 kg and 1.8 kg in the AIT and MTG groups respectively, after three months. This explains the reason why there was no change in weight because the body fat mass was replaced with lean mass. It was concluded that AIT improved cardiovascular risk factors to a better extent than the normal multi-treatment approach to weight loss.

The traditional method of weight loss has usually been caloric restrictions and low to moderate exercise and has not been very effective. However, the recent adoption of HIIT in reducing weight has been shown to be effective as many benefits have been associated with HIIT in the improvement of health even though not much research has been done on the effect of HIIT on overweight/obese populations. It can be concluded that HIIT is tolerable by overweight/obese individuals and capable of reducing weight and other health-related risk factors detrimental to the individuals' health.

C. BARRIERS TO EXERCISE

Even though participation in physical activity is widely advocated for many reasons, the percentage of individuals' participation is low and has remained unchanged over a long time. The American Heart Association Expert Panel on cardiovascular diseases (CVD) prevention in women proposes that a physically active lifestyle is the best approach to the prevention of CVD, and that women should engage in at least 30 min of moderate intensity exercise such as brisk walking or jogging for most days of the week (Mosca *et al.*, 2007), yet the response to this recommendation is still low. From a psychological point of view, the social cognitive theory explains the five constructs that concurrently work to affect physical activity involvement (Bandura, 1997). These constructs include self-efficacy, self-regulatory behavior, perceived barriers, social support and outcome expectancies. Research proposes that the difference in the range of health behavior together with physical activity is most often the consequence of these constructs (Resnick *et al.*, 2000).

1. Self-efficacy

Among the five constructs, self-efficacy emerges as the main determinant of the social cognitive theory (Bandura, 1997) and appears to be one of the psychosocial factors that correlate strongest with physical activity and exercise (Rogers *et al.*, 2005; Netz and Raviv, 2004; Resnick *et al.*, 2000; Sherwood and Jeffery, 2000). Psychologically, self-efficacy is a general reflection of an individual's opinion concerning what he or she is capable of achieving with the resources available at different circumstances. However, it is not merely the resources available or the skills he or she possess, but what can be done with them (Bandura, 1997). Thus a skillful person who has all the necessary resources available can fail

to perform if he or she does not believe in his or her own capabilities (Bandura and Jourden, 1991).

Ayotte *et al.* (2010) found that self-efficacy was indirectly related to physical activity through outcome expectancies, perceived barriers and self-regulatory behavior. However, these authors indicated that individuals who had greater self-efficacy were optimistic about the outcome of exercise, had less perceived barriers, involved themselves with more self-regulatory behaviors and were more physically active than individuals who had lower self-efficacy.

2. Self-regulatory

Self-regulatory behaviors are also a relevant component of the social cognitive theory which cannot be ignored in relation to physical activity participation. Individuals' engagement in physical activity is compromised by self-regulatory behaviors in the sense that the individual has full control over his or her own behavior in spite of environmental influence. However, developing self-regulatory behaviors reflects an individual's ability to control him or herself in maintaining a behavior (Bandura, 2005). According to Maes and Karoly (2005) self-regulatory behavior comprise three main processes which include goal setting, enacting these goals and making plans to maintain the behavior. Thus the individual is able to strategize his/her behavior in relation to participation in physical activity through planning and setting of goals towards a successful outcome. Hence, these self-regulatory behaviors help the individuals to be guided towards their participation in physical activity (Anderson *et al.*, 2006; Bandura, 2004).

3. Outcome expectancies

This construct is mainly about the results that are expected after the execution of a particular behavior. According to Bandura (1997), individuals have a tendency of judging the expected results based on how well they believe they are capable of executing the task when the outcome mainly depends on performance. Thus the relation between self-efficacy and behavior tend to be partially mediated by outcome expectancies in such cases (Bandura, 1997).

4. Social support

Another important component of the social cognitive theory is the social support. This construct plays a major role by acting on physical activity via self-efficacy. For instance, when there is full social support, it tends to boost the individual's feeling of self-efficacy thus increasing levels of physical activity participation (Bandura, 1997). A typical example of the social support is the family which was evident in the work of Anderson *et al.* (2006). These authors investigated the influence of the components of the social cognitive determinants of physical activity among participants in a church based health promotion and found that the family was truly related to physical activity indirectly via self-efficacy and self-regulatory behaviors (Anderson *et al.*, 2006). Similarly, Ayotte *et al.* (2010) found an indirect relation between social support and physical activity through self-efficacy and self-regulatory behaviors. For instance, these authors found that increased social support was directly related to increased self-efficacy and increased self-regulatory behaviors. Moreover, these authors indicated that higher self-efficacy was directly related to more positive outcome expectancies and fewer perceived barriers. Additionally, Rovniak *et al.* (2002) also discovered an indirect relation between physical activity and social support via self-efficacy.

5. Perceived barriers

A perceived barrier is an indirect effect of self-efficacy on physical activity. This construct emerges in two forms which include the personal and environmental barriers to physical activity (Bandura, 1997). Personal barriers refer to self internally generated obstacles and for that matter the individual has direct control over such obstacles, for instance, tiredness or health problems (McSweeney and Coon, 2004). On the other hand, environmental barriers usually consist of those obstacles that cannot be controlled by the individual directly, for instance, changes in the weather (Salmon *et al.*, 2003). Ayotte *et al.* (2010) found an indirect association between physical activity and perceived barriers. According to these authors individuals who had a lot of perceived barriers had no plans or goals for participation in physical activity because they were not optimistic about the outcomes from participating in physical activity. Additionally, Ayotte *et al.* (2010) found that various health conditions related positively to perceived barriers and outcome expectancies. However, these authors suggested that, even though individuals who complain a lot about chronic health conditions and report several barriers that inhibit their participation in physical activity, seem to be acutely aware of the positive health effects of exercise.

Researchers have also identified some common barriers believed to mediate the achievement of the recommended physically active lifestyle and the consequent health benefits (Rye *et al.*, 2009). These include;

5.1. Lack of time

Lack of time is usually mentioned as the common barrier to being physically active. Individuals have various reasons for not having time to exercise (Ball *et al.*, 2006). For example, Ball *et al.* (2006) conducted a qualitative study on the influence of socio-economic

status of women on physical activity. These authors reported lack of time as the common barrier to physical activity due to work obligations and also the fact that it hooked up with certain priorities they held even when they got free time from work. Wright (1999) conducted a survey among West Virginians who were physically inactive and found that the main barrier to physical activity in women was lack of time. Moreover, Eyster (2003) studied the correlates of personal, social and environmental factors to physical activity in rural Midwestern white women and discovered insufficient time as the reason for not exercising. Participants felt that exercising is time consuming and that it compromises their available time with family. Chinn *et al.* (1999) also found that almost 50% of UK women considered lack of time the key barrier to regular exercise.

5.2. Lack of motivation

Lack of motivation is another identified barrier that tends to obstruct individual's engagement in exercise training (Ball *et al.*, 2006; Eyster, 2003; Brownson *et al.*, 2001). Wright (1999) also found lack of motivation (11.4%) as a barrier among women in West Virginia. Likewise, Chin *et al.* (1999) discovered lack of motivation (47.9%) to be a major barrier second to lack of time among UK women. Additionally, Rye *et al.* (2009) identified lack of motivation as the dominant barrier to exercise in the West Virginia same as Wright (1999). Motivation from family, friends and others as well as the individual's self-motivation is very important to ensure regular and life-long participation in physical activity (Canuto *et al.*, 2013).

5.3. Participant circumstances

Personal circumstances of individuals tend to influence their involvement in exercise training irrespective of the person's strong motivation to exercise. Personal health matters, competing

obligations, 'logistics' (e.g. access to training facilities) and financial cost, have been identified as possible restrictors to engagement in exercise (Canuto *et al.*, 2013). Among these personal circumstances, personal health matters appears as the strongest barrier (Canuto *et al.*, 2013), such that poor health condition will restrain the person from taking part in exercise training. However, for some individuals the state of their health can become a motivational benefit to engage in exercise because they are aware of the health benefits of exercise.

Competing obligations has also been identified as one of the many personal reasons influencing involvement exercise. These include family responsibilities, work, studies and other specific commitments depending on the individual (Canuto *et al.*, 2013).

5.4. Lack of support

Lack of support is another strong barrier that could affect the participation in physical activity (Rye *et al.*, 2009; Eyler, 2003). Canuto *et al.* (2013) found that support offered by family or friends modified competing priorities or other logistical matters at some point. For instance, considering a wife who always takes her child to school, if her husband decides to assist her as they take turns to taking the child to school, both will have the opportunity to engage in exercise.

5.5. Lack of facilities

In some cases lack of facilities (walking trails, gymnasias, swimming pools etc.) has been mentioned as a barrier to exercise training (Brownson *et al.*, 2001). Casper and Harrolle (2013) found that accessibility to facilities was a major constraint to participation in exercise,

while a lack of knowledge about the benefits of physical activity in the community can also be regarded as a barrier to exercise.

D. CONCLUSION

HIIT has been shown to be an effective, time efficient and enjoyable exercise protocol capable of promoting health benefits, even with little time commitment. The short time spent in the execution of this type of exercise coupled with numerous physiological benefits are advantageous over the traditional low to moderate intensity exercise which require plenty of time in performing. However, the individual participation in physical activity and exercise is affected by five constructs (self-efficacy, self-regulatory behavior, perceived barriers, social support and outcome expectancies constructs). Of these constructs perceived barriers emerges as a major problem where lack of time is the major obstacle to physical activity involvement. Thus the introduction of HIIT will assist people in overcoming the common barriers to exercise at only minimal time spent for effective results.

CHAPTER FIVE

PROBLEM STATEMENT

A. SUMMARY OF THE LITERATURE

PEH has been well and widely investigated among healthy and patient populations, using aerobic and resistance exercise, a combination of the two types of exercise (concurrent exercise) and water exercise. However, studies utilized exercise at low to moderate intensity which is described as a traditional method of training as recommended by the ACSM (Haskell *et al.*, 2007) to achieve health benefits. According to Jones *et al.* (2007), if exercise is to be accepted as an interventional approach in the management of blood pressure, knowledge is needed about the different characteristics of exercise considered necessary to stimulate PEH, particularly in terms of the intensity and duration of the exercise session. There is no apparent agreement on the precise magnitude of the reduction in blood pressure after exercise bouts or the precise duration of PEH. This disparity in results of studies could be attributed to differences in intensity and duration of the chosen exercise session. One of the questions is whether HIIT could be an alternative exercise modality with similar health benefits to the traditional endurance training.

Even though the ACSM asserts that 30 min of moderate intensity exercise on most days of the week helps to achieve health benefits (Haskell *et al.*, 2007), a lot of people are unable to achieve this despite the associated increased risk of cardiovascular diseases and metabolic-related disorders related to a sedentary lifestyle; due to lack of time which remains the most common barrier to regular exercise (Nybo *et al.*, 2010). Thus the need for a time efficient strategy is necessary to counteract this problem. In view of this, adoption of high intensity interval training (HIIT) has been shown to provoke similar benefits as the traditional method

of training and could be achieved with shorter-duration (≤ 20 min) exercise sessions (Burgomaster *et al.*, 2008) and produce even superior benefits than moderate intensity exercise (Rognmo *et al.*, 2004). It has also been shown that higher intensity exercises have: 1) greater positive effects on body composition than energy-matched lower-intensity exercise (Irving *et al.*, 2008); 2) an improvement in metabolic risk factors (Whyte *et al.*, 2010; Burgomaster *et al.*, 2005; Burgomaster *et al.*, 2007); 3) an improvement in cardiovascular function (Trilk *et al.*, 2010) and 4) a reduction in blood pressure (Eicher *et al.* 2010; Nybo *et al.*, 2010) with just a minimum time spent exercising.

The main cause of morbidity and mortality in developing countries all over the world is the fast growing nature of cardiovascular diseases, with hypertension being one of the main risk factors for congestive heart failure, stroke, peripheral vascular diseases, myocardial infarction and end-stage renal disease. Hypertension still remains insufficiently treated and leads appreciably to the financial and epidemiological burden of cardiovascular diseases despite the accessibility of the cost-effective and non-pharmacological treatment approach of which South Africa is not an exception (Steyn *et al.*, 2001).

Obesity has also become known as a challenging and prevalent condition which is considered an independent risk factor for atherosclerotic cardiovascular diseases. Atherosclerosis is a progressive inflammatory disease characterized by the buildup of lipids and fibrous elements in medium to large arteries and is a symptom of endothelial dysfunction. Overweight/obese individuals show signs of an increase in systemic inflammation and are more likely to develop risk factors and comorbidities of cardiovascular diseases. There also appears to be a positive relationship between being overweight and all-cause mortality in men; however, active overweight individuals may have a lower risk factor for heart diseases than their inactive counterparts (Harris *et al.*, 2008). Victims of these diseases frequently turn out to be

inactive and/or deconditioned. This may result in decreased muscle strength, high percentage of body fat and reduced exercise capacity. Furthermore, the quality of life is reduced and this further promotes cardiovascular risk factors (Painter, 2008).

Regular steady state exercise at moderate intensities such as walking and jogging have most often been utilized as exercise protocol to induce fat loss. Nonetheless, people do not normally meet the expected outcome because of failure to adhere to these types of training protocols which take a long time to show effective changes. Thus exercise protocols that more efficiently decrease body fat and can be accomplished by sedentary overweight, individuals are required (Boutcher, 2011). A growing number of evidence suggests that HIIT has the potential of being an efficient exercise protocol for lessening fat in overweight/obese individuals. HIIT can address obesity (already shown to be effective), which will impact risks for cardiovascular diseases. Possibly PEH would be an added advantage. Since time is the major barrier for the participation in regular physical activity, shorter duration exercise as well as the manifestation of significant results in a few weeks will be a motivating factor. Furthermore, very few studies have been done on women.

B. RESEARCH LIMITATION

Very little research has focused on the effect of HIIT on PEH in overweight/obese women. Researchers have focused more on the peripheral and metabolic response to HIIT than cardiovascular responses especially with little attention to the effect of HIIT on PEH.

C. AIM OF THE STUDY

The primary aim of the study was to investigate the training and detraining effects of a short HIIT intervention on post-exercise hypotension in young overweight/obese women.

Specific aims:

1. To determine the magnitude of PEH after an acute bout of HIIT.
2. To determine the magnitude of PEH after six sessions of HIIT.
3. To determine whether two weeks of detraining change the effect of HIIT on PEH.
4. To determine the relationship between changes in SBP and DBP after the HIIT intervention and anthropometric variables, maximal exercise capacity and resting BP.

CHAPTER SIX

METHODOLOGY

A. RESEARCH DESIGN

This study followed an experimental design, with pre-training and two post-training testing sessions. All participants completed a high-intensity interval training (HIIT) program and participants served as their own control (CON). Outcome variables were measured before and after the intervention period, as well as after a two week detraining period.

B. PARTICIPANTS

Twenty young overweight/obese women between the ages of 18 and 25 years were asked to volunteer for this research. Participants were recruited through advertisements on campus and the distribution of flyers, as well as personal communication with students; thus a sample of convenience was used. Participants were screened to identify those who met the inclusion criteria. The physical activity readiness questionnaire (PAR-Q) (Appendix A) was employed to assess the participant's ability to exercise and to ensure that participants had no health conditions that might be aggravated by exercise.

1. Inclusion Criteria

Participants were included if:

- they were between 18 and 25 years old;
- they had a BMI of $>25\text{kg}\cdot\text{m}^{-2}$;

- they did not engage in structured exercise more than one day per week;
- they had resting systolic blood pressure (SBP) <130 mmHg and diastolic blood pressure (DBP) <90mmHg.

2. Exclusion Criteria

Participants were excluded if they were clinically diagnosed with:

- type 1 or 2 diabetes;
- stage 1, 2, or 3 hypertension (that is SBP >130 and DBP >90 mmHg, respectively);
- any cardio-respiratory diseases;
- any musculo-skeletal abnormalities or injuries;
- any medical condition for which they used chronic medication (i.e. antidepressant, antianxiety, thyroid or hypertension medication).

3. Assumptions

It was assumed that participants truthfully provided answers to the questions when completing the PAR-Q, as well as their consumption of caffeine, tobacco and medications. It was also assumed that participants adhered to the pre-exercise testing regulations.

4. Limitations

Day-to-day variations in participants' activity and stress levels may affect blood pressure measurements. Activities of daily living, as well as other stressful events could not be

controlled. Participants therefore acted as their own control in order to reduce these limitations.

5. Delimitations

The study was limited to young overweight/obese women between the ages of 18 to 25 years. Volunteers were used which might not be a true representation of this population.

C. EXPERIMENTAL DESIGN

1. Laboratory visits

Participants were required to visit the laboratory for a number of testing and training sessions over a six week period. The following marks the schedule of participants' visits to the laboratory.

Visit 1: Screening and inclusion

The participants were informed on the general overview and the aims of the project, as well as the possible risks and stresses associated to the experimental procedures. They underwent a screening process to determine their possible inclusion in the study. The screening test included anthropometric measurements (height, body mass and bio-electrical impedance analysis), resting blood pressure, physical activity readiness (PAR-Q) and general health questionnaire (Appendix B). Participants were asked to volunteer for the study if they met the inclusion criteria and were then requested to sign a consent form (Appendix C).

Visit 2: Baseline measurements

Baseline measurements were taken which included resting blood pressure, body composition measures, resting heart rate (HR) and VO_{2max} . All participants completed a maximal exercise capacity test to exhaustion on the treadmill, which was used to determine each individual's running intensity during the HIIT protocol. SBP and DBP, as well as HR were continuously monitored with an ambulatory blood pressure monitor every 10 min for an hour post exercise and in a seated position after the maximal exercise capacity test.

Visit 3 – 8: Training sessions

Participants were asked to visit the laboratory three times per week, with at least 24 hours rest in between for a two week period (total six sessions). All sessions were done on the treadmill. SBP and DBP, as well as HR were continuously monitored with an ambulatory blood pressure monitor after the first and the last training sessions. The first reading was taken 10 min post-exercise, followed by readings every 10 min for an hour post exercise in a seated position.

Visit 9: Post testing

Post-training measurements were recorded after the sixth HIIT session after at most 48 hours of rest period from the last training sessions. This included resting BP, resting HR, VO_{2max} , and body composition. The post-training measurements were the same as the measurements taken during baseline.

Visit 10: Detraining and retention test

Participants were asked to stop the HIIT training and not engage in any form of structured exercise for two weeks and then return to the laboratory for the same measurements as in visit 9 during the post testing.

2. Place of study

All testing and exercise sessions were conducted in the Sport Physiology Laboratory in the Department of Sport Science at Stellenbosch University. This laboratory is completely furnished with all the apparatus that was needed for this research. All tests and measurements including physical and physiological, were done at temperatures between 17 and 25°C and at a humidity of 47.5%.

3. Ethical aspects

The study protocol was approved by the Ethics Committee of Research Subcommittee A at Stellenbosch University (DESC_Terblanche2012). The research used no invasive procedures and subjects were informed that their participation was completely voluntary. For this reason they could withdraw from the project at any point in time. An informed consent form was completed by subjects and all procedures and tests were also clearly explained. There were no serious risks involved in the study; nonetheless participants may have experienced muscle pain and discomfort during or after the exercise sessions. Participants might also have been uncomfortable with the high intensity running on the treadmill and could stop if they felt they could not continue the activity. During the running sessions on the treadmill, subjects were fitted to a safety harness to prevent serious injury in the event of a fall.

D. PROCEDURE OF MEASUREMENTS AND TESTS

All participants completed an anthropometric and body composition screening, resting blood pressure and heart rate, as well as maximal incremental exercise test to exhaustion before participating in the exercise sessions.

1. Anthropometric measurements

Anthropometric measurements included body mass, height, and bio-electrical impedance analysis (BIA) to assess percentage of body fat was performed.

Body mass

This was verified with a standardized electronic scale (UWE BW- 150, 1997 model, Brisbane Australia) and recorded to the nearest 0.1 kg. Participants were barefoot and clothed in light-weight clothing. They were asked to stand on the center of the scale, so that their weight was evenly distributed on both legs.

Height

Standing height was measured using a standing stadiometer (Seca, Germany). Measurements were recorded in centimeters to one decimal place (0.1 cm). Participants were barefoot and stood with heels together and upper back, buttocks and heels against the stadiometer with the head placed in the Frankfurt plane. The Frankfurt plane is achieved by positioning the lower edge of the eye socket (Orbitale) in the same horizontal plane as the notch just above the tragus of the ear (Tragion). The measurement was recorded from the interior side of the feet to

the vertex of the skull (the highest point of the skull). The height and body mass was then used to calculate the body mass index ($\text{BMI} = \text{kg}\cdot\text{m}^{-2}$).

Bioelectrical impedance analysis (BIA)

The participant's lean and fat mass was recorded with a Bodystat unit (Quadscan 400, Isle of Man United Kingdom). The BIA operates by sending a low electrical current through the body ($80\mu\text{A}$ at 50 kHz) which measures the resistance and reactance of the body tissue due the variation in resistance of lean and adipose tissue. Participants were asked to empty their bladders and abstain from any strenuous exercise, drinks that may contain caffeine and smoking for at least four hours prior to the test. The subject lay quietly in a supine position with arms away from the center of the body and legs wide apart in order not touch each other. Two electrodes were placed on the dorsal part 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 other electrodes were placed on the dorsal part of the bare right foot between the lateral medial malleoli, as well as at the base of the toes between the hallux and the third phalange, after cleaning with an alcohol swab. The Bodystat unit cables were then connected to the electrodes, measurement of resistance and reactance values was recorded when the analyzer was switched on.

2. Blood pressure and heart rate reading

Resting blood pressure and heart rate were recorded after a five minute rest period before exercise. The average of three readings was recorded with an automated ambulatory air bladder containing cuff (Ergoline Ergoscan 2008, Germany). The same automated blood pressure monitor was used to take all measurements of blood pressure and heart rate in all

subjects and throughout the study. This monitor uses an oscillometric method to measure blood pressure. The duration of measurement was 30 – 40 seconds with a dilation rate between 3 – 5 mmHg per second. Participants were seated upright in a back supported chair with the left arm at the level of the heart and resting on the armrest of the chair, after which anything that could cause obstruction or occlusion of the circulation above the cuff site was taken away. The cuff was placed on the left arm, 2.5 cm above the antecubital space, with the palm facing up and the air bladder directly over the brachial artery. Participants remained quiet without talking or any form of body movement while measurement was taken when the device was switched on. If the two readings for either systolic or diastolic blood pressure differed more than 5 mmHg, a third reading was taken and the average was calculated. If the difference between the first two readings was not greater than 5 mmHg, the average of these readings was taken as the final measurement. The measurements were taken two minute apart.

3. Maximal exercise capacity test

Participants' maximal aerobic capacity (VO_{2max}) was assessed by means of an incremental exercise protocol to exhaustion performed on the h/p/cosmos Saturn treadmill (Nussdorf-Traunstein, Germany), and with the use of a COSMED® Quark CPET (Rome, Italy) metabolic system for constant monitoring of metabolic variables. The gas analyzers were calibrated prior to each test with atmospheric gas and known gas concentrations of 16% O_2 , 5% CO_2 , balance N_2 and the turbine flow meter was calibrated with a 3L calibration syringe. Blood samples were taken via finger prick to measure the resting blood lactate concentrations with the use of a Lactate Pro meter. The participant's finger was cleaned with an alcohol swab and then pricked with an Accucheck Soft Clix (Roche diagnostics, Mannheim,

Germany). Blood was then drawn into the capillary tube of the Lactate Pro lactate analyser (ARKRAY, Inc., Kyoto, Japan). The reading was then recorded at the end of the countdown. A blood sample was taken at the end of each completed work load till exhaustion.

The participants were fitted with an adjustable body safety harness on the treadmill, as well as a COSMED® CPET heart rate monitor. The test began with two minutes warm up at 5 km.h⁻¹ with a zero incline. Participants were allowed to drink water after the warm up if required. The protocol in Table 6.1 was employed to assess the maximal effort. The test was stopped if any of test termination criteria as outlined in the ACSM's guidelines for exercise testing and prescription were visible (ACSM, 2006). These criteria include:

1. onset of angina or angina-like symptoms;
2. significant drop (20 mm Hg) in systolic blood pressure or a failure of the systolic blood pressure to rise with an increase in exercise intensity;
3. excessive rise in blood pressure: systolic pressure >260 mm Hg or diastolic pressure > 115 mm Hg;
4. signs of poor perfusion: light-headedness, confusion, ataxia, pallor, cyanosis, nausea, or cold and clammy skin;
5. failure of heart rate to increase with increased exercise intensity;
6. noticeable change in heart rhythm;
7. subject requests to stop;
8. physical or verbal manifestations of severe fatigue;
9. failure of the testing equipment.

On the other hand the test was stopped when participants reached exhaustion which was verified when accomplishing two of the following criteria:

1. VO_2 does not increase by more than 150 ml per successive workload;
2. a respiratory quotient (R) value equal or above 1.15 is reached;
3. heart rate (HR) is more than 90% of the age-predicted maximal heart rate;
4. the rating of perceived exertion (RPE) is above 19 on the 6 – 20 Borg scale;
5. the subject indicates that she is exhausted.

The results of the individuals' $\text{VO}_{2\text{max}}$ tests were then used to determine the exercise intensity for the high intensity training sessions.

Table 6.1: The protocol for $\text{VO}_{2\text{max}}$ assessment

Stage	Time (min)	Speed (km.h⁻¹)	Incline %
1	3	6	0
2	6	7	0
3	9	8	0
4	12	9	0
5	14	10	1
6	16	11	1
7	18	12	1
8	19	13	2
9	20	14	2
10	21	15	2
11	22	16	3

4. Exercise training session

The training program consisted of six sessions of one minute running on the h/p/cosmos Saturn treadmill, separated by one minute active recovery. The training session started with a 5 minute warm-up at a velocity corresponding to 50% maximum heart rate (HR_{max}), followed by 10 x 1 minutes bouts at a running velocity corresponding to 90 - 95% HR_{max} . The high intensity interval was separated by a minute active recovery at a running velocity of 70% HR_{max} . This was repeated in the first three training session (first week of training sessions). The last three training sessions (second week of training session) comprised 15 x 1 minute bouts at 90 - 95% HR_{max} separated by a one minute rest. This was followed by another 5 minutes of cool-down at a running velocity corresponding to 50% HR_{max} at the end of every training session. In the first week, the exercise protocol consisted of 10 minutes of high-intensity exercise and 10 minutes of active recovery, giving a total of 20 minutes. In the second week, the protocol comprised 15 minutes HIIT and 15 minutes active recovery. When including warm-up and cool-down, the total duration of the exercise protocol was 30 minutes for the first week and 40 minutes for the second week.

5. Post-exercise monitoring

Measurements similar to baseline which included blood pressure and heart rate, VO_{2max} , body composition, body mass and height were repeated at least 24 hours after the last HIIT session and before the two weeks of detraining. The blood pressure and heart rate were measured after 10 minutes after the exercise test and then continuously at 10 minutes intervals over an hour recovery period with an ambulatory blood pressure monitor (Ergoline, Ergoscan 2008, Germany). All these measurements followed the VO_{2max} tests and the first and the last HIIT sessions of the training intervention.

6. Detraining and retention test

Participants were asked to discontinue the HIIT training, as well as refrain from any structured exercise for two weeks and then return to the laboratory for measurements similar to baseline measurements.

E. STATISTICAL ANALYSIS

The statistical analysis was performed with Microsoft Office Excel (2010) and STATISTICA 11 (Statsoft, Inc. 2012, USA). Descriptive statistics were analyzed as means and standard deviations (SD). The differences in means of PEH following the maximal exercise capacity tests was compared by single factor analysis of variance (ANOVA), whereas differences in the means of PEH following the HIIT sessions were compared by the paired t-test. The test and time effects were independent factors with the interaction effect (tests*time) as the dependent factor. Fischer's Least Significant Difference (LSD) Post Hoc tests were performed to identify significant differences among variables. The level of significance was set at $p < 0.05$ for all analyses. The magnitude of PEH was accepted as clinically significant at ≥ 3 mmHg (Touyz *et al.*, 2004).

Cohen's effect sizes were used to determine practically significant differences between PEH responses following maximal exercise capacity tests and HIIT sessions. Cohen's effect sizes were determined according to the following criteria: trivial practical effect, < 0.20 ; small practical effect, ≥ 0.20 ; moderate practical effect, ≥ 0.50 ; large practical effect, ≥ 0.80 ; and very large practical effect, ≥ 1.20 (Hopkins *et al.*, 2009). Correlations between outcome variables (resting BP, VO_{2max} , BMI and %BF) and BP variables were determined with Pearson correlation coefficients. The strength of these correlations were determined as follows: $r = 0$, no correlation; $0.00 \leq r \leq 0.24$, weak correlation; $0.25 \leq r \leq 0.49$, moderate

correlation; $0.50 \leq r \leq 0.74$, moderate to good correlation; and $0.75 \leq r \leq 1.00$, strong correlation (Rosner, 1986).

CHAPTER SEVEN

RESULTS

A. DESCRIPTIVE CHARACTERISTICS

1. Participants

Twenty young women between the ages of 18 and 25 years were included in the study. These participants were either overweight or obese (BMI: 25 – 30 or $>30\text{kg}\cdot\text{m}^{-2}$, respectively), did not engage in structured exercise more than one day per week and had a resting SBP <130 mmHg and DBP <90 mmHg. The physical and physiological characteristics of the participants are summarized in Table 7.1. Over the testing period of all participants, the temperature in the laboratory ranged between 17°C and 25°C and the humidity was on average 47.5%.

Table 7.1: Physical and physiological characteristics of participants

Variables	Mean \pm SD	Range
Age [years]	21.2 \pm 1.93	19.0 - 25.0
Height [cm]	160.0 \pm 6.70	145.8 - 172.7
Weight [kg]	74.3 \pm 10.0	61.0 - 103.1
BMI [$\text{kg}\cdot\text{m}^{-2}$]	29.0 \pm 3.10	25.1 - 37.3
Body fat [%]	35.5 \pm 5.00	25.9 - 45.5
Resting SBP [mmHg]	119.2 \pm 5.60	108.0 - 129.0
Resting DBP [mmHg]	78.8 \pm 4.12	71.0 - 89.0
Resting MAP [mmHg]	91.7 \pm 4.31	84.9 - 102.5
Resting HR [$\text{b}\cdot\text{min}^{-1}$]	72.7 \pm 5.57	65.0 - 83.0
$\text{VO}_{2\text{max}}$ [$\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$]	27.8 \pm 5.69	19.3 - 37.0

cm, centimeter; kg, kilogram; BMI, body mass index; $\text{kg}\cdot\text{m}^{-2}$, kilogram per square meter; %, percentage; mmHg, millimeters mercury; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate; $\text{b}\cdot\text{min}^{-1}$, beats per minute; $\text{VO}_{2\text{max}}$, maximum aerobic capacity; $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, milliliters per kilogram per minute.

1.1. Resting BP and HR

Table 7.2a illustrates the resting BP and HR before and after the training intervention together with the responses after detraining. The practical significance of the training and detraining effects on resting BP and HR is shown in Table 7.2b.

Resting SBP, DBP and MAP after the training intervention were statistically significantly lower ($p = 0.00$) than at baseline. The mean SBP was significantly reduced by 3.40 mmHg which is equal to 2.94% ($p = 0.00$; ES = 0.66) following training. However, detraining returned resting SBP ($p = 0.58$; ES = 0.08) to near baseline values. Resting DBP and MAP, on the other hand, were significantly different among pre-, post- and detraining ($p = 0.00$). The mean DBP was significantly reduced by 4.50 mmHg, equivalent to 6.11% ($p = 0.00$) and 2.40 mmHg (3.17%; $p = 0.01$) respectively, following post HIIT and detraining. The mean MAP was also significantly lowered by 4.14 mmHg equal to 4.72% ($p = 0.00$) and 14.50 mmHg (18.78%; $p = 0.00$) respectively following post HIIT and detraining. The effect of HIIT on resting HR was not statically significant, ($p = 0.15$; ES = 0.50) and was statically significantly increased after detraining ($p = 0.02$; ES = 0.83).

Table 7.2a: Resting BP and HR characteristics of participants

Variables	Pre			Post			Detraining		
	Mean	±	SD	Mean	±	SD	Mean	±	SD
Resting SBP [mmHg]	119.2	±	5.60	115.8	±	4.68*	118.7	±	6.64 [§]
Resting DBP [mmHg]	78.2	±	4.12	73.7	±	4.89*	75.8	±	5.98 ^{#§}
Resting MAP [mmHg]	91.7	±	4.31	87.6	±	4.52*	77.2	±	5.94 ^{#§}
Resting HR [b.min ⁻¹]	72.7	±	5.57	69.7	±	7.57	75.7	±	7.83 [§]

Pre, pre-training; Post, post-training; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; mmHg, millimeter mercury; HR, heart rate; b.min⁻¹, beats per minute; * statistically significant difference between pre- and post-training, $p < 0.05$; [§] statistically significant difference between post-training and detraining, $p < 0.05$; [#] statistically significant difference between pre-training and detraining, $p < 0.05$.

Table 7.2b: Effect sizes of Resting BP and HR characteristics of participants

Variables	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
Resting SBP [mmHg]	0.66 ^m	0.50 ^e	0.08 [†]
Resting DBP [mmHg]	1.00 ^m	0.38 ^e	0.47 ^e
Resting MAP [mmHg]	0.94 ^m	1.96 ^t	2.79 [‡]
Resting HR [b.min ⁻¹]	0.50 ^e	0.83 ^m	0.45 ^e

Pre, pre-training; Post, post-training; ES, effect size; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; mmHg, millimeter mercury; HR, heart rate; b.min⁻¹, beats per minute; SBP, systolic blood pressure; [†] trivial practical significant effect; ^e small practical significant effect; ^m moderate practical significant effect; ^t large practical significant effect, [‡] very large practical significant effect

2. The effect of HIIT on maximal exercise capacity

Twenty participants volunteered to partake in the study. Of the twenty individuals who volunteered, two could not complete the final maximal exercise capacity test after detraining due to travel reasons. In all a total of 20 individuals completed baseline assessments, 20 individuals completed post training and 18 individuals finally completed a detraining maximal exercise capacity test. According to the norms of Hoffman (2006), the aerobic fitness levels of the participants can be categorized as fair (VO_{2max} : 25 - 33 ml.kg⁻¹.min⁻¹).

Table 7.3a compares the results of the maximal exercise capacity tests during baseline (pre training), post training and detraining assessments. HIIT improved the participants' maximal exercise response as there was a significant increase in maximal aerobic capacity. Relative VO_{2max} increased by 6.7% ($p < 0.05$; ES = 0.28) but detraining statistically significantly decreased VO_{2max} by 5.7% ($p < 0.05$; ES = 0.35). Absolute VO_{2max} [L.min⁻¹] slightly increased by 4.5% ($p = 0.11$; ES = 0.21) but detraining statistically significantly decreased absolute VO_{2max} by 3.3% ($p < 0.05$; ES = 0.43). VE_{max} ($p < 0.05$; ES = 0.49) and La_{max} ($p < 0.05$; ES = 0.52) were also statistically significantly increased after HIIT but returned to near pre-training values following detraining. Even though there were no statistically significant effects of training on HR_{max} , RER_{max} , and RPE, Table 7.3b shows that training caused small

practically significant effects. However, detraining returned these responses to near pre training values.

Table 7.3a: The effect of training and detraining on maximal exercise capacity of participants

Variables	Pre			Post			Detraining		
	Mean	±	SD	Mean	±	SD	Mean	±	SD
VO _{2max} [L.min ⁻¹]	2.0	±	0.38	2.1	±	0.32	2.0	±	0.25 [§]
VO _{2max} [ml.kg ⁻¹ .min ⁻¹]	27.8	±	5.69	29.3	±	5.06*	27.6	±	4.33 [§]
VE _{max} [L.min ⁻¹]	86.0	±	13.85	93.1	±	15.15*	89.1	±	14.10
HR _{max} [b.min ⁻¹]	194.4	±	7.37	191.7	±	7.97	192.9	±	9.45
La _{max} [mmol.L ⁻¹]	10.1	±	1.88	11.2	±	2.11*	10.2	±	1.76
RER _{max}	1.2	±	0.07	1.1	±	0.07	1.1	±	0.05
RPE	18.9	±	1.29	19.4	±	0.94	18.9	±	1.26

Pre, pre-training; Post, post-training; VO_{2max}, maximum aerobic capacity; L.min⁻¹, liters per minute; ml.kg⁻¹.min⁻¹, milliliters per kilogram per minute; VE_{max}, maximum minute ventilation; HR_{max}, maximum heart rate; b.min⁻¹, beats per minute; La_{max}, maximum lactate; mmol.L⁻¹, millimol per liter; RER_{max}, maximum respiratory exchange ratio; RPE; rating of perceived exertion; *statistically significant difference between pre- and post-training, $p < 0.05$; [§] statistically significant difference between post-training and detraining, $p < 0.05$.

Table 7.3b: Effect sizes of the changes in maximal exercise capacity of participants during training and detraining

Variables	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
VO _{2max} [L.min ⁻¹]	0.21 ^²	0.43 ^²	0.16 [†]
VO _{2max} [ml.kg ⁻¹ .min ⁻¹]	0.28 ^²	0.35 ^²	0.03 [†]
VE _{max} [L.min ⁻¹]	0.49 ^²	0.27 ^²	0.22 ^²
HR _{max} [b.min ⁻¹]	0.34 ^²	0.14 [†]	0.17 [†]
La _{max} [mmol.L ⁻¹]	0.52 ^²	0.47 ^²	0.06 [†]
RER _{max}	0.51 ^²	0.18 [†]	0.39 ^²
RPE	0.44 ^²	0.42 ^²	0.03 [†]

Pre, pre-training; Post, post-training; ES, effect size; VO₂, aerobic capacity; L.min⁻¹, liters per minute; VO_{2max}, maximum aerobic capacity; ml.kg⁻¹.min⁻¹, milliliter per kilogram per minute; VE_{max}, maximum minute ventilation; HR_{max}, maximum heart rate; b.min⁻¹, beats per minute; La_{max}, maximum lactate; mmol.L⁻¹, millimol per litre; RER_{max}, maximum respiratory exchange ratio; RPE; rating of perceived exertion; [†] trivial practical significant effect; ^² small practical significant effect; ^³ moderate practical significant effect.

3. HIIT and detraining effects on BP and HR after maximal capacity tests

After each of the three maximal exercise capacity tests ambulatory SBP, DBP, MAP and HR were measured over 60 minutes.

3.1. Systolic blood pressure

Figure 7.1a illustrates the absolute change in SBP over 60 min after the maximal exercise capacity tests. There were no statistically significant differences in mean SBP during the pre-, post- and detraining tests (pre: 118.6 ± 9.7 mmHg; post: 118.8 ± 11.4 mmHg; detraining: 122.0 ± 11.4 mmHg; $p = 0.25$) even though the detraining values were consistently higher compared to pre- and post-training values. On all three occasions there were statistically significant decreases in SBP over 60 min ($p = 0.00$), however, the (test x time) interaction effect was not statistically significant ($p = 0.94$). Similarly, the ES analysis indicates that the training and detraining effects of HIIT on blood pressure recovery after maximal exercise (Table 7.4a and 7.4b) were of trivial and small practical significance, respectively (ES = 0.02 – 0.47).

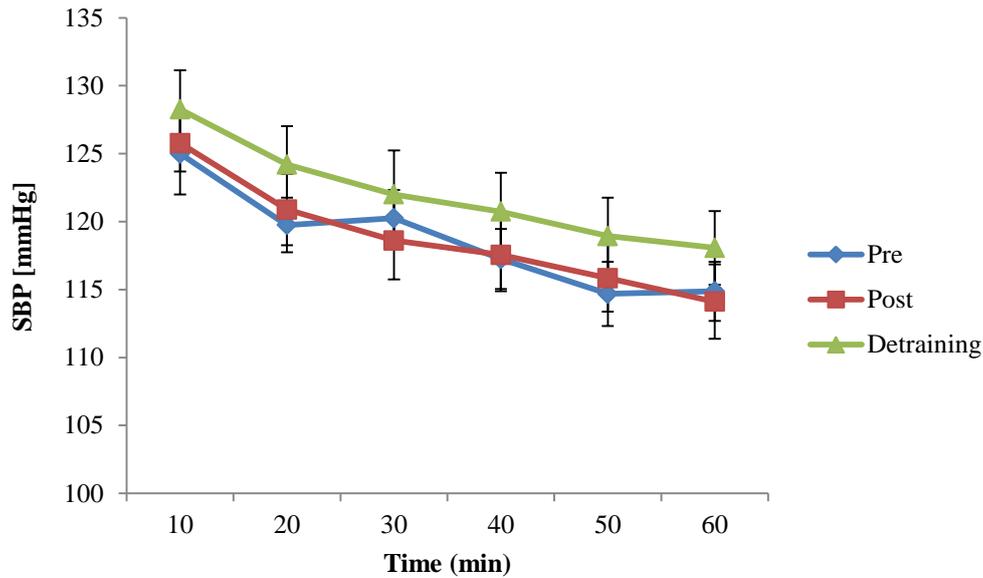


Figure 7.1a: Absolute change in SBP during recovery after the maximal exercise capacity tests

Table 7.4a: Effect sizes of mean SBP after maximal exercise capacity tests

Variable	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
SBP [mmHg]	0.02 [†]	0.28 ^²	0.32 ^²

ES, effect sizes; SBP, systolic blood pressure; Pre, pre-training; Post, post-training; [†] trivial practical significant effect; ^² small practical significance

Table 7.4b: Effect sizes of mean SBP at each time point during 60 min recovery

SBP at time points	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
SBP10	0.07 [†]	0.25 ^²	0.28 ^²
SBP20	0.12 [†]	0.30 ^²	0.47 ^²
SBP30	0.16 [†]	0.27 ^²	0.17 [†]
SBP40	0.03 [†]	0.28 ^²	0.35 ^²
SBP50	0.11 [†]	0.29 ^²	0.42 ^²
SBP60	0.07 [†]	0.36 ^²	0.33 ^²

ES, effect sizes; SBP, systolic blood pressure; Pre, pre-training; Post, post-training; [†] trivial practically significant effect; ^² small practical significant effect

The magnitude of the change in SBP during recovery after the maximal exercise capacity tests was statistically significant at each assessment. Figure 7.1b indicates that SBP lowered by 10.13 mmHg 7.76% ($p = 0.00$) after the baseline assessment, by 11.11 mmHg (8.84%; $p = 0.00$) after the HIIT intervention and by 10.2 mmHg (7.84%; $p = 0.00$) after detraining. However, the magnitude of these changes were not statistically significantly different between assessments ($p = 0.84$). The ES analysis also indicated only trivial practically significant differences in the training and detraining effects of HIIT on blood pressure recovery after maximal exercise.

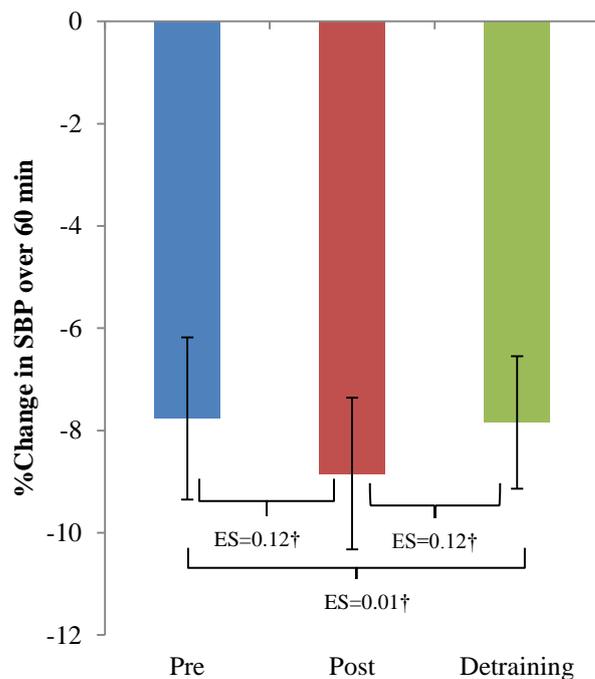


Figure 7.1b: Relative change in SBP over 60 min after maximal exercise capacity tests
 † Trivial practical significant effect between tests

Figure 7.1c depicts the change in SBP over 60 min after the maximal exercise capacity tests relative to the resting values. The magnitude of the systolic hypotensive responses were slightly greater after HIIT compared to pre-training (5.04 mmHg vs. 4.28 mmHg) and both would be considered clinically significant (> 3 mmHg decrease). This hypotensive response

was reached between 25 and 35 min after cessation of exercise. The PEH response after the detraining period was only 1.08 mmHg lower than resting values and was only reached after approximately 55 min.

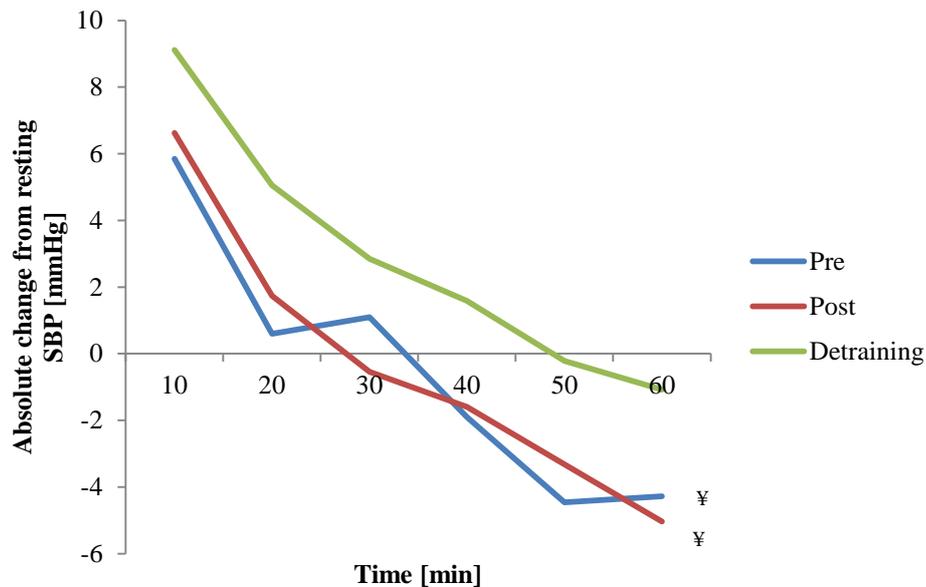


Figure 7.1c: Magnitude of PEH response in SBP relative to resting SBP
 ¥ Clinically significant (> 3 mmHg)

3.2. Diastolic Blood Pressure

Figure 7.2a displays the absolute change in DBP during recovery after the maximal exercise capacity tests. No statistically significant differences in mean DBP were observed after the tests (pre: 77.5 ± 6.1 mmHg; post: 76.9 ± 6.0 mmHg; detraining: 78.1 ± 6.8 mmHg; $p = 0.50$; ES = 0.08 – 0.54). There was a statistically significant time effect on mean DBP during recovery following all tests ($p = 0.00$), but the (test x time) interaction showed no statistically significant difference ($p = 0.06$). The training and detraining effects on mean DBP during recovery varied from trivial to small (Table 7.5a and 7.5b).

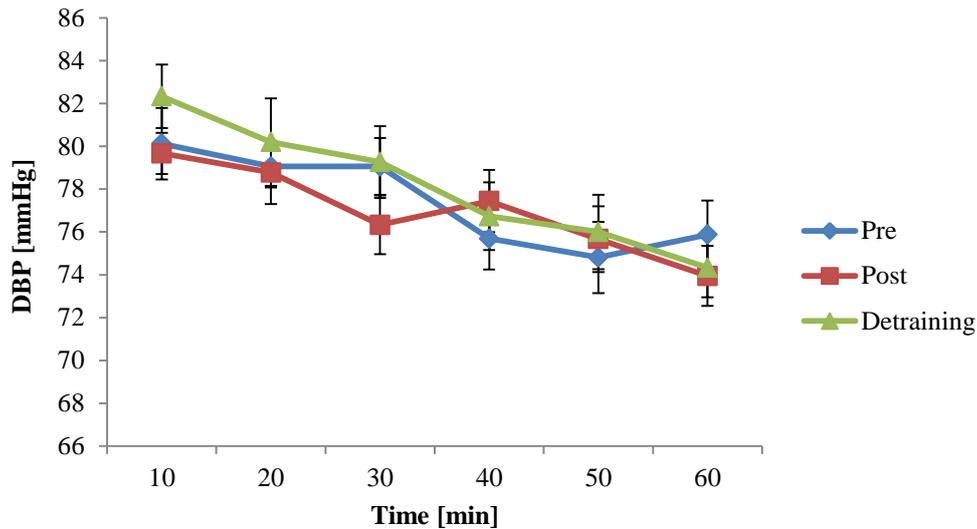


Figure 7.2a: Absolute change in DBP during recovery after the maximal exercise capacity tests

Table 7.5a: Effect sizes of mean DBP after maximal exercise capacity tests

Variable	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
DBP [mmHg]	0.08 [†]	0.18 [†]	0.11 [†]

ES, effect sizes; DBP, diastolic blood pressure; Pre, pre-training; Post, post-training; [†] trivial practically significant effect

Table 7.5b: Effect sizes of mean DBP at each time point during 60 min recovery

DBP at time points	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
DBP10	0.08 [†]	0.54 ^e	0.35 ^e
DBP20	0.05 [†]	0.20 ^e	0.18 [†]
DBP30	0.49 ^e	0.48 ^e	0.03 [†]
DBP40	0.29 ^e	0.12 [†]	0.18 [†]
DBP50	0.13 [†]	0.05 [†]	0.18 [†]
DBP60	0.31 ^e	0.07 [†]	0.26 ^e

ES, effect sizes; DBP, diastolic blood pressure; Pre, pre-training; Post, post-training; [†] trivial practical significant effect; ^e small practical significant effect

Figure 7.2b depicts the magnitude of change in DBP after each maximal capacity test. The change in DBP during recovery was least after baseline test (4.25 mmHg; 4.82%; $p = 0.04$),

followed by post-training assessment (5.72 mmHg; 7.13%; $p = 0.00$). The decrease in DBP during recovery was most pronounced after detraining assessment (8.0 mmHg; 9.63%; $p = 0.00$). The differences in the magnitudes of the changes were not statistically significant among tests periods ($p = 0.21$), however, these differences were classified as small to moderately practically significant ($ES = 0.22 - 0.61$).

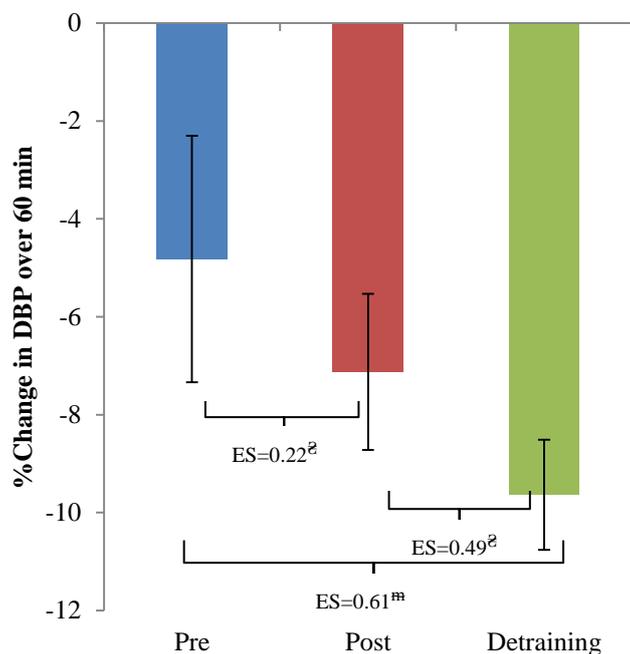


Figure 7.2b: Relative change in DBP over 60 min after maximal exercise capacity tests
^e Small practical significant effect between tests; ^m moderate practical effect between tests

Figure 7.2c shows the magnitude of the PEH response in DBP relative to resting value. After 60 min recovery, DBP were 2.33 mmHg, 4.26 mmHg and 3.87 mmHg, respectively lower compared to resting DBP. The magnitude of the PEH response in DBP was considered clinically significant (> 3 mmHg) after HIIT and the detraining period, but not at pre-training. The hypotensive effect was reached between 20 and 35 min post-exercise.

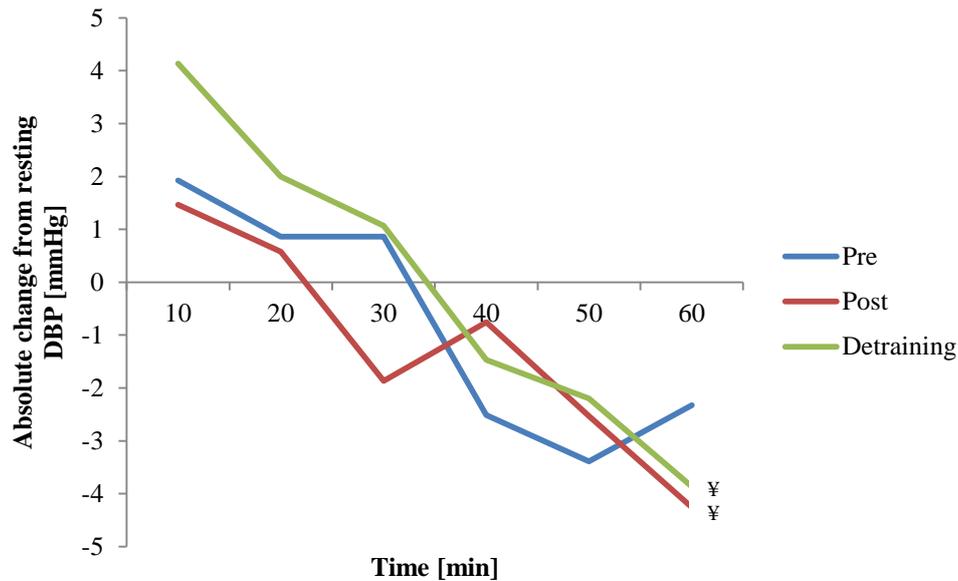


Figure 7.2c: Magnitude of PEH response in DBP relative to resting DBP
 ¥ Clinically significant (> 3 mmHg)

3.3. Mean arterial pressure

Figure 7.3a depicts the absolute change in MAP over 60 min after the maximal exercise capacity tests. The mean MAP were not statically significantly different between the pre-, post- and detraining tests (pre: 91.0 ± 6.8 mmHg; post: 90.1 ± 8.5 mmHg; detraining: 92.6 ± 7.7 mmHg; $p = 0.20$). However, there were statistically significant reductions in MAP over 60 min ($p = 0.00$) after all three tests. The (test x time) interaction effect was not statistically significant ($p = 0.15$) and ES analysis show that the training and detraining effects of HIIT on blood pressure recovery after maximal exercise (Table 7.6a and 7.6b) were trivial and small, respectively (ES = 0.05 – 0.51).

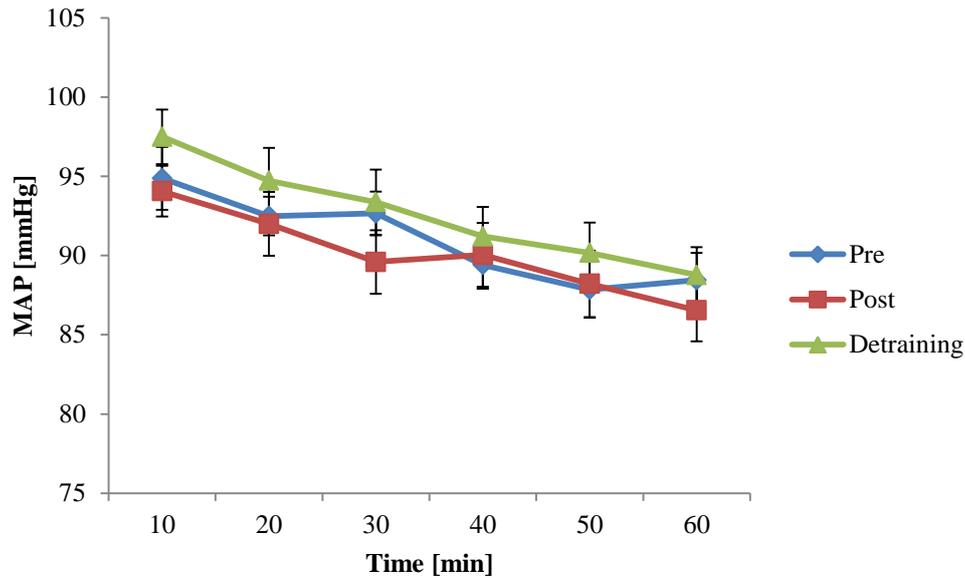


Figure 7.3a: Absolute change in MAP during recovery after the maximal exercise capacity tests

Table 7.6a: Effect sizes of mean MAP after maximal exercise capacity tests

Variable	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
MAP [mmHg]	0.11 [†]	0.31 ^²	0.23 ^²

ES, effect sizes; MAP, mean arterial pressure; Pre, pre-training; Post, post-training; [†] trivial practical significant effect; ^² small practical significant effect

Table 7.6b: Effect sizes of mean MAP at each time point during 60 min recovery

MAP at time points	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
MAP10	0.11 [†]	0.51 ^²	0.36 ^²
MAP20	0.07 [†]	0.33 ^²	0.34 ^²
MAP30	0.42 ^²	0.46 ^²	0.11 [†]
MAP40	0.09 [†]	0.15 [†]	0.27 ^²
MAP50	0.05 [†]	0.24 ^²	0.32 ^²
MAP60	0.25 ^²	0.29 ^²	0.05 [†]

ES, effect sizes; MAP, mean arterial pressure; Pre, pre-training; Post, post-training; [†] trivial practical significant effect; ^² small practical significant effect

The magnitude of the changes in MAP during recovery was statically significant after each maximal exercise capacity test (Figure 7.3b). These changes were 6.42 mmHg (6.47%; $p =$

0.00), 7.52 mmHg (8.03%; $p = 0.00$) and 8.73 mmHg (8.92%; $p = 0.00$), respectively after pre-, post- and detraining. The magnitude of the changes were not statistically significantly different among tests periods ($p = 0.46$) and these differences were also trivial to small (ES = 0.18 – 0.41).

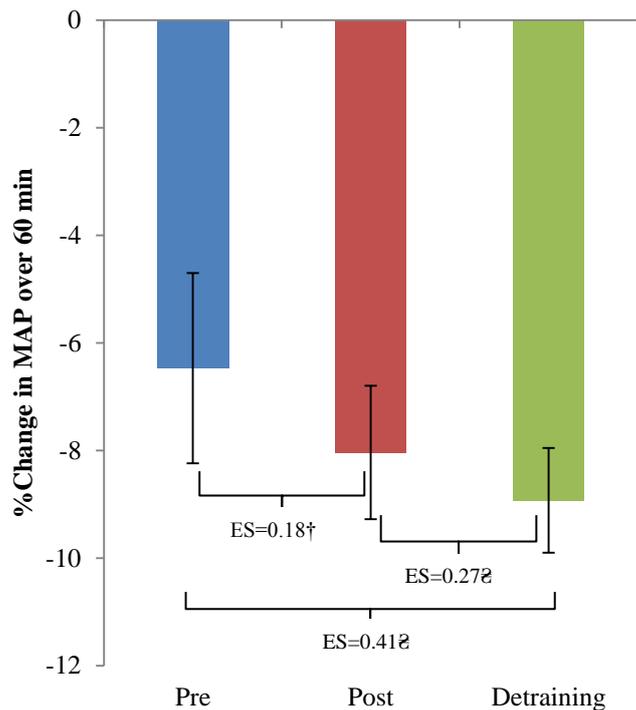


Figure 7.3b: Relative change in MAP over 60 min after maximal exercise capacity tests

⊘ Small practical significant effect between tests; † trivial practical significant effect between tests

Figure 7.3c shows the change in MAP relative to the resting values following the maximal exercise capacity tests. The magnitude of the hypotensive responses in MAP were greater after HIIT post-training compared to pre-training (5.17 mmHg vs. 3.26 mmHg) and both would be considered clinically significant (> 3 mmHg decrease). This hypotensive response was reached between 20 and 30 min after end of exercise. The PEH response after the

detraining period approached near clinical significance, as it was 2.94 mmHg lower than resting values and was only reached after approximately 40 min.

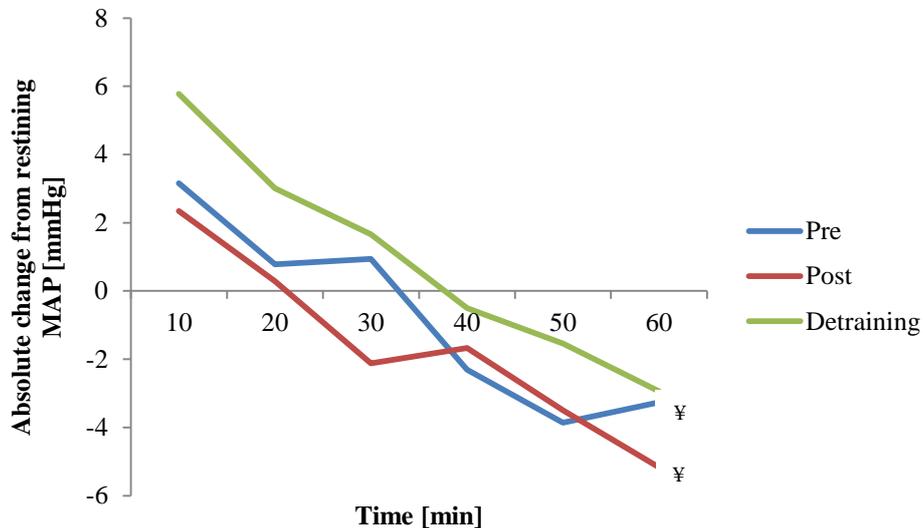


Figure 7.3c: Magnitude of PEH response in MAP relative to resting MAP
 ¥ Clinically significant (> 3 mmHg)

3.4. Heart rate

Figure 7.4a represents the absolute change in HR during 60 min recovery after maximal exercise capacity tests. There were no statistically significant differences in mean HR after the tests (pre: 96.6 ± 11.9 b.min⁻¹; post: 93.0 ± 11.1 b.min⁻¹; detraining: 96.6 ± 9.7 b.min⁻¹; $p = 0.16$; ES = 0.1 - 0.82). However, the time effect on mean HR during recovery following all tests ($p = 0.00$) as well as the (test x time) interaction ($p = 0.03$) were statistically significant. The differences in HR between assessments varied from trivial to moderate practical significance (Table 7.7a and 7.7b).

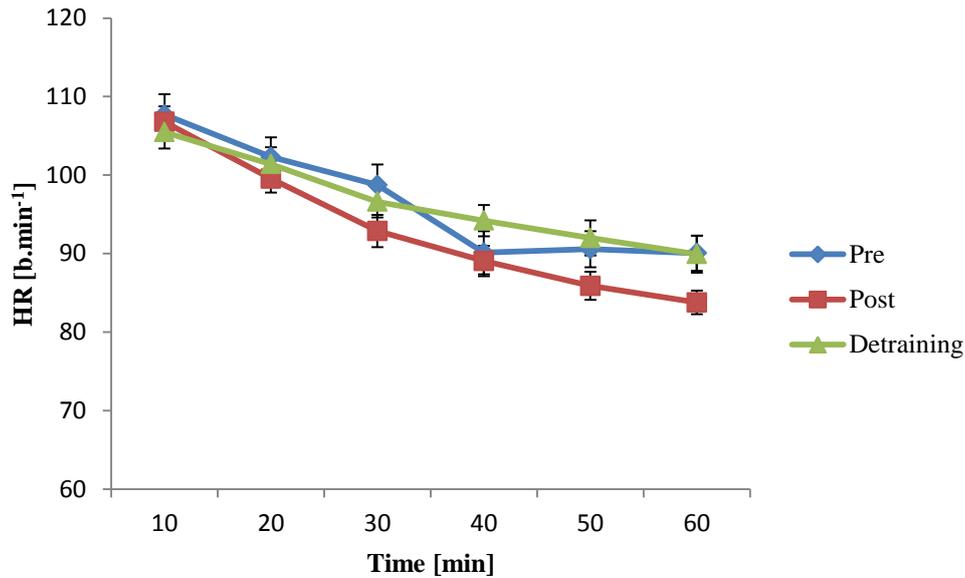


Figure 7.4a: Absolute change in HR during recovery after maximal exercise capacity tests

Table 7.7a: Effect sizes of mean HR after maximal exercise capacity tests

Variable	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
HR [b.m ⁻¹]	0.00	0.34 ^e	0.33 ^e

ES, effect sizes; HR, heart rate; Pre, pre-training; Post, post-training; ^e small practical significant effect

Table 7.7b: Effect sizes of mean HR at each time point during 60 min recovery

HR at Time points	Pre vs. Post ES	Post vs. Detraining ES	Pre vs. Detraining ES
HR10	0.10 [†]	0.16 [†]	0.24 ^e
HR20	0.32 ^e	0.23 ^e	0.10 [†]
HR30	0.62 ^m	0.45 ^e	0.23 ^e
HR40	0.11 [†]	0.65 ^m	0.43 ^e
HR50	0.56 ^e	0.76 ^m	0.16 [†]
HR60	0.82 ^m	0.80 ^m	0.01 [†]

ES, effect sizes; HR, heart rate; Pre, pre-training; Post, post-training; [†] trivial practical significant effect; ^e small practical significant effect; ^m moderate practical significant effect

The magnitude of changes in HR after the maximal exercise capacity tests was statistically significant for each assessment by 17.63 b.min⁻¹ at 15.96% ($p = 0.00$), 23.0 b.min⁻¹ at 21.28% ($p = 0.00$) and 15.53 b.min⁻¹ at 14.67% ($p = 0.00$) respectively after pre-, post- and detraining.

There were statistically significant differences in the magnitude of the changes among tests periods ($p = 0.02$). Similarly, analysis of ES, as depicted in Figure 7.4b, shows small to moderate practically significant faster recovery in all tests after maximal exercise. The largest change in HR recovery was seen after the HIIT intervention ($23.0 \text{ b}\cdot\text{min}^{-1}$; 21.28% ; $p = 0.00$), while the magnitude of decrease in HR at pre- training and after detraining was similar ($17.63 \text{ b}\cdot\text{min}^{-1}$ vs. $15.53 \text{ b}\cdot\text{min}^{-1}$ respectively).

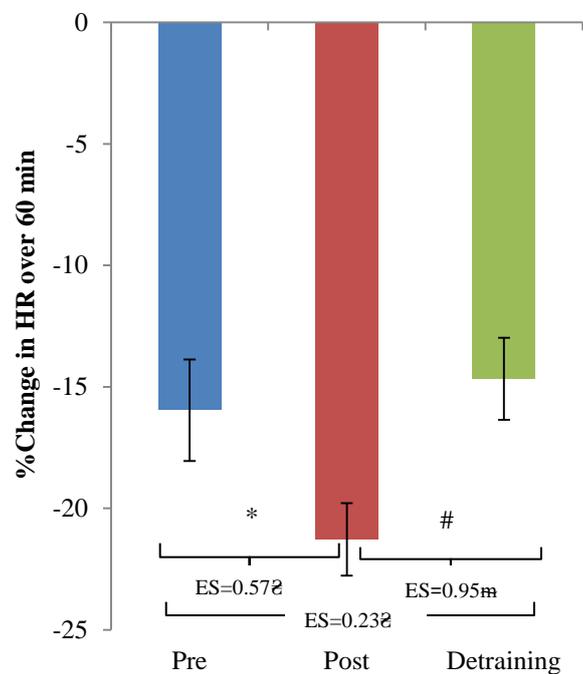


Figure 7.4b: Relative change in HR over 60 min after maximal exercise capacity tests

* Significantly different between pre- and post-training, $p < 0.05$; # significantly different between post- and detraining, $p < 0.05$; ^m moderate practical significant effect between tests; ^s small practical significant effect between test

4. The effects of acute and six HIIT sessions on BP and HR

All twenty subjects completed six HIIT sessions within two weeks. Ambulatory SBP, DBP and HR were measured over 60 min as well as MAP calculated after the first (acute) HIIT

session and the last (following six sessions) HIIT session. These measurements were taken every 10 min over 60 min duration post exercise seated rest.

4.1. Systolic blood pressure

Figure 7.5a illustrates the absolute change in SBP during recovery after HIIT sessions. Although mean SBP was not statistically significantly different between the first and the last training sessions (118.8 ± 9.1 mmHg and 116.7 ± 12.2 mmHg; $p = 0.28$; ES = 0.20), the analysis of the effect size shows a small practically significant difference (Table 7.8). There were statistically significant reductions in SBP over 60 min after both the first and the last training sessions ($p = 0.00$), however, the (test x time) interaction effect was not statistically significant ($p = 0.31$). Likewise ES analysis showed trivial to small practically significant differences at each time point between the first and the last HIIT sessions (Table 7.9).

The magnitude of change in SBP was statistically significantly after the first and the last HIIT sessions (8.25 mmHg; 6.56%; $p = 0.00$ vs. 7.65 mmHg; 6.42%; $p = 0.00$). However, the difference in SBP during recovery between the first and the last HIIT sessions were not statistically significantly different ($p = 0.93$) and the ES revealed only a trivial practical significant difference (ES = 0.03) (Figure 7.5b).

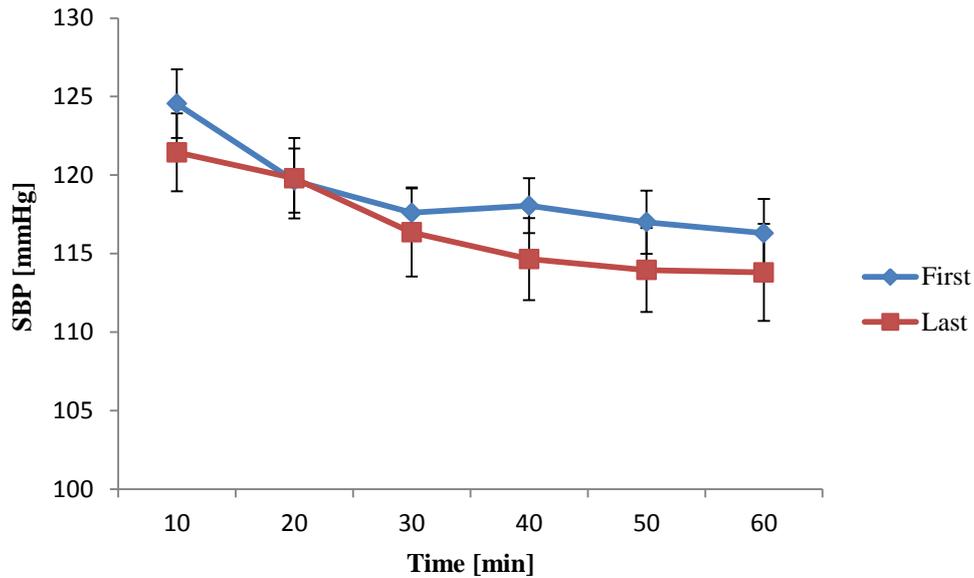


Figure 7.5a: Absolute change in SBP during recovery after HIIT sessions

Table 7.8: Effect sizes of mean BP and HR after the first and last HIIT sessions

HIIT sessions	SBP [mmHg] ES	DBP [mmHg] ES	MAP mmHg] ES	HR [b.m ⁻¹] ES
First vs. Last	0.20 [Ⓔ]	0.21 [Ⓔ]	0.18 [†]	0.16 [†]

ES, effect sizes; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; mmHg, millimeter mercury; HR, heart rate; b.min⁻¹, beats per minute; [Ⓔ] small practical significant effect; [†] trivial practical significant effect

Table 7.9: Effect sizes of SBP at each time point during 60 min recovery after HIIT sessions

HIIT sessions	SBP 10 ES	SBP 20 ES	SBP 30 ES	SBP 40 ES	SBP 50 ES	SBP 60 ES
First vs. Last	0.30 [Ⓔ]	0.01 [†]	0.12 [†]	0.34 [Ⓔ]	0.29 [Ⓔ]	0.21 [Ⓔ]

ES, effect sizes; SBP, systolic blood pressure; [†] trivial practical significant effect; [Ⓔ] small practical significant effect

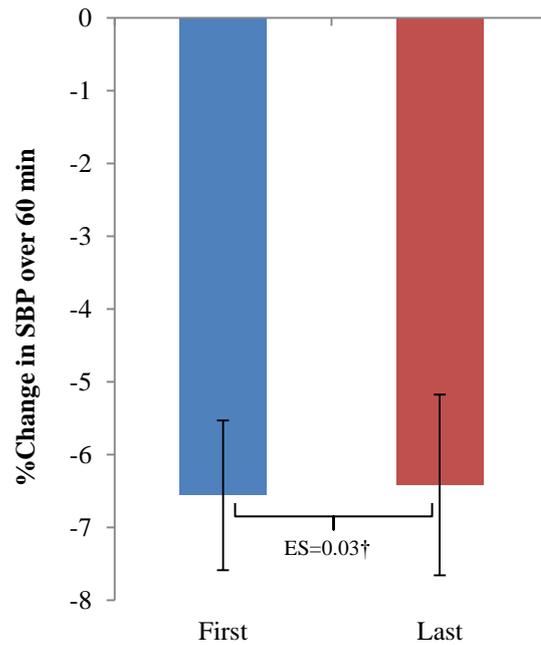


Figure 7.5b: Relative change in SBP during recovery after HIIT sessions
† Trivial practical significant effect between tests

Figure 7.5c indicates the change in SBP after the first and last HIIT sessions relative to the resting values. The magnitude of the systolic hypotensive response following the last HIIT session was greater compared to the first HIIT session (5.35 mmHg vs. 2.85 mmHg, respectively), but only the last HIIT resulted in a clinically significant PEH response, while the first HIIT approached near clinical significance. This hypotensive response was reached between 20 and 30 min after termination of exercise.

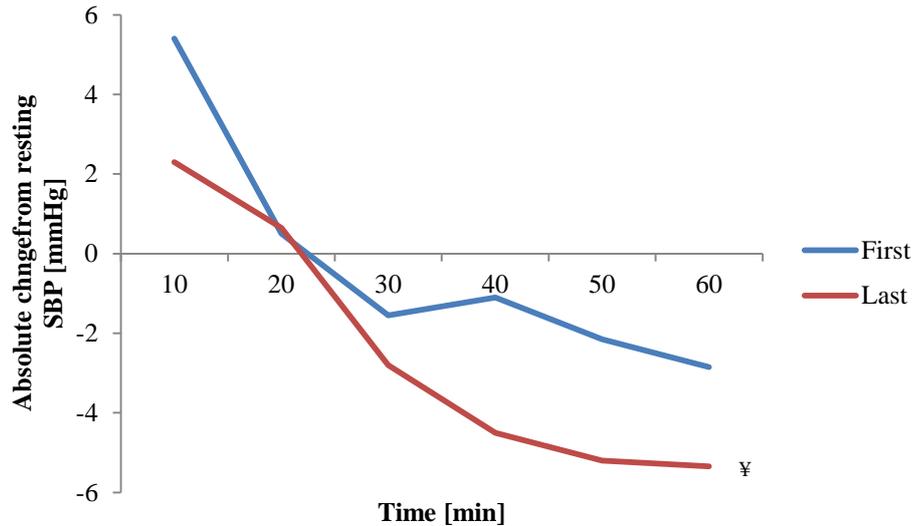


Figure 7.5c: Magnitude of PEH response in SBP after HIIT sessions relative to resting SBP
 ¥ Clinically significant (> 3 mmHg)

4.2. Diastolic Blood Pressure

Figure 7.6a represents the absolute change in DBP during recovery after the HIIT sessions. There was no statistically significant difference in mean DBP between the first and the last training sessions (77.6 ± 6.0 mmHg and 76.2 ± 7.1 mmHg; $p = 0.18$; ES = 0.21), and ES analysis confirms a small practically significant difference (Table 7.8). There were statistically significant reductions in DBP over 60 min after both the first and the last HIIT sessions ($p = 0.00$). Conversely, the (test x time) interaction effect was not statistically significant ($p = 0.58$). ES analysis, as depicted in Table 7.10, showed trivial to small practically significant differences at each time point.

The magnitude of the reductions in DBP was statistically significant on both occasions (2.55 mmHg; 2.62%; $p = 0.00$ vs. 3.14 mmHg; 4.30%; $p = 0.01$) after the first and last HIIT session, respectively. These magnitudes of change during 60 min recovery were not

statistically significantly different between the training sessions ($p = 0.42$; $ES = 0.14$) and a trivial practically significant difference was observed (Figure 7.6b).

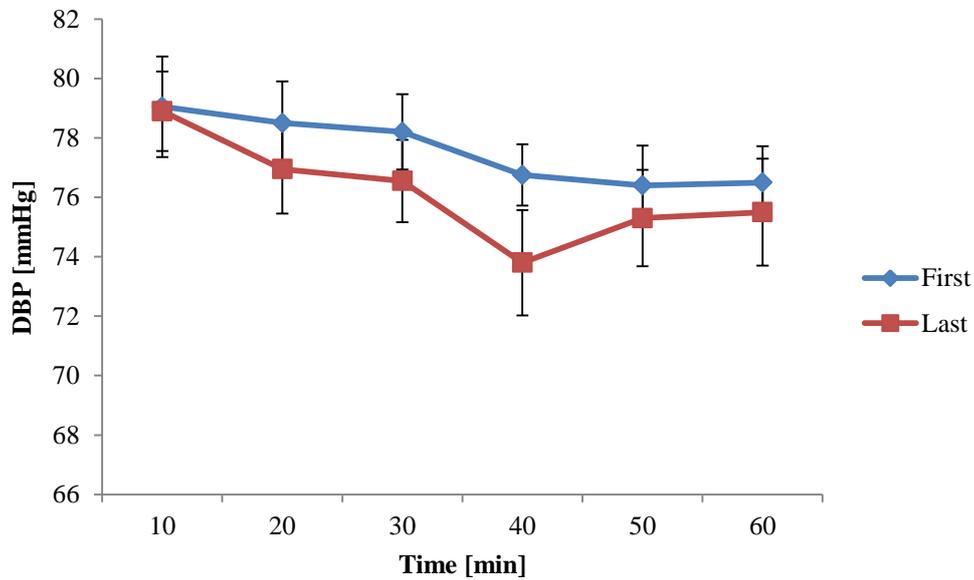


Figure 7.6a: Absolute change in DBP during recovery after HIIT sessions

Table 7.10: Effect sizes of DBP at each time point during 60 min recovery after HIIT sessions

HIIT sessions	DBP 10 ES	DBP 20 ES	DBP 30 ES	DBP 40 ES	DBP 50 ES	DBP 60 ES
First vs. Last	0.02 [†]	0.24 [Ⓜ]	0.28 [Ⓜ]	0.46 [Ⓜ]	0.17 [†]	0.15 [†]

ES, effect sizes; DBP, diastolic blood pressure; [†] trivial practical significant effect; [Ⓜ] small practical significant effect

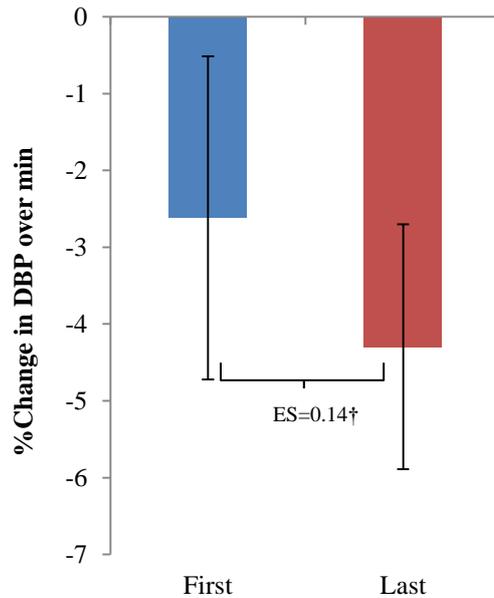


Figure 7.6b: Relative change in DBP during recovery after HIIT sessions

† Trivial practical significant effect between tests

Figure 7.6c displays the change in DBP after the first and last HIIT sessions relative to the resting values. The magnitude of the diastolic hypotensive response following the last HIIT session was greater compared to the first HIIT session (2.7 mmHg vs. 1.7 mmHg respectively) and was reached between 10 and 30 min after the end of exercise. Both hypotensive responses would not be considered clinically significant (< 3 mmHg decrease), even though the hypotensive response following the last HIIT session approached clinical significance.

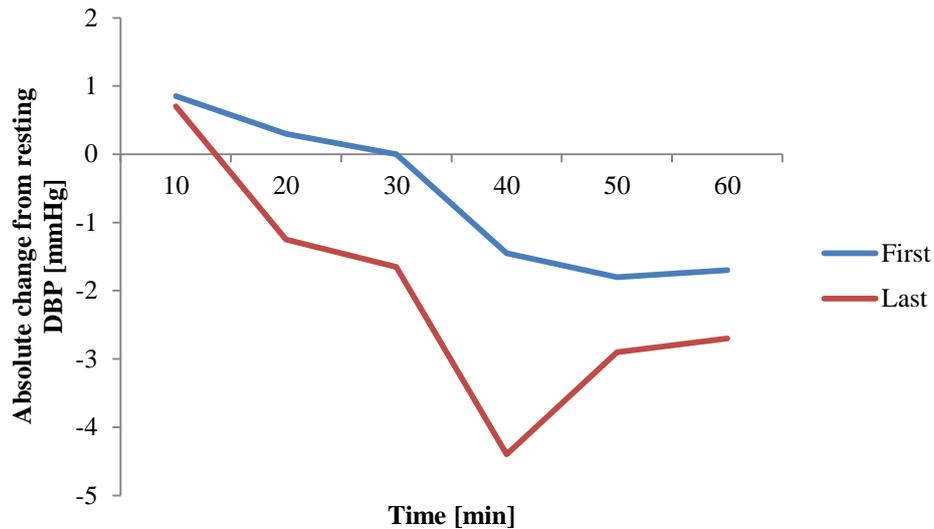


Figure 7.6c: Magnitude of PEH response in DBP after HIIT sessions relative to resting DBP

4.3. Responders and non-responders to PEH following HIIT sessions

More than half of the participants (55%) showed a clinically significant PEH response in SBP following the first HIIT session, while 75% of participants responded with a clinically significant PEH response in SBP following the last HIIT session. In terms of DBP, 60% and 80% of participants responded with a clinically significant reduction in DBP after first and last HIIT session respectively.

4.4. Mean Arterial Pressure

Figure 7.7a shows the absolute change in MAP during recovery after the HIIT sessions. The mean MAP was not statistically significantly different between the first and the last training sessions, respectively (90.8 ± 6.1 mmHg and 89.5 ± 8.1 mmHg; $p = 0.31$; ES = 0.18). MAP decreased statistically significantly over 60 min after both the first and the last training sessions ($p = 0.00$), however, the (test x time) interaction effect was not statistically

significant ($p = 0.55$). Also ES analysis showed trivial to small practically significant effects at each time point (Table 7.11).

The magnitude of changes in MAP was statistically significant after both HIIT sessions (4.05 mmHg; 4.1%; $p = 0.01$ vs. 4.80 mmHg; 5.27%; $p = 0.00$). The difference of the magnitudes between the first and last HIIT sessions were, however, not statistically significant and only of trivial practical significance ($p = 0.40$; ES = 0.14) (Figure 7.7b).

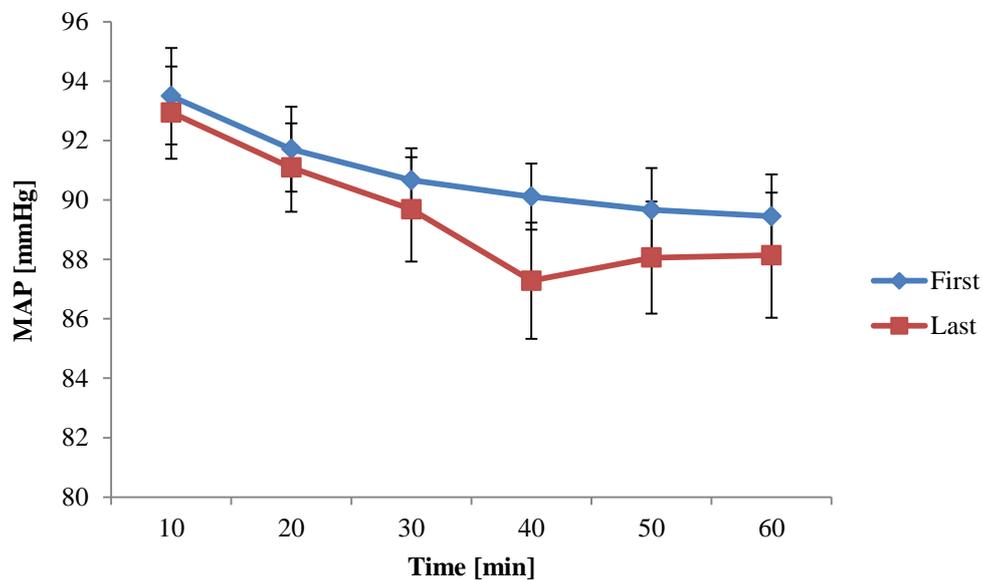


Figure 7.7a: Absolute change in MAP during recovery after HIIT sessions

Table 7.11: Effect sizes of MAP at each time point during 60 min recovery after HIIT sessions

HIIT sessions	MAP 10 ES	MAP 20 ES	MAP 30 ES	MAP 40 ES	MAP 50 ES	MAP 60 ES
First vs. Last	0.08 [†]	0.10 [†]	0.15 [†]	0.40 [Ⓐ]	0.22 [Ⓐ]	0.16 [†]

ES, effect sizes; MAP, mean arterial pressure; [†] trivial practical significant effect; [Ⓐ] small practical significant effect

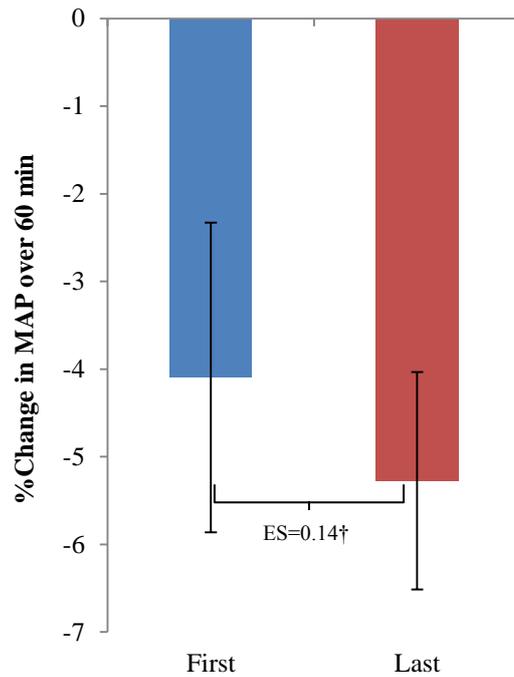


Figure 7.7b: Relative change in MAP during recovery after HIIT sessions

† Trivial practical significant effect between tests

Figure 7.7c shows the change in MAP after the first and last sessions of HIIT relative to the resting values. The magnitude of the PEH response in MAP following the last HIIT session was greater compared to the first HIIT session (3.57 mmHg vs. 2.26 mmHg, respectively) but only the last HIIT was considered clinically significant. This hypotensive response was reached between 10 and 20 min following cessation of exercise.

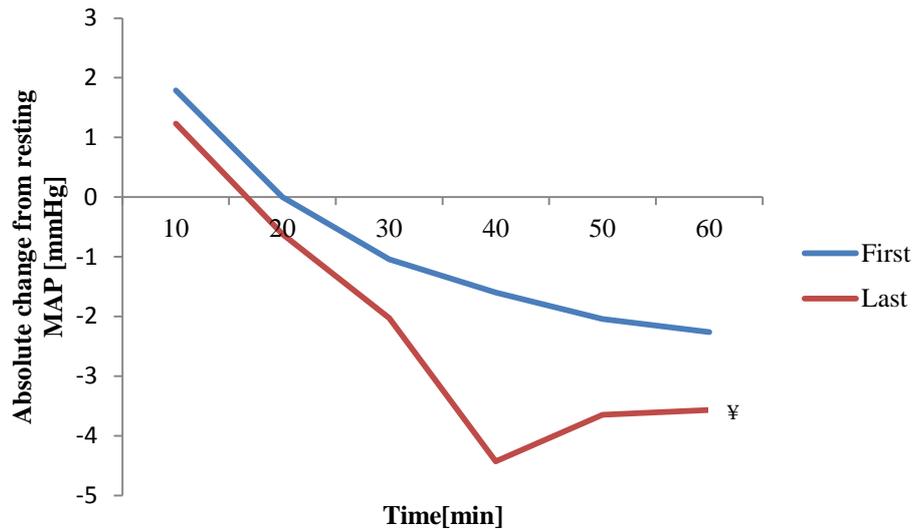


Table 7.7c: Magnitude of PEH response in MAP after HIIT sessions relative to resting MAP

¥ Clinically significant (> 3 mmHg)

4.5. Heart rate

Figure 7.8a refers to the absolute change in mean HR duration recovery after HIIT sessions. There was no statistically significant difference in mean HR between the first and the last training sessions ($93.0 \pm 12.0 \text{ b}\cdot\text{min}^{-1}$ and $91.4 \pm 9.2 \text{ b}\cdot\text{min}^{-1}$; $p = 0.36$; $ES = 0.16$). Although HR decreased statistically significantly over the 60 min period ($p = 0.00$), the (test x time) interaction was not statically significant ($p = 0.41$). Trivial to small practically significant effects were observed at each time point over 60 min (Table 7.12).

The magnitude of change in HR was statistically significantly after both HIIT sessions ($16.20 \text{ b}\cdot\text{min}^{-1}$; 15.53% ; $p = 0.00$ vs. $14.15 \text{ b}\cdot\text{min}^{-1}$; 13.88% ; $p = 0.00$). However, the difference in HR recovery were not statistically significant between the first and last HIIT session ($p = 0.45$; $ES = 0.27$), although the difference can be described as a small practically significant effect (Figure 7.8b).

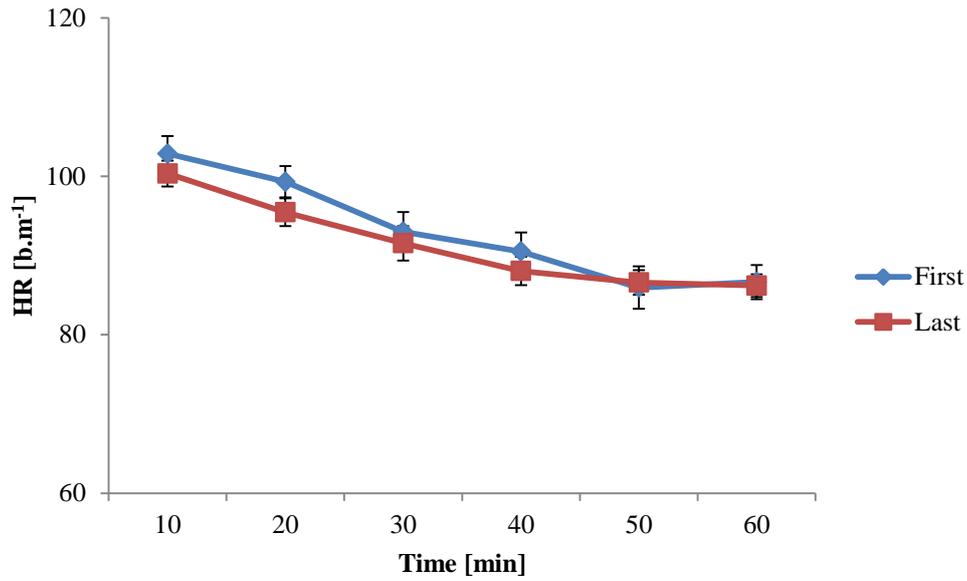


Figure 7.8a: Absolute change in HR during recovery after HIIT sessions

Table 7.12: Effect sizes of HR at each time point during 60 min recovery after HIIT sessions

HIIT sessions	HR 10 ES	HR 20 ES	HR 30 ES	HR 40 ES	HR 50 ES	HR 60 ES
First vs. Last	0.35 ^e	0.46 ^e	0.14 [†]	0.26 ^e	0.07 [†]	0.05 [†]

ES, effect sizes; HR, heart rate; [†] trivial practical significant effect; ^e small practical significant effect

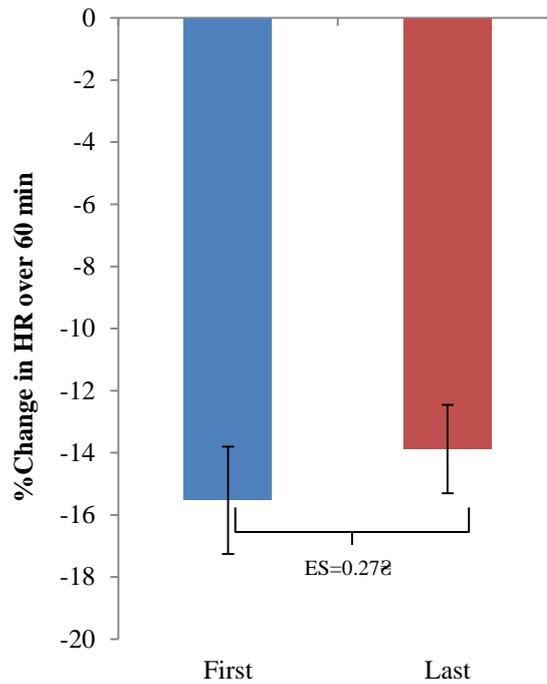


Figure 7 8b: Relative change in HR during recovery after HIIT sessions
 ‡ Small practical significant effect between tests

5. Relationship between cardiovascular risk factors and changes in BP

Table 7.13 shows the correlations between selected cardiovascular risk factors and changes in SBP, DBP and MAP. Reduction in SBP correlated moderately with resting SBP, VO_{2max} , BMI and %BF ($p < 0.05$) following the last HIIT, session but correlated weakly with all the cardiovascular risks factors after the first HIIT session. Reductions in DBP on the other hand correlated poorly with all the cardiovascular risks factors following the HIIT sessions, except BMI and %BF ($p < 0.05$), which had moderately to good correlations following the first HIIT session. Reduction in MAP also correlated moderately to all risk factors following the last HIIT session but poorly with resting MAP and VO_{2max} and moderately to good with % BF and BMI following the first HIIT session.

Table 7.13a: The relationship between the change in mean SBP after the HIIT sessions and selected cardiovascular risk factors

Variables	First HIIT	Last HIIT
Δ SBP vs. resting SBP	-0.06	0.31
Δ SBP vs. VO_{2max}	0.06	-0.43
Δ SBP vs. BMI	0.05	0.30
Δ SBP vs. %BF	-0.19	0.47

Δ , change in; SBP, systolic blood pressure; VO_{2max} , maximum aerobic capacity; BMI, body mass index; %BF, percentage body fat

Table 7.13b: The relationship between the change in mean DBP after the HIIT sessions and the selected cardiovascular risk factors

Variables	First HIIT	Last HIIT
Δ DBP vs. resting DBP	-0.08	0.16
Δ DBP vs. VO_{2max}	-0.30	-0.07
Δ DBP vs. BMI	0.48	0.16
Δ DBP vs. %BF	0.54	0.10

Δ , change in; DBP, diastolic blood pressure; VO_{2max} , maximum aerobic capacity; BMI, body mass index; %BF, percentage body fat

Table 7.13c: The relationship between the change in mean MAP after the HIIT sessions and the selected cardiovascular risk factors

Variables	First HIIT	Last HIIT
Δ MAP vs. resting MAP	-0.07	0.32
Δ MAP vs. VO_{2max}	-0.19	-0.24
Δ MAP vs. BMI	0.52	0.25
Δ MAP vs. %BF	0.35	0.28

Δ , change in; MAP, mean arterial pressure; VO_{2max} , maximum aerobic capacity; BMI, body mass index; %BF, percentage body fat

CHAPTER EIGHT

DISCUSSION

A. INTRODUCTION

The purpose of the current study was to investigate the effect of a short-term HIIT intervention on the magnitude of the PEH response in overweight/obese young women. Research on PEH relating to HIIT is limited, especially in women. Therefore this study will not only add to the broad knowledge base of this field, but also could have important practical application.

The main finding of the study was that both an acute bout and six HIIT sessions resulted in a significant reduction in SBP. The reductions were greater after the six HIIT sessions than the acute HIIT session, although the magnitude of reduction was not statistically significantly different between the two. DBP also lowered significantly after both HIIT sessions with no difference between the magnitudes of reduction. Nevertheless, DBP remained lower than resting values after detraining while SBP was consistently higher than resting values following a maximal capacity test after two weeks of detraining. HR decreased significantly after HIIT sessions with greater effects after the six HIIT sessions than the acute session, but detraining returned HR to pre-training values. The results suggest that HIIT is effective in reducing BP after both the acute and six sessions of training.

B. DESCRIPTIVE STATISTICS

The main cause of morbidity and mortality in developing countries all over the world is the fast growing nature of cardiovascular diseases (CVD), with hypertension being one of the

main risk factors (Krisela *et al.*, 2001). According to Steyn *et al.* (2008) the main predictor for hypertension is an urbanized lifestyle, with a high stress job, inactivity and an unhealthy diet. These life styles are not different among individuals who are overweight or obese as the positive relationship between being overweight and all-cause mortality indicates another risk factor for CVD (Harris *et al.*, 2008). Considering the fact that SBP increases with advancing age (Pescatello *et al.*, 2004a), a high BP can be problematic at any age and may actually exacerbate the natural and inevitable effects of ageing.

In this study the subjects were normotensive young women between 18 to 25 years. These subjects were overweight or obese and sedentary. Their aerobic fitness levels were fair (VO_{2max} ; 27.8 ± 5.69) according to the norms of Hoffman (2006) (VO_{2max} : 25 - 33 ml.kg⁻¹.min⁻¹). Considering that these are young individuals, and that VO_{2max} declines fairly rapidly with age in the absence of regular physical activity, these women are at risk to present with rather low VO_{2max} values within the next decade or two. For this reason, as well as for the fact that they are already overweight or obese, it is necessary to address these issues with powerful interventions that will lead to significant and lasting effects.

Body mass index (BMI) is generally used as an overall indicator of health status and the relative risk of developing CVD. The American College of Sports Medicine (ACSM) classifies a person with a BMI between 25.0 kg.m⁻² and 29.9 kg.m⁻², and between 30.0 kg.m⁻² and 40.0 kg.m⁻² as overweight and obese, respectively. Cardiovascular risk is higher in individuals within this category (ACSM, 2006). In this study six individuals were classified as obese (30%) and 14 (70%) of the subjects were overweight.

A positive PEH response (i.e. BP after exercise that is lower than resting values), essentially indicates the short-term decline in BP observed in the minutes or hours following an acute bout of exercise (Kenney and Seals, 1993). The results emanating from this study revealed

that both the acute and six sessions of HIIT were effective in causing PEH. 55% of subjects had lower SBP after the acute HIIT session compared to resting values, while 75% of the subjects had a hypotensive response after the six HIIT sessions. Thus an additional 20% of subjects improved their cardiovascular health after only six exercise sessions. Of the non-responders, some participants remained unchanged or showed an increase in their BP after HIIT. 60% and 75% of the subjects had lower DBP after exercise compared to resting values, following the acute and six HIIT sessions, respectively. The positive PEH response observed in the responders is considered clinically significant as such reductions in BP were greater than 3 mmHg (Touyz *et al.*, 2004). This might explain the difference in the correlation between resting values and PEH responses (six HIIT sessions: DBP; $r = 0.16$; SBP; $r = 0.31$ and acute HIT: DBP; $r = -0.08$; SBP; $r = -0.06$). The finding is in line with Forjaz *et al.* (2000) who reported that 67% of normotensive subjects in their study presented with reductions in BP, while 65% of hypertensive individuals responded following 45 min of cycling exercise at 50% VO_{2max} .

Cardiovascular fitness (VO_{2max}) was improved (6.7%; $p < 0.05$) after only six sessions of HIIT even though the participants as a group could still be categorized as in the fair range. It is suggested that cardiovascular morbidity and mortality may possibly be decreased with substantially less exercise only if it is performed at an intensity that is intense enough than what is generally recommended (Fletcher *et al.*, 2001). In a review on the effects of physical inactivity and obesity on morbidity and mortality, Blair and Brodny (1999) observed that overweightness and VO_{2max} are both independent predictive markers of cardiovascular mortality and there appears to be an important relationship between these two variables. Myers *et al.* (2002) re-emphasized this observation by suggesting that of all established cardiovascular risk factors, low VO_{2max} appears to be the strongest predictor of mortality, while improvements in VO_{2max} are related with lower mortality. Additionally, studies have

shown that one metabolic equivalent of task rise (1-MET $\approx 3.5\text{ml.kg}^{-1}.\text{min}^{-1}$) in aerobic exercise capacity induce an 8 to 17% decrease in cardiovascular and all-cause mortality (Gulati *et al.*, 2003; Myers *et al.*, 2002; Blair *et al.*, 1995).

In this study the significance of the increase in $\text{VO}_{2\text{max}}$ post-training in these overweight and obese subjects suggests improvement in cardiovascular fitness which could protect against premature cardiovascular mortality. This is consistent with the findings of Schjerve *et al.* (2008) who showed a 16 - 46% increment in $\text{VO}_{2\text{max}}$ after 8 – 16 weeks of HIIT at $\sim 90\%$ HR_{max} in overweight and obese individuals. Likewise, the finding of this study is in line with Tjønnå *et al.* (2013) where both a single bout of 4-min and 4 x 4 min at 90% HR_{max} aerobic interval training performed three times per week for 10 weeks improved $\text{VO}_{2\text{max}}$ by 10% and 13%, respectively, in overweight middle-aged men. Even though the percentage improvement in this study was smaller (5.4%), it is promising that such a short intervention of two weeks (six sessions) actually resulted in an improvement in $\text{VO}_{2\text{max}}$. The difference in relative change of improvement could be due to the longer duration (8 – 16 weeks) of training compared to the current study which was only two weeks (six HIIT sessions). Nevertheless, the prospect of this increase in $\text{VO}_{2\text{max}}$ will likely have a positive effect on individuals' compliance and encourage them to continue training to gain even better results. In this regard the single bout 4-min intervention of Tjønnå *et al.* (2013) and the positive results they obtained are so important, as it suggests that once individuals are accustomed to HIIT (i.e. after six sessions), they can improve their maximal exercise capacity further by adding just one HIIT session per week to their training program. This positive outcome on $\text{VO}_{2\text{max}}$, as well as its possible effect on long-term exercise adherence, is unlikely with traditional endurance based training, in which individuals have to invest a lot more time and effort.

Unfortunately, detraining in two weeks reversed (reduced) the VO_{2max} values to the original values (pre-training values). This is supported by the finding of Coyle *et al.* (1986) who studied eight endurance-trained subjects who detrained for two weeks and experienced a 6% reduction in VO_{2max} . Houston *et al.* (1979) demonstrated that 15 days of detraining (7 days of leg casting followed by 8 days of no training) resulted in a 4% reduction in VO_{2max} in well-trained endurance runners. Similarly, VO_{2max} also decreased by 4.7% in a group of endurance-trained runners after two weeks of detraining (Houmard *et al.*, 1992). This is probably because cardiovascular function following a few weeks of detraining is largely due to a reduction in blood volume, which appears to limit ventricular filling during upright exercise (Coyle *et al.*, 1986). Mujika and Padilla (2001) contend that the reduction in VO_{2max} during detraining appears to be dependent on the time period and initial fitness level. Moreover, according to the principle of training reversibility, cessation or marked reduction in training result in a partial or complete reversal of physiological adaptations induced by regular exercise training (McArdle *et al.*, 2010). This suggests that the acquired protective benefits of improved VO_{2max} will return to initial values if training is not continued. It is therefore advised that exercise training be a continual program to maintain the protective benefits of exercise. However, this study, as well as the study by Tjønnå *et al.* (2013) emphasizes the benefit of HIIT in maintaining, and improving maximal exercise capacity. Thus, although the training effect is almost completely reversed within two weeks of detraining, individuals will be encouraged by the fact that a minimal investment in training time (i.e. one to three sessions of HIIT, which is equivalent to 4 to 30 min exercise per week), can actually improve their endurance capacity.

C. OUTCOME VARIABLES

1. Changes in resting BP and HR

Resting SBP, DBP and MAP were significantly lowered after the training intervention compared to pre-training values, but detraining reversed the training effects on resting SBP to near pre-training values (Table 7.2a). However, resting DBP and MAP remained significantly lower after detraining.

The findings of this study demonstrated that the magnitude of reduction in resting BP after the training intervention was clinically significant and can be compared to the results of Murray *et al.* (2006) who reported significant reductions in resting SBP (14 mmHg), DBP (13 mmHg) and MAP (14 mmHg) after a four week program of cycle ergometry (30 min at 60% VO_{2peak}) performed 3 – 4 times per week. Similar to this study, they also observed detraining effects but in the case of the present study, only SBP returned to near baseline values following detraining. However, within two weeks of detraining SBP, DBP and MAP increased by 7 mmHg each in the study by Murray *et al.* (2006). The reason for the difference in results after detraining is not known. However, it can be suggested that the difference in training intensity might have caused the difference in results, such that the HIIT utilized in the present study might have caused longer lasting lower blood pressures.

The findings of this study disagreed with Astorino *et al.* (2012) who observed no reduction in resting BP of recreationally active young men and women following two weeks of high intensity cycle sprints (Wingate test). In this study, subjects exercised twice per week and thus the rest days outnumbered the training days. Furthermore, the recovery rest interval was longer (30 sec x 5 min) compared to the rest intervals of the current study (1 min). It is therefore suggested that the volume of training in the study of Astorino *et al.* (2012) was possibly not sufficient to cause an accumulation of physiological adaptations. This reiterates

the recommendation that exercise training should be performed for most days of the week to ensure an adequate training stimulus for adaptations to take place.

Resting HR was slightly reduced after the training intervention which was not statistically significant, but detraining significantly increased resting HR to pre-training levels.

The following selected cardiovascular risk factors, namely resting SBP, VO_{2max} , BMI and %BF correlated moderately with the change in SBP after six sessions of HIIT ($r = 0.31$, -0.43 ; 0.30 and 0.47 , respectively), with only weak correlations between the SBP responses and risk factors after the first (acute) session of HIIT. This suggests that individuals with higher resting SBP, higher VO_{2max} , lower BMI and lower %BF values are more responsive to PEH. This is consistent with findings of Forjaz *et al.* (2000) who found younger subjects with higher initial BP, higher VO_{2max} and lower BMI to be more responsive to PEH. It is also reported that endothelium-dependent vasodilatation is directly correlated with VO_{2max} (Rinder *et al.*, 2000) and inversely correlated with age (Gerhard *et al.*, 1996) and obesity (Steinberg *et al.*, 1996). Thus, it could be suggested that an increased endothelium-dependent vasodilatory response may partly contribute to the observed PEH in less fit, overweight and obese young individuals.

Changes in DBP correlated weakly with the predictors of cardiovascular risk (VO_{2max} , BMI and %BF) after HIIT training, but moderately after the first (acute) session of HIIT ($r = -0.30$; 0.48 and 0.54 , respectively). Resting DBP also correlated poorly with the change in DBP after training. The reason for this poor correlation is not apparent, although it may be related to the relatively small effect of HIIT on DBP (2.6% and 4.3% change before and after HIIT respectively).

The results of this study were consistent with Liu *et al.* (2012) who found significantly greater PEH responses (SBP and DBP: 15.1 ± 1.7 mmHg and 9.8 ± 2.1 mmHg; $p < 0.05$) in

middle aged (52 ± 0.9 years) athletes who had high resting BP (SBP/DBP: $124 \pm 3.0/79 \pm 2.0$ mmHg) compared to their younger (28 ± 1.1 years) counterparts (SBP and DBP: 5.7 ± 2.5 mmHg and 4.0 ± 1.9 mmHg; $p < 0.05$) who had lower initial BP (SBP/DBP: $111 \pm 2.0/69 \pm 1.8$ mmHg). It is essential to note that changes in SBP are more important than changes in DBP in predicting future hypertension and all-cause mortality (Zanchetti and Waeber 2006; Kaplan, 2000). This matter is further discussed under the next heading.

2. Effect of HIIT on BP response after maximal exercise capacity test

A high SBP used to be regarded less dangerous in terms of future cardiovascular disease than high DBP, but now elevated SBP alone has gained recognition as an important cardiovascular risk factor (Kaplan, 2000). Kaplan (2000) contended that such effect can be attributed to the widening pulse pressure due to atherosclerotic stiffening of the aorta and large capacitance vessels. This invariably provides a smaller rigid reservoir raising the systolic inflow pressure and lowering diastolic pressures to a higher degree than occurs with more flexible vessels. Thus it has been reported that individuals with only high SBP have higher risks for cardiovascular disease than those with high DBP alone (Pescatello *et al.*, 2004a; Kannel, 1999). Moreover, SBP is known to increase all through adult life as age progresses exposing one to the risk of developing hypertension. This therefore explains reasons for the importance of the PEH response, as it provides an effective lowering of BP which will serve as a protective mechanism against cardiovascular risk and the development of cardiovascular disease.

The magnitude of PEH in terms of SBP (Figure 7.1c) following the maximal exercise capacity test in the present study was clinically significantly greater after the training intervention than pre-training (-5.04 mmHg vs. -4.58 mmHg), but two weeks detraining

caused the effect to disappear as the hypotensive response was reduced to 1.08 mmHg. DBP (Figure 7.2c) observed post-training was clinically significantly lowered by 4.3 mmHg which was greater compared to pre-training changes (-2.3 mmHg). This clinically significant effect (-3.5 mmHg) was sustained after detraining. The greatest change in MAP (Figure 7.3c) was detected after training (-5.17 mmHg), although the magnitude of change was smaller following detraining (-2.94 mmHg). However, this reduction still approached clinical significance.

Similar to the current study, Meredith *et al.* (1990) found BP reductions of 8 mmHg in SBP and 5 mmHg in DBP after four weeks of cycling exercise at 60 - 70% $\text{VO}_{2\text{max}}$ in normotensive male adults, however, after two weeks of detraining SBP returned to baseline values. Meredith *et al.* (1990) upon analysis of the hemodynamics indicated that TPR was significantly lower after training but increased after detraining to baseline values. This may suggest that TPR may have contributed to the reduction in BP after the exercise intervention, as the reverse occurred after detraining. Even though hemodynamic variables were not assessed in the current study, it is possible that the observed reduction in BP after training may have caused a decreased sympathetic vasoconstrictor nerve activity outflow to the vascular beds of the active skeletal muscles and then the arterial and baroreflex are reset to a lower BP than initial levels before exercise (Floras *et al.*, 1989). Moreover, Brandão Randon *et al.* (2002) highlighted that a reduction in BP was associated with a reduction in left ventricular end-diastolic volume and a consistent decrease in SV and HR. It is therefore possible that these hemodynamic functions may have reversed with detraining to increase BP again to baseline values.

Forjaz *et al.* (1998) reported significant, but similar reductions in BP after 45 min exercise performed at varying intensities of 30, 50 and 70% $\text{VO}_{2\text{max}}$ in young normotensive subjects

which were similar in magnitude. Since intensity of exercise plays a role in the hemodynamic, thermoregulatory and neural responses during exercise, differences in the magnitude of the PEH responses were expected. Rather, similar responses were observed in the varying intensities of exercise and it cannot be explained why the response observed after the higher intensity exercise (70% $\text{VO}_{2\text{max}}$) was not significantly higher than for intensities at 30 and 50% $\text{VO}_{2\text{max}}$. Perhaps the exercise at 70% $\text{VO}_{2\text{max}}$ relative to 30 and 50% $\text{VO}_{2\text{max}}$ was not intense enough to cause a significant difference in magnitude of PEH in normotensive individuals, as compared to the current study (90 - 95% $\text{VO}_{2\text{max}}$).

In contrast, Piepoli *et al.* (1994) found that maximal exercise, but not light exercise caused PEH in young normotensive individuals. These authors did not specify the exact intensity in terms of $\text{VO}_{2\text{max}}$ or HR_{max} but it is believed that the difference in intensity was wide enough such that the maximal exercise would be intense enough to cause a significant difference in PEH. Similarly, Hagberg *et al.* (1987) showed that the hypotensive response was greater after exercise at 70% $\text{VO}_{2\text{max}}$ than exercise performed at 50% $\text{VO}_{2\text{max}}$ in older, hypertensive subjects. It is clear that the difference in population of the two studies in terms of age and hypertension may have accounted for the difference in results, as older hypertensives have high resting BP. It is well known that the PEH response after any type of exercise is greater in hypertensive than normotensive (Kenney and Seals, 1993).

From the above, one can conclude that the magnitude of the PEH response is possibly related to the exercise intensity of the intervention, i.e. the higher the exercise intensity, the greater the PEH response. However, more studies should be conducted in at risk populations (hypertensive and overweight/obese) where the exercise intensity is carefully quantified before conclusive affirmations can be made.

The mean HR following the maximal exercise capacity test was not significantly different among test periods. However, the magnitude of change in HR was significantly reduced to a greater extent after post-training than pre-training and detraining. This was expected as HR increases during training but gradually declines to values below pre-training. Detraining, on the other hand, reversed the effect by increasing HR to similar pre-training values. Moreover, it is known that short-term detraining returns HR to initial levels before training (Wang *et al.*, 1997).

3. Effect of HIIT on BP response and HR

A clinically significant PEH was observed in BP following six HIIT sessions, but not after the acute session (Figures 7.5c, 7.6c and 7.7c). SBP was lowered by 5.4 mmHg following the six HIIT sessions which was almost twice greater than the response after the acute HIIT session (2.9 mmHg). However, it can be said that the latter approached clinical significance. DBP decreased after the acute HIIT session by 1.7 mmHg and 2.7 mmHg following the six sessions, but neither was considered clinically significant. Similarly, MAP lowered by 3.6 mmHg after six HIIT sessions, but the acute session of HIIT had no clinically significant effect (2.3 mmHg). This explains that the accumulated acute responses resulted in the greater magnitude in reduction following the six sessions.

The findings of this study is in line with Millen *et al.* (2013) who reported significant reductions of 8 mmHg in SBP and DBP among overweight or obese hypertensive middle-aged adults who performed six weeks of cycling exercise (4 x 4 min bouts at 80 - 90% VO_{2max} interspersed by 3 min active recovery at 50 – 60% VO_{2max}) 3 days per week. Apart from the difference in the length of the interventions (six versus two weeks), the other important difference is in the duration of the exercise bouts, i.e. long duration (4 x 4 min

bouts) versus short duration (10 x 1 min bouts). Both these factors may explain the difference in the magnitudes of PEH. Furthermore, there was also a difference in study populations (older vs. younger adults), as well as BP status (pre-hypertensive and hypertensive vs. normotensive). Likewise, Eicher *et al.* (2010) reported a greater reduction in SBP of 11.7 mmHg and 4.9 mmHg in DBP following interval exercise at 100% VO_{2max} in overweight/obese hypertensive individuals. The greater magnitude of the PEH response is likely attributed to the fact that their subjects were hypertensive and thus had higher resting BP.

The current study is consistent with other studies that utilized similar training methods and performed at similar intensity (Chan and Burns, 2013; Liu *et al.*, 2012; Ciolac *et al.*, 2010; Nybo *et al.*, 2010; Rossow *et al.*, 2010; Whyte *et al.*, 2010; Tjønnå *et al.*, 2009). It is therefore evident that the higher intensities of exercise induce greater magnitudes of PEH. Although the physiological mechanisms that mediate the PEH response was not the focus of this study, it is generally believed that PEH occurs as a result of a reduction in TPR, which is not completely offset by an increase in CO (Rossow *et al.*, 2010). Although this mechanism was described in response to other exercise modes, i.e. endurance and resistance exercise, it is unlikely that it would be different for HIIT.

A possible reason for the lack of PEH responses in previous studies may relate to the number of non-responders in the study populations (Pescatello *et al.*, 2007; Blanchard *et al.*, 2006), but unfortunately few studies specifically report on the number of responders and non-responders in their studies. Other methodological issues that should be considered are the small sample sizes of some studies ($n = 7$) and the use of auscultatory BP measurements which may not always be as accurate as ambulatory measurements.

The possible reasons for the positive results obtained in the current study are that the intensity was high enough to cause a clinically significant PEH response that could be sustained during an hour of recovery. The study subjects lived a sedentary lifestyle and as such this HIIT protocol was very intense. Furthermore, very short rest intervals were used (1 min each), which were possibly just enough to help individuals recover before the next bout, but that the volume was enough in total to cause a sufficient training stimulus. The positive results obtained from this study suggest that this type of HIIT protocol is a suitable and practical model for an unfit, overweight/obese population. Lastly, the fact that the majority of subjects responded with a positive PEH response shows that this protocol (10 - 15 bouts 1 min x 1 min) is effective in stimulating the necessary physiological adaptations.

The findings of this study and others with similar results suggests that both the intensity of exercise and the number of sessions per week are important training variables that should be considered in high intensity training programs, especially when interventions are conducted over short periods (i.e. two weeks). As low intensity exercise causes very small magnitudes of PEH, high intensity exercise (at maximal or near-maximal capacities) might cause sufficient hemodynamic, neural and/or hormonal responses which would result in clinically significant hypotensive responses after exercise.

During the HIIT intervention, the acute effect on HR was not different after completion of six sessions of HIIT even though a small practically significant difference was found, yet the magnitude of change was not significant. This was unexpected as it was believed that the magnitude of change in HR following six session of HIIT would be significantly greater than the acute session. The reason for this lack of response is not known. However, it is possible that the training period was too short and that it could not cause greater differences between the acute HIIT session and the six sessions of HIIT even though both sessions increased HR.

It is promising to note that, with longer periods of training (i.e. ≥ 4 weeks) the accumulated effects will be greater than for a single session (Ciolac *et al.*, 2011; Collier *et al.*, 2008).

D. CONCLUSION

To my knowledge, there has been no previous research on the effects of high intensity interval training and detraining on the magnitude of the PEH response in overweight and obese young women. The main finding of this study is that HIIT (both acute and six sessions of training) resulted in a sustained PEH response during 60 min seated recovery immediately after training. The acute session of HIIT caused hypotensive effects in SBP and DBP, although the latter was not clinically significant (< 3 mmHg). The accumulated acute effects following six sessions of HIIT in two weeks of training caused clinically significant reductions in resting SBP and DBP which was sustained over 60 min seated recovery.

Most studies investigating PEH utilized aerobic exercise at low to moderate intensity in hypertensive and non-hypertensive healthy populations, but not specifically in overweight and obese populations. However, overweight is known to be a risk factor to cardiovascular morbidity. Thus it is important that HIIT be encouraged due to its health-related benefits as well as the capacity to affect hypotensive effects. Also considering lack of time being the most frequent excuse for not exercising, HIIT interventions will go a long way to encourage individuals to exercise, as less time needs to be invested compared to traditional endurance training. Moreover, HIIT has been shown to elicit similar or better physiological adaptations in comparison with longer duration (i.e. ≥ 30 min) traditional endurance training.

The two weeks of detraining reversed the hypotensive response that was induced by two weeks of HIIT sessions such that SBP returned near to resting values. However DBP was not

affected by detraining as it remained consistently lower and the magnitude remained clinically significant. Therefore it is important that regular exercise training be encouraged so that acquired benefits would be sustained.

E. LIMITATIONS AND FUTURE STUDIES

The study limitation was that BP measurements after detraining was only taken after the maximal exercise capacity test but not before, which would have reflected the direct response right away following detraining without any additional exercise (i.e. the maximal exercise capacity can be considered an extra exercise session). For this reason, the effect of detraining on PEH was only compared following the maximal exercise capacity tests. Thus in future research, BP monitoring can be done on a separate day right after the detraining period to better compare the detraining and the training effects.

As in most previous HIIT studies, BP was monitored for only 60 min in this study. Only Whyte *et al.* (2010) monitored BP for 24 hours post-exercise. Therefore future studies should consider monitoring BP after HIIT for 24 hours or more in order to confirm that this physiological response is sustained throughout the day or even beyond.

Moreover, there are limited studies on the hemodynamic changes following HIIT. Thus future studies should take this into consideration, so that the mechanisms responsible for PEH after HIIT can be described. On the whole there is sufficient literature that examined the metabolic adaptations to and performance effects of HIIT, but comparatively few studies have been done on HIIT and PEH. Thus research in this area is of great importance, especially in populations at risk for metabolic and cardiovascular diseases.

Another limitation was that the subjects' diets and pre-exercise meals were not controlled. It is unlikely that this would have affected the BP data, but it could have influenced the anthropometric data. Nevertheless, the main purpose of this study was to investigate the BP response to HIIT and not changes in body composition.

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APPENDIX A

PAR-Q & YOU

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

(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?

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

Comment: _____

SIGNS OR SYMPTOMS SUGGESTIVE OF CARDIOVASCULAR AND PULMONARY DISEASE

Have you experienced any of the following?

- Yes No Pain/discomfort in your chest, jaw, or arms _____
- Yes No Shortness of breath at rest or mild exertion _____
- Yes No Dizziness or fainting spells _____
- Yes No Difficulty breathing while lying down _____
- Yes No Swelling of your ankles _____
- Yes No Skipped heartbeats or racing heartbeat _____
- Yes No Occasional leg pain, especially while walking _____
- Yes No Heart murmur _____
- Yes 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 ≤ 45 y, female ≤ 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 ≤ 45 y, female ≤ 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% VO₂max) maximal exercise intensity. ACSM recommends anyone in the moderate risk category get medical clearance before vigorous exercise. Lower intensity exercise (< 60% VO₂max) 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 training on post-exercise hypertensive response

You are asked to participate in a research study conducted by Biggie Bonsu, Masters Student, from the Department of Sport Science at Stellenbosch University. This research is part of my Masters in Sport Science thesis. You were selected as a possible participant in this study because I am young woman between the age of 18 and 25.

1. PURPOSE OF THE STUDY

The purpose of the study is to investigate whether high-intensity interval training (HIIT) cause post-exercise hypotension in young overweight / obese women.

2. PROCEDURES

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

During the first session, you will undergo a screening process to determine your inclusion by taking anthropometric measurements as well as blood pressure. You will also be asked to complete the Physical Activity Rating Questionnaire to assess your physical activity level and then complete a maximal exercise test on the treadmill to determine your VO_{2max} .

EXERCISE PROTOCOL

During this session, you will undergo training that will consist six sessions of one minutes all “all-out” efforts of running on the Saturn treadmill. This will commence with a 5 minutes warm-up at velocity corresponding to 50% HR_{max} followed by 10 x 1 minutes bouts at running velocity corresponding to 90 - 95% HR_{max} . The high intensity intervals will be separated by one minute active recovery at a running velocity of 70% HR_{max} for the first three training sessions (first week of training). The last three training sessions (second week of training) will comprise 15 x 1 minute bouts session at 90 - 95% HR_{max} separated by the one minute active recovery at 70% HR_{max} . This will be followed by another 5 minutes of cool-down at a running velocity corresponding to 50% HR_{max} .

POST EXERCISE MONITORING

This will be recorded after the last session of HIIT and will include resting blood pressure, VO_{2max} , and body composition similar to base line measurements.

DETRAINING

Participants will be asked to stop the HIIT training for two weeks and then return to the laboratory for measurements similar to base line measurements.

All testing will be done in the Sport Physiology laboratory at Department of Sport Science, Stellenbosch University.

3. POTENTIAL RISKS AND DISCOMFORTS

The study does not carry any serious risk for you as a participant. You may experience discomfort with the training on the treadmill. However, this will be not more than the usual discomfort you feel while training.

4. POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY

Participants will exercise as training will lead to fitness; however serve as anti-hypertensive effect from risk factors.

The results of this research will contribute to science as it will give us a better understanding of the magnitude of the post-exercise hypotensive response after bouts of HIIT.

5. PAYMENT FOR PARTICIPATION

Subjects will not receive any payment for participation in the study.

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 assigning each participant a code. This code will be used rather than the name of the participant. Data will be kept on personal computer that is pass word protected and will only be accessible to the researcher.

Confidentiality with the publication of results will be kept by not publishing the raw data as well as making use of codes assigned to participants.

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. Participants can withdraw any time they cannot continue because of illness.

8. IDENTIFICATION OF INVESTIGATORS

If you have any questions or concerns about the research, please feel free to contact

Biggie Bonsu
Phone: 021 808 4915
Email: 16198492@sun.ac.za

Prof E Terblanche
Phone: 021 808 2742
Email: 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 participant by Biggie Bonsu in English and in command of this language or it was satisfactorily translated to her. The participant was given the opportunity to ask questions and these questions were answered to her satisfaction.

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

Name of Subject/Participant

Name of Legal Representative (if applicable)

Signature of Subject/Participant or Legal Representative

Date

SIGNATURE OF INVESTIGATOR

I declare that I explained the information given in this document to _____
[*name of the subject/participant*] and/or her] representative _____ [*name of the representative*]. She was encouraged and given ample time to ask me any questions. This conversation was conducted in English and no translator was used.

Signature of Investigator

Date

APPENDIX D**DATA SHEET**

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

1	Blood Pressure (BP)			
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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	

14	Resting Glucose	
15	Resting Cholesterol	
16	VO ₂ max	
17	Speed(km/hr)	
18	Gradient %	
19	Time to exhaustion	
20	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	