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# Acute physiological effects and perceived enjoyment of a single bout of aerobic interval and continuous moderate-intensity cycling in overweight and obese individuals

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**EDITH COWAN UNIVERSITY**

**School of Exercise and Health Sciences**

**Acute physiological effects and  
perceived enjoyment of a single bout  
of aerobic interval and continuous  
moderate-intensity cycling in  
overweight and obese individuals**

**By**

**Koh Fujie, BSc.**

This thesis is presented for the award of Master of Science (Sports Science) from the  
School of Exercise and Health Sciences; Faculty of Health, Engineering and Science;

Edith Cowan University, Western Australia

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**Date of Submission: 30/03/2014**



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## ACKNOWLEDGMENTS

The completion of this thesis marks the end of an eventful period in my life. Throughout this academic journey, I have experienced a myriad of emotional states. On several occasions, I have even questioned my own capabilities and self worth. It is during these tenuous times that I realised how friendship is important. Importantly, I wouldn't have managed to get to where I am without the constant support and advice of my supervisors, seniors and peers. Therefore, this section is very much dedicated to these people, in no particular order of importance.

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## ABSTRACT

Aerobic interval training, a form of high intensity interval training, is commonly prescribed to both the general and clinical populations. However, the acute physiological effects from a single bout of aerobic interval session are not fully understood. In training studies, these acute physiological effects may confound actual training adaptations when they occur following the final training session [1]. Furthermore, while recreationally active men perceived aerobic interval training to be more enjoyable than continuous moderate-intensity exercise [2], the preference of overweight and obese individuals has not been extensively researched. Since overweight and obese individuals tend to have lower exercise tolerance, it is possible that their perceived enjoyment may differ to recreationally active participants. Thus, the aim of this study was to examine the physiological effects and perceived enjoyment of an acute bout of aerobic interval (AI) and a continuous moderate-intensity (CME) in an overweight and obese population.

In a randomised and counterbalanced order, eight overweight/obese (waist:  $103 \pm 10$  cm) participants performed bouts of CME (40 min at 50% of peak power output (PPO)) and AI cycling (13 x 1 min at 85% of PPO: 13 x 2 min at 30% of PPO). CME and AI were matched for duration and total work performed. Salivary cortisol was measured before, 10 and 30 min post exercise and analysed using standard enzyme-linked immunosorbent assay kits. Blood pressure (BP), blood metabolites (glucose and lipid), insulin and resting metabolism were measured at baseline, post 24, 48 and 72 h exercise trials in a fasted and rested state. Peripheral BP was measured in duplicate using a manual sphygmomanometer and central BP and its associated hemodynamic parameters were derived from the radial pulse wave using a sphygmocardiogram (SphygmoCor). Resting metabolic profile was determined from gas exchange (TrueOne® gas analyser) measured in a supine and rested state over 40 min. Anthropometric (waist and hip circumferences and body composition) measurements were taken at baseline and at post 72 h after the last exercise trial. Participants completed the Physical Activity Enjoyment Scale (PACES) at the end of each trial.

Salivary cortisol expressed as a percentage of baseline level increased in both trials and was significantly (mean  $\pm$  SD)  $189 \pm 35\%$  ( $p < 0.05$ ) higher at 30 min post exercise. Resting brachial diastolic BP was lower in the AI trial compared with CME trial ( $82 \pm 5$  vs.  $84 \pm 5$

mmHg respectively,  $p < 0.05$ ). Resting brachial diastolic BP was lower than baseline ( $86 \pm 12$  mmHg) at 24 h ( $83 \pm 5$  mmHg,  $p < 0.05$ ) and at 72 h ( $82 \pm 5$  mmHg,  $p < 0.05$ ) post exercise, however, there was no significant interaction between exercise trials and time points ( $p = 0.07$ ). Resting brachial mean arterial pressure was lower than baseline ( $102 \pm 14$  mmHg) at 24 h ( $99 \pm 6$  mmHg,  $p < 0.05$ ), 48 h ( $98 \pm 6$  mmHg,  $p < 0.05$ ) and 72 h ( $98 \pm 6$  mmHg,  $p < 0.05$ ) post exercise. Resting brachial mean arterial pressure was also lower in the AI trial compared with CME trial ( $98 \pm 6$  vs.  $100 \pm 5$  mmHg respectively,  $p < 0.05$ ), however, there was no significant interaction between exercise trials and time points ( $p = 0.07$ ). Derived resting aortic diastolic BP was lower in the AI trial, compared with the CME trial ( $83 \pm 5$  vs.  $85 \pm 5$  mmHg respectively,  $p < 0.05$ ). Compared with baseline ( $87 \pm 4$  mmHg), derived resting aortic diastolic BP was lower at 24 h ( $83 \pm 5$  mmHg,  $p < 0.05$ ) and 72 h ( $82 \pm 5$  mmHg,  $p < 0.05$ ) post exercise. No significant interaction effects were observed for derived resting central diastolic BP ( $p = 0.10$ ). Derived resting mean aortic pressure was lower in the AI trial, compared with the CME trial ( $98 \pm 6$  mmHg vs.  $100 \pm 5$  mmHg, respectively,  $p < 0.05$ ). Compared with baseline ( $102 \pm 5$  mmHg), derived resting mean aortic pressure was lower at 24 h ( $99 \pm 6$  mmHg,  $p < 0.05$ ), 48 h ( $98 \pm 6$  mmHg,  $p < 0.05$ ) and 72 h ( $98 \pm 6$  mmHg,  $p < 0.05$ ) post exercise. There was a trend towards interaction effects for resting derived mean aortic BP, however significance was not reached ( $p = 0.063$ ). Resting carbohydrate oxidation rate was higher than baseline ( $0.10 \pm 0.06$  g/min) at 72 h post exercise ( $0.16 \pm 0.02$  g/min), with no significant difference observed between trials. PACES scores were significantly higher for the AI than the CME trial ( $109 \pm 13$  vs.  $96 \pm 10$ ).

The main finding from this study is that a single AI and CME session can elicit acute physiological effects that last up to 72 h, with no significant difference observed between conditions. Significant decrements in diastolic and mean arterial pressures were observed up to 72 h and resting carbohydrate oxidation rate was significantly higher 72 h following exercise. Therefore, training studies should look at scheduling post study measurement sessions more than 72 h after the last exercise session bout to avoid confounding acute physiological effects with training adaptations. Finally, the study has also demonstrated that overweight/obese males enjoy the AI session more than the CME session.

# TABLE OF CONTENTS

Declaration .....	i
Copyright and Access Statement .....	ii
Acknowledgments.....	iii
Abstract .....	v
Table of Contents .....	vii
List of Tables .....	ix
List of Figures .....	x
CHAPTER ONE Introduction.....	1
1.1 Purpose.....	3
1.2 Research Questions .....	4
1.3 Abbreviations of Selected Terms.....	5
CHAPTER TWO Literature Review.....	6
2.1 Physical Inactivity and its Implications .....	6
2.2 The Undefined “Optimal” Exercise Dose.....	6
2.3 High-intensity Interval Training (HIIT).....	8
2.4 Metabolic adaptations from HIIT in both healthy and clinical populations .....	10
2.5 Cardiometabolic benefits from high-intensity interval training in both healthy and clinical populations .....	12
2.5.1 Aerobic capacity and exercise tolerance .....	12
2.5.2 Cardiovascular adaptations and improvements in hemodynamic parameters .....	13
2.5.3 Anthropometric and body composition .....	15
2.5.4 Glucose homeostasis .....	16
2.5.5 Lipid metabolites .....	17
2.6 Comparisons of metabolic adaptations and health benefits between HIIT and conventional CME protocols .....	18
2.6.1 Intramuscular metabolic and ion handling adaptations .....	18
2.6.2 Cardiovascular adaptations and improvements in hemodynamic parameters .....	20
2.6.3 Metabolic risk factors .....	22
2.7 Gaps in the research .....	24
2.7.1 Comparison of acute responses to a single high-intensity interval session versus a single continuous moderate-intensity session .....	25

2.7.2	Comparison of acute cortisol response immediately after a single session of AI versus CME.....	28
2.7.3	Comparison of exercise preference between HIIT versus CME .....	30
2.7.4	Conclusion: Addressing the gaps.....	31
CHAPTER THREE Methods.....		34
3.1	Subjects .....	34
3.2	Study design.....	34
3.2.1	Incremental exercise test.....	36
3.2.2	Exercise trials.....	36
3.2.3	Measurements .....	37
3.3	Statistical analysis .....	41
CHAPTER FOUR Results .....		42
4.1	Exercise responses during trials.....	42
4.2	Anthropometric measurements .....	45
4.3	Blood metabolites and insulin resistance .....	45
4.4	Salivary Cortisol .....	45
4.5	Resting hemodynamic parameters .....	47
4.6	Resting metabolism and substrate utilisation.....	49
4.7	PACES scores .....	50
CHAPTER FIVE Discussion .....		50
5.1	Study objectives and primary findings .....	51
5.2	Hemodynamic parameters .....	52
5.3	Blood metabolites, insulin, HOMA2-IR and resting metabolism.....	54
5.4	Cortisol response.....	57
5.5	Perceived “enjoyment” of the exercise trials .....	58
5.6	Limitations .....	59
5.7	Practical implications.....	60
CHAPTER SIX References.....		61
CHAPTER SEVEN Appendices.....		75

## LIST OF TABLES

Table 1. Demographics of participants .....	42
Table 2. Blood metabolites and insulin, hemodynamic parameters and resting metabolism measured at baseline, post 24, 48 and 72 h after both aerobic interval (AI) and continuous moderate-intensity (CME) exercise trials. ....	44

## LIST OF FIGURES

Figure 1. Schematic illustration of the study design.....	35
Figure 2. Whole body rating of perceived exertion (RPE; A) and heart rate (HR; B) measured during bouts of continuous moderate-intensity (CME) and aerobic interval (AI) exercise. ....	43
Figure 3. Absolute salivary cortisol (A) and salivary cortisol presented as percentage change from baseline (B) measured at post 10 and post 30 min after bouts of aerobic interval, (AI) and continuous moderate-intensity (CME) exercise.....	46
Figure 4. Resting brachial diastolic blood pressure (DBP; A), resting brachial mean arterial pressure (MAP; B), derived resting aortic diastolic BP (CDBP; C), derived resting mean aortic pressure (CMAP; D) measured at baseline, 24 h, 48 h and 72 h post exercise after bouts of aerobic interval, (AI) and continuous moderate-intensity (CME) exercise... ..	48
Figure 5. Fasted state resting CHO oxidation rate ( $M_{CHO}$ ) measured at baseline, 24 h, 48 h and 72 h post exercise after bouts of aerobic interval, (AI) and continuous moderate-intensity (CME) exercise. ....	49
Figure 6. The physical activity enjoyment scale (PACES) score awarded by participants immediately after bouts of bouts of aerobic interval, (AI) and continuous moderate-intensity (CME) exercise. ....	50

## **CHAPTER ONE                      INTRODUCTION**

In developed countries with a high national income and aging population, physical inactivity is established as one of the major risk of mortality [3, 4]. Furthermore, its detrimental effect is not isolated, as physical inactivity increases the risk of other mortality factors, such as high blood pressure, elevated blood glucose and obesity, all of which are associated with many chronic diseases such as cardiovascular diseases and cancer [4-10]. The incidence of physical inactivity also has significant negative social and economic impacts. In Australia, the total direct cost attributable to obesity, as defined by both waist circumference and body mass index (BMI), amounted to \$21 billion in 2005 [11]. Since prevalence of obesity in Australia is projected to increase by 65% in 2025, obesity attributed cost will likely to increase in tandem [12]. However, physical activity can lead to a 32% reduction in mortality [5] and a 2.5% reduction in global physical inactivity can avert an estimated 1.3 million deaths annually [13]. Collectively these data clearly show that physical activity is essential in maintaining general health and reducing mortality risk, which translates to reduction in health costs associated with ageing. Indeed, exercise has been established as one of the most cost-effective strategies for the prevention and management of several chronic diseases, including diabetes, cardiovascular disease (CVD) and some cancers.

Many international and national agencies have outlined exercise prescription guidelines to encourage healthy active living [14-17]. While these guidelines vary slightly between countries, they generally convey the analogous message of encouraging individuals to engage in regular moderate-intensity physical activity between 30 to 60 min on most if not all days [18]. However, these guidelines are often voluminous and difficult for some individuals to commit to. Supporting this, the most commonly cited barrier against exercise engagement and adherence is a lack of time [19-22]. There has recently been a paradigm shift in exercise recommendation from the sole emphasis on exercise volume towards the additional consideration of exercise intensity. Indeed, the increasing body of research highlighting the benefits of vigorous intensity physical activity [23-25] has prompted the American College of Sports Medicine (ACSM) and

American Heart Association (AHA) to advocate it explicitly in their physical activity guidelines [14].

High-intensity interval training (HIIT) allows participants to perform cumulative vigorous bouts of exercise and is characterised by intermittent periods of work and rest. The contribution from anaerobic and aerobic energy systems vary with the duration and number of work bouts performed and the recovery between bouts. For the purpose of this thesis, HIIT protocols will be broadly classified into sprint (i.e. 10 – 30 sec “all-out” maximal work) interval (SIT) and aerobic (i.e. 80% - 100%  $\dot{V}O_{2max}$ ) interval (AIT) training protocols. While HIIT may result in rapid favourable adaptations, the high-intensity nature of HIIT protocols especially those involving “all-out” sprints may be unsafe, impractical and thus unsuitable for many untrained, unhealthy or ‘at risk’ individuals [26]. Indeed, poor cardiovascular health of overweight and obese individuals may increase the risk of acute cardiovascular accidents during HIIT in these individuals [27-30]. Despite this, HIIT, especially AIT has been previously prescribed to various clinical populations with reports of favourable skeletal, cardiac and endothelial adaptations, resulting in improvements in blood lipid profile and blood glucose, aerobic capacity and exercise performance [1, 31-35].

These favourable adaptations can occur after as little as two weeks of HIIT (i.e. either AIT or SIT) [1, 26, 36]. Although training adaptations resulting from long term adoption of AIT have been extensively studied, our understanding of the acute physiological responses to a single session of AIT is limited. The acute physiological responses following a training intervention have previously been termed ‘last exercise bout’ effects [37]. Understanding these responses is important as they can confound actual training adaptations, especially following short term intervention studies [1]. Furthermore, prolonged physiological effects after an acute exercise intervention may convey transient favourable health benefits which can be accumulative if the exercise modality is performed over multiple sessions [37]. As such, detailed understanding of the acute physiological effects and stress induced by AIT may aid in improving exercise program prescription in athletic, general and clinical populations.



Cortisol is an adaptive endocrinal response towards both physical and mental stress [38]. It is responsible for substrate mobilisation during increased metabolic demands [39] and partly explains stress-induced metabolic and physiological adaptations [40, 41]. Both exercise duration and intensity significantly influence cortisol secretion during and following exercise [42-44]. In comparison to lean individuals, overweight and obese participants have previously been reported to have an elevated cortisol response following exercise [45]. However, we are currently unaware of any studies that have examined post exercise cortisol response after a single aerobic interval (AI) session in overweight and obese individuals. These individuals are at greater risk of developing metabolic diseases; therefore cognizance of any favourable acute physiological responses to a single bout of AI session would help further contribute to better exercise prescription. Exercise intensity plays a crucial role in exercise adherence [46]. Indeed, it has previously been found that the prescription of exercise at an intensity greater than the preferred intensity diminish enjoyment of exercise in overweight individuals [47]. Hence, while AIT may be a potential alternative to the conventional continuous moderate-intensity exercise (CME), it is possible that such exercise may be less preferred by certain clinical populations. Conversely, prospective findings from Bartlett et al. (2011) show that recreationally active men perceived AIT to be more enjoyable than CME, indicating that it may be an ideal exercise model that encourages exercising at higher intensities without compromising on perceived enjoyment [2]. However, Sim, Wallman, Fairchild and Guelfi (2014) reported that overweight individuals perceived similar enjoyment from both AI and CME [48]. This indicates that perceived enjoyment of AIT differs between recreationally active and overweight individuals and is diminished in the latter. Therefore, further research is needed in order to better understand the acute physiological responses to AIT and the perceived enjoyment of such exercise in overweight individuals.

## **1.1 Purpose**

The primary objective of this thesis was to examine and compare the influence single bouts of aerobic interval and continuous moderate-intensity exercise have on

fasting blood lipid and glucose profiles, insulin, resting central and peripheral blood pressures, resting metabolism (i.e. metabolic rate, energy expenditure and lipid/carbohydrate oxidation rates) and measurements of body composition (i.e. lean, fat and bone tissue masses) over a 72 h post exercise period in overweight and obese individuals. A secondary purpose of this study was to compare the post exercise (i.e. 10 and 30 min) salivary cortisol response between aerobic interval and continuous moderate-intensity cycling performed by overweight and obese individuals. The final objective of the study was to determine and compare the perceived enjoyment of single bouts of aerobic interval and continuous moderate-intensity cycling in overweight and obese individuals.

## **1.2 Research Questions**

- i. What influence does single bouts of aerobic interval and continuous moderate-intensity cycling have on fasting blood lipid, glucose and insulin profiles, resting central and peripheral blood pressures and its associated parameters and resting metabolism in overweight and obese individuals in the 24 h, 48 h, and 72 h following exercise.
- ii. Is there a difference in post-exercise (i.e. 24 h, 48 h, and 72 h) fasting blood lipid, glucose and insulin profiles, resting central and peripheral blood pressures and its associated parameters and resting metabolism following aerobic interval and continuous moderate-intensity cycling in overweight and obese individuals.
- iii. Is there a difference in post-exercise salivary cortisol response between single bouts of aerobic interval and continuous moderate-intensity exercise in overweight and obese individuals?
- iv. Is there a difference in perceived enjoyment between single bouts of aerobic interval and continuous moderate-intensity exercise in overweight and obese individuals?

### 1.3 Abbreviations of Selected Terms

AIT	aerobic interval training
AI	aerobic interval session
BMI	body mass index
BPM	beats per min
CDBP	derived aortic diastolic blood pressure
CMAP	derived mean aortic pressure
CME	continuous moderate exercise
CSBP	derived aortic systolic blood pressure
DBP	diastolic blood pressure (brachial)
DEXA	dual-energy X-ray absorptiometry
HDL	high density lipoprotein
HIIT	high-intensity interval training
HOMA	homeostatic model assessment
HR	heart rate
LDL	low density lipoprotein
MAP	mean arterial pressure (brachial)
$M_{CHO}$	carbohydrate oxidation rate
$M_{lipid}$	lipid oxidation rate
OGTT	oral glucose tolerance test
PACES	physical activity enjoyment scale
PAR-Q	physical activity readiness questionnaire
PPO	peak power output
REE	resting energy expenditure
RER	respiratory exchange ratio
RMR	resting metabolic rate
RPE	rating of whole body perceived exertion
SBP	systolic blood pressure (brachial)
SI	sprint interval session
SIT	sprint interval training
$\dot{V}O_{2peak}/\dot{V}O_{2max}$	peak/maximal oxygen consumption

## **CHAPTER TWO      LITERATURE REVIEW**

### **2.1      Physical Inactivity and its Implications**

In 2004, physical inactivity was identified as the fourth leading risk factor of mortality, contributing to 6% in global mortality [4]. However, physical inactivity does not elicit an isolated effect but also modifies other metabolic risks factors such as increased blood glucose, elevated blood pressure and obesity [10]. These in turn increase the risk of many chronic diseases, such as coronary heart disease, type 2 diabetes and various cancers [5-9]. The increased prevalence of physical inactivity has been largely attributed to rapid urbanisation, industrial mechanisation and use of mechanised transport [3, 49]. In 2012, 31.1% of adults globally were physically inactive with prevalence greatest in countries with a high average income and an aging population [3]. Physical inactivity not only negatively influences health but also has significant social and economic impacts. For example, in Canada, physical inactivity costed the economy \$6.8 billion in 2009, which equates to about ~3.7% of the total health care cost [50]. In Australia, health care cost of sedentary middle-aged women was reported to be 26.3% greater than those who were moderately active [51]. This is a pressing issue as a recent population study revealed that more than half of Australians are either sedentary or reported low level of physical activity [52]. Fortunately, physical inactivity is a modifiable behavioural risk factor. On a global scale, a reduction in physical inactivity by 2.5% can avert an estimated 1.3 million deaths every year [5]. For an individual, becoming physically active can confer a 32% reduction in risk of all-caused mortality [13]. Clearly, increasing the level of physical activity of the general population is therefore pertinent in improving a variety of health, economic and social outcomes.

### **2.2      The Undefined “Optimal” Exercise Dose**

To address the increasing prevalence of physical inactivity, several international and national agencies have developed guidelines to encourage physical activity. Most of these conventional guidelines endorse the daily engagement in moderate-intensity

physical activity for a minimum duration, varying between 30-60 min [18]. The rationale behind these guidelines was based on findings that demonstrated a clear association between accumulated total or weekly caloric expenditure and cardiovascular mortality [18, 19]. However, there are studies in the literature suggesting that exercise volumes lower than the conventional minimum recommendation may be sufficient to elicit a reduction in health risks [53-55]. For example, Wen et al. (2011) suggested that health benefits may be accrued with as little as 15 min of moderate-intensity exercise [56]. Indeed, the volume, frequency and intensity of physical activity may all significantly influence the adaptations and consequential health benefits of exercise, with the best possible permutations of these factors yet to be clearly established [55, 57]. Therefore, while previous research has clearly presented a strong dose-response relationship between physical activity and health benefits, the specifics of this relationship remains largely undefined [13]. On the premise that the total energy expended through physical activity is partly mediated by exercise intensity, it has been suggested that exercise intensity plays an influential role in the dose-response relationship between physical activity and health benefits. Regular participation in vigorous intensity physical activity and the associated greater energy expenditure has been associated with decreased mortality and cardiovascular related morbidity and a healthier state of being when compared with lower intensity exercise [25, 58, 59]. Consequentially, there has been a paradigm shift in exercise recommendations with added emphasis on exercise intensity apart from total exercise volume [23-25, 60]. Such a change in consensus was reflected in the update to the American College of Sports Medicine (ACSM) and American Heart Association (AHA) physical activity guidelines in 2007 which explicitly included recommendations on the participation of vigorous-intensity physical activity [14].

Independent of the volume (i.e. duration) spent engaged in the physical activity; there is also evidence to suggest that exercise intensity can confer a reduction in risk of chronic diseases. For instance, it has been reported that high-intensity physical activity of  $\geq 6$  metabolic equivalents (METs), independent of exercise volume, was associated with lower relative risk of coronary heart disease (CHD) compared to both moderate-intensity (4 – 6 METs) and lower-intensity (1 – 4 METs) exercise [59]. Within this study

every increment of 1 MET was accompanied by a 4% reduction in CHD risk [59]. This research is supported by isocaloric training studies, which have found that high-intensity exercise (i.e. 70 – 80 %  $\dot{V}O_{2peak}$ ) training results in greater improvements in cardiovascular fitness, glucose tolerance, blood lipid profile and blood pressure when compared with moderate-intensity exercise (i.e. 50 – 60 %  $\dot{V}O_{2peak}$ ) training [61, 62]. As such, the observed benefits are not simply the result of an increase in total training load (i.e. volume and intensity) but instead may be caused by the high-exercise intensities per se. These observations highlight that individuals can maintain a healthy state of being with lower time commitment by regular participation of vigorous-intensity physical activity. This is important since ‘lack of time’ is a commonly cited barrier against exercise adherence and participation in physical activity [19-22].

Studies such as that published by Talanian, Galloway, Heigenhauser, Bonen and Spriet (2007) which reported training adaptations comparable to those observed in longer duration endurance type training studies from just 2 weeks of aerobic interval training, prompted further research on high-intensity interval exercise modality as a time-efficient alternative against the traditionally recommended continuous moderate-intensity exercise modality [36]. In fact, studies comparing between both these modalities have reported greater if not comparable favourable metabolic adaptations after high-intensity interval training despite having controlled for or a marked difference in total exercise volume [33, 63-65]. Therefore, the following sections of this chapter will review the current body of literature relating to the possible benefits of high-intensity exercise. This literature will be discussed in comparison with traditional continuous moderate-intensity training and within the context of its application in both the general and clinical populations.

### **2.3 High-intensity Interval Training (HIIT)**

High-intensity interval training (HIIT) is an exercise modality which is characterised by repetitions of short high-intensity work interspersed with bouts of low-intensity exercise or complete rest. Due to the intermittent nature of most sports and the ability of HIIT to rapidly improve performance this modality has been commonly

utilised in athletic populations [66-69]. Numerous factors may be altered within HIIT in order to vary the exercise stimulus and adaptation, including exercise and recovery modality, exercise intensity and work-rest ratio duration. As a result, considerable possible combinations exist for HIIT. Therefore, for the purpose of this literature review, the discussion on HIIT will be limited to sprint interval training (SIT) and aerobic interval training (AIT). The protocol for a typical SIT session usually involves 4 - 6 sets of 10 - 30 seconds “all-out” sprints, interspersed with 4 - 4.5 min of complete rest or low-intensity recovery [1, 70-73]. In contrast, AIT is typically characterised by longer periods (i.e. 1 - 4 min) of high-intensity exercise (i.e. 80% - 100% of  $\dot{V}O_{2max}$ ,  $HR_{max}$  or HRR) interposed with similar periods (e.g. 1 - 4 min) of rest, performed over 4 or more sets [70, 74]. The contrast between a SIT and AIT is defined by the difference in the contribution from each energy system during exercise. The short supra-maximal work intervals during SIT results in a high anaerobic energy demand, rapidly impairing phosphocreatine and anaerobic glycolytic pathways and aerobic energy demand increases with the increasing number of intervals [68, 75, 76]. However, the lower exercise intensity (i.e. at or near maximal aerobic power or velocity at  $\dot{V}O_{2max}$ ) and longer interval duration of the AIT results in considerable contribution from aerobic system throughout each effort [66, 77]. Due to the high-intensity and very short duration of SIT many of the observed adaptations have been observed in the periphery (i.e. muscle). Indeed, SIT has been shown to elicit an upregulation of key intramuscular regulatory enzymes of all three energy systems (i.e. creatine kinase, phosphofuctokinase and citrate synthase), enhancing oxygen utilisation [66, 78]. Furthermore, SIT can also elicit muscular morphological changes such as muscular hypertrophy, a bidirectional shift in muscle fibre towards type IIa and improve muscle contractile characteristics via an increase in the volume of the sarcoplasmic reticulum [78]. Conversely, AIT has been shown to result in significant central (i.e. cardiac output) and peripheral adaptations [73, 79, 80]. For instance, improvement in aerobic capacity in tandem with an increase in left ventricle end diastolic volume and the resultant stroke volume was observed after 13 weeks of AIT in obese adolescents [79] and enhancement in key intramuscular regulatory enzymes associated with anaerobic glycolytic and aerobic energy systems was observed after 6 weeks of AIT in recreationally active individuals [81]. The premise

for the prescription of HIIT, in untrained and clinical populations lies within the work-rest interval bouts which exposes individuals to intermittent bouts of high cellular stress over extended periods, thereby allowing for greater adaptations than would be possible with steady state exercise [70, 82]. Importantly, HIIT has been safely prescribed to a number of clinical populations (i.e. individuals with type two diabetes, metabolic syndrome, post-myocardial infarction), despite the concern for an increased risk of acute cardiovascular accidents [1, 30-35, 83-86]. Therefore, the following sections of the review examine the literature pertaining to the intramuscular, metabolic and health benefits of HIIT in order to illustrate how HIIT can be used as an alternative exercise intervention to the traditional continuous moderate-intensity modality.

## **2.4 Metabolic adaptations from HIIT in both healthy and clinical populations**

Skeletal muscle fibres are extremely adaptable to physiological stress. Neurological, morphological and metabolic adaptations can occur in response to exercise training resulting in enhanced skeletal muscle work capacity [78, 87]. In this section, we will limit the discussion to metabolic adaptations occurring in response to HIIT. Observed intramuscular metabolic adaptations in response to HIIT includes an increase in the protein content and activity of key regulatory metabolic enzymes, increase in intramuscular substrate availability and its delivery and lastly, improvement in its buffering capacity [66, 78].

Peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1 $\alpha$ ) is considered a 'key regulator' of several transcriptional factors that are associated with adaptive thermogenesis, fibre type switching in skeletal muscle, mitochondrial biogenesis, cellular metabolism and heart development [88]. The expression of PGC-1 $\alpha$  after exercise is activated through several upstream signalling pathways including Ca<sup>2+</sup>/calmodulin-dependent protein kinase IV (CaMKIV) and calcineurin A (CnA) (i.e. calcium-mediated signalling via nerve stimulation) [89, 90], p38 mitogen-activated protein kinase (p38 MAPK) [91] and AMP-activated protein kinase (AMPK) (i.e. when AMP:ATP ratio is high) [92]. Numerous studies have shown that PGC-1 $\alpha$ , along with



downstream markers of mitochondrial biogenesis and both lipid and glucose oxidation, may be upregulated following AIT and SIT [26, 32, 33, 65, 93, 94]. For instance, a brief (i.e. 1 week, 3 sessions per week) period of SIT resulted in a significant increase in the protein content of the mitochondrial enzyme cytochrome c oxidase subunit IV (COX IV) and the cellular glucose transporter GLUT IV, which will improve cellular glucose utilisation and oxidative capacity [95]. However, within this study no training effect was observed in lipid transporters (i.e. fatty acid translocase and fatty acid binding protein) [95]. Furthermore, 6 to 7 weeks of SIT can increase hexokinase (HK), phosphofructokinase (PFK), citrate synthase (CS) [96], pyruvate dehydrogenase (PDH) and  $\beta$ -hydroxyacyl-CoA-dehydrogenase ( $\beta$ -HAD) [65], which will improve both glucose and lipid oxidative capacities. Alternatively, 2 weeks of AIT in healthy individuals has been shown to upregulate the enzymatic activity of CS [26, 36] and elicit an increase in the protein content of COXII, COXIV, GLUT IV [26] as well as an increase in fatty acid binding protein with no increase in fatty acid translocase [36]. Longer AIT (i.e. 20 weeks) in healthy individuals has also been shown to result in significant increases in the activity of other enzymes involved in both glycolytic and oxidative pathways (i.e. HK, PFK, malate dehydrogenase (MDH) and  $\beta$ -HAD) [97]. Collectively these studies indicate that both AIT and SIT are effective in activating cell signalling cascades that ultimately improve the oxidative and metabolic capacity of muscle.

Furthermore, while there are extant AIT studies on intramuscular metabolic adaptations in the clinical population, to the best of the author's knowledge, there are no existing SIT studies which have observed for these adaptations in this population. The beneficial effects of HIIT are not limited to healthy populations. Indeed, AIT has been shown to be effective in enhancing aerobic enzymatic activity and increasing protein content of PGC-1 $\alpha$ , COX I, II and IV and GLUT IV and in a variety of clinical populations including type 2 diabetics, patients with medically treated post-infarction heart failure and overweight and obese individuals [31-33, 94]. These results are important since many of these markers have been implicated the development or progression of chronic diseases. For instance, citrate synthase activity (i.e. indicator of mitochondria content) is reduced in obese insulin resistant individuals and type 2 diabetics [98-100], thus exercise induced increase in expression and content of PGC-1 $\alpha$

leading to mitochondria biogenesis may assist in the treatment and prevention of metabolic syndrome. Collectively, these studies have shown that HIIT can induce favourable intramuscular metabolic adaptations in both healthy and clinical populations. Specifically, both AIT and SIT can enhance intramuscular oxidative capacity and improve glucose utilisation, however it seems that while AIT can improve intramuscular lipid oxidation and utilisation, the effect that SIT has on lipid utilisation requires more research.

## **2.5 Cardiometabolic benefits from high-intensity interval training in both healthy and clinical populations**

Undesirable metabolic and physiological changes are corollary to assuming a more sedentary lifestyle and together with environmental factors such as increased availability of food can strongly condition the obesity phenotype [4, 101]. A chronic state of obesity may result in a pre-morbid state, known as metabolic syndrome, which is usually characterised by a large waist circumference, dyslipidaemia, glucose intolerance, hypertension, low grade inflammatory and prothrombotic states [102, 103]. Metabolic syndrome predisposes an individual towards greater risks of developing related co-morbidities such as type II diabetes and cardiovascular diseases [104, 105]. This section will further illustrate how HIIT can elicit favourable systemic physiological adaptations, thus attenuating metabolic risk factors.

### **2.5.1 Aerobic capacity and exercise tolerance**

In both clinical and healthy individuals, short term (i.e. 2 – 3 weeks) AIT and SIT have been shown to increase aerobic capacity [1, 36, 83, 106]. For instance, 2 weeks of AIT (i.e. 4 min at 90% of  $\dot{V}O_{2peak}$  interspersed with 2 min rest, 13 sessions) increased absolute  $\dot{V}O_{2peak}$  by 13% in recreationally active women [36]. In patients with severe chronic heart failure, 3 weeks of AIT (i.e. cycling: 30 sec at 50% peak power output (PPO) interspersed with 60 sec at 15 W, 5 times a week, treadmill walking: 60 sec at 2.4 mph interspersed with 60 sec at 0.9 mph, 3 times a week) increased relative  $\dot{V}O_{2peak}$  by 20% [83]. Following 2 – 3 weeks of progressive SIT, relative  $\dot{V}O_{2max}$  increased by 5% in recreationally active individuals [106] and absolute  $\dot{V}O_{2peak}$  increased by 8.4% in

overweight/obese individuals [1]. Likewise, long term AIT (i.e. 4 weeks to 6 months) [32-34, 64, 84, 94, 107, 108] and SIT (i.e. 4 – 15 weeks) [35, 73, 96, 106, 109-111] studies also reported improvement in aerobic capacity. Importantly, improvement in aerobic fitness is a favourable adaptation, since it is a robust indicator of all-cause mortality [112-115]. Furthermore, in some SIT and AIT studies, an increase in exercise tolerance is concomitant with improvement in aerobic capacity [35, 36, 83]. For instance, after two weeks of AIT (i.e. 4 min at 90% of  $\dot{V}O_{2peak}$  interspersed with 2 min rest) with improvement in aerobic capacity, recreationally active women responded to a similar 60 min cycling trial (~60% of  $\dot{V}O_{2peak}$ ) at a lower percentage of their  $\dot{V}O_{2peak}$ , with increased fat oxidation rate, reduced carbohydrate oxidation rate and with both lower HR and plasma epinephrine levels [36]. The attenuated epinephrine indicates a reduced sympathetic response towards the similar workload following training resulting in a lower HR response and reduction in glycogenolysis that is supported by the changes in substrate utilisation [116, 117]. Similarly, two weeks of SIT in healthy individuals improved relative aerobic capacity and responded to a similar workload (i.e. 1 h at 65% of  $\dot{V}O_{2peak}$ ) with reduced absolute oxygen consumption and carbohydrate utilisation in tandem with an increase in lipid oxidation rate [118]. Notably, in patients with stable chronic heart failure, an increase in exercise tolerance was evident by the similar HR, blood pressure (BP) and whole body rating of perceived exertion (RPE) responses despite increase training workload.

### **2.5.2 Cardiovascular adaptations and improvements in hemodynamic parameters**

Improvement in aerobic fitness may partially explained by favourable cardiovascular adaptations. Both SIT and AIT studies have reported concomitant improvement in endothelial function with the observed improvement in aerobic fitness in both healthy and clinical populations [32, 33, 84, 94, 107, 110]. This improvement may be partly explained by an increase in plasma nitric oxide observed in two of the AIT studies [33, 84]. The literature also suggests that AIT can reduce the risk of coronary artery disease. For instance, oxidised low density lipoprotein (LDL), a sensitive marker for coronary artery disease was markedly reduced after 4 months of AIT (i.e. 4 min at 90% of  $HR_{max}$  interspersed with 3 min at 70% of  $HR_{max}$ , 3 sessions a

week) in individuals with metabolic syndrome [33]. However, this finding is not observed in all studies, thus further research is warranted [84, 94]. Furthermore, AIT and SIT can also significantly lower blood pressure (BP). For instance, long term AIT can favourably lower both systolic [33, 84, 107] and diastolic [33, 84, 94, 107] BP. For instance, 4 months of AIT (i.e. 4 min at 90% of  $HR_{max}$  interspersed with 3 min at 70%  $HR_{max}$ , 3 sessions a week) decreased systolic BP ( $144 \pm 5$  vs.  $135 \pm 5$  mmHg), diastolic BP (i.e.  $95 \pm 3$  vs.  $89 \pm 3$  mmHg) and mean arterial pressure (MAP) (i.e.  $111 \pm 3$  vs.  $105 \pm 3$  mmHg). Alternatively, 2 to 6 weeks of SIT can decrease systolic BP in both adolescents [119] and overweight/obese individuals [1]. For instance, 7 weeks of SIT (i.e. progressive increment from 4 sets of 20 maximal sprint running interspersed with 20 – 30 sec of active recovery, 3 sessions a week) decreased systolic BP ( $112 \pm 10$  vs.  $106 \pm 11$  mmHg) in adolescents [119]. However, Rakobowchuk et al., 2008 did not observe any changes in brachial BP following 6 weeks of SIT (i.e. progressive increment from 4 sets of 30 sec maximal “all-out” sprints interspersed with 4.5 min of cycling at 30 W) in healthy individuals [110]. While short term studies suggest that SIT can lower systolic BP, longer term studies are warranted to elucidate if it can lower both systolic and diastolic BP. Lastly, favourable cardiac remodelling such as reverse left ventricular remodelling resulting in reduced end diastolic and systolic volumes and a greater left ventricular ejection fraction from 12 weeks AIT (i.e. 4 min at 90 – 95% of  $HR_{peak}$ , 3 min at 70% of  $HR_{peak}$ , 3 sessions a week) have been observed in patients with post infarction heart failure [32]. In fact, further insights can be observed from isolated cardiomyocytes from rats and mice extracted after AIT. 4 – 6 weeks of AIT can induce distinctive adaptations (i.e. balanced hypertrophy, improved contractility and intracellular calcium handling and enhanced myofilament calcium sensitivity) in cardiomyocytes [120-122]. Notably, AIT intervention studies involving patients with previous history of cardiovascular diseases (i.e. chronic heart failure and coronary artery disease) suggests that AIT can be safely prescribed to those who are at risk of developing cardiovascular diseases. No studies have yet to examine the efficacy of SIT in cardiac rehabilitation, possibly due to its supramaximal intensity, making it unsuitable for cardiac rehabilitation.

### 2.5.3 Anthropometric and body composition

Obesity is associated with increased risk of developing both type 2 diabetes and cardiovascular diseases [123, 124]. Therefore, an important objective for obese individuals or those with metabolic syndrome is improvement in body composition. Weight loss and reduction in waist circumference can reduce the related risks for these individuals [125, 126]. Long term (i.e. 3 – 12 months) studies observed significant reduction in body fat percentage in overweight [94], healthy [97] individuals and adolescents [84] after AIT intervention. For example, 3 months of AIT (i.e. 4 min at 85-95% of  $HR_{max}$ , 3 min at 50-60% of  $HR_{max}$ ) resulted in a 2% reduction in body weight, reduction in BMI (i.e.  $36.6 \pm 1.2$  vs.  $36.0 \pm 1.2$  kg/m<sup>2</sup>) and 2.2% reduction in body fat in obese individuals [94]. However, Nybo et al. (2010) reported that 5 months of AIT (i.e. 2 min at above 95% of  $HR_{max}$ , 1 min of passive rest, 2 sessions a week) did not result in any changes in lean body mass and body fat percentage in healthy individuals [63]. It is possible that the lack of change in body composition in the study by Nybo et al. (2010) could be due to difference in participants' demographics and training protocols. For instance, participants in Nybo et al. (2010) exercised for 20 min (includes warm-up and cool down) per session, twice a week, which is lower in training volume in comparison to the other studies, in which participants exercised more than 20 min per session, thrice a week [63, 84, 94]. Therefore, while AIT can improve body composition by reducing fat mass, the training duration plays a crucial role in determining its efficacy. Few SIT studies have examined changes in body composition, in a study by Buchan et al. (2011), body composition (i.e. body fat percentage) was not affected after 7 weeks of SIT (i.e. progressive increment from 4 sets of 20 maximal sprint running interspersed with 20 – 30 sec of active recovery, 3 sessions a week) in adolescents [119]. However, Trapp, Chisholm, Freund and Boutcher (2008) reported that 15 weeks of SIT (i.e. progressive increment of 8 sec sprinting against 0.5kg followed by 12 sec of slow cycling over 15 weeks) significantly reduced body mass, fat mass and trunk fat in health women [111]. The difference in both recruited populations and exercise protocols could have explained the disparity in findings between Buchan et al. (2011) and Trapp et al. (2008) however; it can also indicate that a longer period of SIT (i.e. 15 weeks) is needed before improvement in body composition can be observed.

Presence of abdominal obesity, as assessed by waist circumference is a criteria for the classification of metabolic syndrome by several health organisations [127]. 3 – 4 months of AIT can reduce waist circumference in individuals classified with metabolic syndrome and overweight adolescents [33, 84, 107]. For instance, 3 months of AIT (i.e. 4 min at 90-95% of  $HR_{peak}$ , 3 min at ~70% of  $HR_{peak}$ , 3 sessions a week) significantly reduced waist circumference (i.e.  $109.6 \pm 10$  vs.  $108.3 \pm 10.7$  cm) in those with metabolic syndrome. However, SIT effect on waist circumference is equivocal. For instance, in one study, 2 weeks of SIT in overweight/obese males significantly reduced both waist (i.e.  $101.3 \pm 2.7$  vs.  $98.9 \pm 3.1$  cm) and hip (i.e.  $110.9 \pm 2.2$  vs.  $109.8 \pm 2.2$  cm) circumferences [1]. However, Buchan et al. (2011) did not observe any changes to waist-hip ratio after 7 weeks of SIT in adolescents [119]. Thus, AIT can elicit reduction in waist circumference, however more SIT studies are warranted to further investigate its efficacy in reducing waist circumference.

At present, it appears that AIT is superior to SIT in eliciting favourable anthropometric and body composition changes; furthermore, longer duration SIT studies examining its efficacy in anthropometric and body composition changes are warranted.

#### **2.5.4 Glucose homeostasis**

Exercise is important in the management of glucose homeostasis in those with metabolic syndrome and type 2 diabetes. Extant studies show that short (i.e. 2 – 3 weeks) [31] and long (i.e. 3 – 5 months) [33, 63, 84] term AIT can mitigate hyperglycaemia during fasting and under glucose loading in both healthy and clinical populations. For instance, 2 weeks of AIT (i.e. 60 sec at 90% of  $HR_{max}$ , 60 sec at 30 W, 3 sessions a week) reduced average 24 h blood glucose concentration (i.e.  $7.6 \pm 1.0$  vs.  $6.6 \pm 0.7$  mmol/L) and sum of the 3 h postprandial area under glucose curves for breakfast, lunch and dinner (i.e.  $965 \pm 483$  vs.  $679 \pm 437$  mmol/L.9 h) in type 2 diabetics [31]. Additionally, 5 months of AIT (i.e. 2 min at above 95% of  $HR_{max}$ , 1 min of passive rest, 2 sessions a week) reduced fasting glucose level (i.e.  $5.7 \pm 0.2$  vs.  $5.2 \pm 0.1$  mM) and glucose level after oral glucose tolerance test (OGTT) (i.e.  $6.1 \pm 0.6$  vs.  $5.1 \pm 0.4$  mM) in healthy individuals [63]. However, a 3 month AIT (i.e. 4 min at 90-96% of  $HR_{peak}$ , 3 min at ~70% of  $HR_{peak}$ , 3 sessions a week) study by Stensvold et al. (2010) did

not improve insulin sensitivity, reduce fasting glucose levels and glycated haemoglobin (HbA<sub>1c</sub>) in individuals with metabolic syndrome [107]. Until others report can corroborate that reported by Stensvold et al. (2010), the literature indicates that AIT can improve glucose homeostasis. Studies suggest that SIT as short as 2 weeks up to 6 weeks can improve insulin sensitivity [1, 109, 128, 129], while two of these studies reported no changes to fasting insulin and glucose [128, 129], Whyte et al. (2010) reported a reduction in fasting insulin after 2 weeks of SIT in overweight/obese individuals [1]. Trapp et al. (2008) also found a significant reduction in fasting insulin level following SIT (i.e. progressive increment of 8 sec sprinting against 0.5kg followed by 12 sec of slow cycling over 15 weeks) in healthy women [111]. Also, Metcalfe, Babraj, Fawcner and Vollaard (2012) reported gender specific improvement in insulin sensitivity such that insulin sensitivity improved by 28% only in males [109]. It appears that there are divergent findings on the efficacy of SIT in improving fasting glucose and insulin levels and more research is warranted, however it may improve peripheral insulin sensitivity under glucose loading. Improvement in peripheral insulin sensitivity under glucose load could have possibly resulted from the previously mentioned intramuscular enhancement in glucose utilisation and transport after SIT.

### **2.5.5 Lipid metabolites**

Dyslipidemia is characterised by having elevated levels of triglycerides rich lipoproteins (VLDL) or remnants concentration of VLDL and low levels of high density lipoproteins (HDL) in the plasma [130]. It is associated with increased risk of cardiovascular diseases [131, 132] and is a criterion for the classification of metabolic syndrome proposed by several health institutions [127, 133, 134]. 3 – 4 months of AIT in both healthy [63] and overweight individuals [94] and those with metabolic syndrome [33, 107] did not result in any change to fasting triglycerides, HDL, total cholesterol and LDL levels. For instance, 3 months of AIT (i.e. 4 min at 90% of HR<sub>max</sub>, 3 min at 70% HR<sub>max</sub>) did not affect triglycerides, total cholesterol and low density lipoprotein (LDL) in those with metabolic syndrome [33]. However, Tjønnå et al. (2009) reported a divergent decrease in HDL levels after 3 months of AIT in overweight adolescents [84]. This disparity could be a result of the difference in population recruited between studies.

Likewise, 2 weeks of SIT did not elicit any changes to fasting triglycerides, total cholesterol, HDL and non-esterified fatty acid (NEFA) in overweight/obese individuals [1]. Similarly, 7 weeks of SIT (i.e. progressive increment from 4 sets of 20 maximal sprint running interspersed with 20 – 30 sec of active recovery, 3 sessions a week) did not elicit any changes to HDL, LDL and total cholesterol levels in adolescents, however authors observed an increase in triglycerides. These studies indicate that both AIT and SIT may have negligible effects on lipid metabolites; however more research is warranted before it can be conclusive.

## **2.6 Comparisons of metabolic adaptations and health benefits between HIIT and conventional CME protocols**

To fully appreciate the rationale behind the burgeoning interest in promoting high-intensity interval training as an alternative to the preferred conventional continuous moderate-intensity exercise (CME) modal, we have to draw insights from comparison studies between these two exercise modals. Existing comparison studies tend to feature two types of study designs. The first uses an isocaloric design, allowing exercise intensity to be the contrasting feature. It is used mostly in the comparison between CME and AIT. For instance, participants performing CME in Tjønnå et al. (2008) had to cycle longer to equalise work volumes between protocols (i.e. AIT: 43 min, includes warm up and cool down vs. CME: 47 min) [33]. In another design, both energy expenditure and exercise duration are not controlled for to highlight the difference in time commitment and energy expenditure. It is used mostly for comparison between SIT and CME. For instance, in the study by Burgomaster et al. (2008), the weekly time commitment (i.e. SIT: 1.5 h vs. CME: 4.5 h) and total training volume (i.e. SIT: 225 kJ/week vs. CME: 2250 kJ/week) between SIT and CME protocols differs markedly.

### **2.6.1 Intramuscular metabolic and ion handling adaptations**

Impairment in calcium handling can contribute to cardiomyocytes dysfunction, conversely, improvement in calcium handling can enhance contractility and increase cardiac power output [135, 136]. Likewise, improvement to calcium handling in skeletal muscles can improve muscle function via an increase in rate of force production and



power output. It is demonstrated in animal models that AIT is superior over CME in enhancing intramuscular calcium handling [137, 138]. Likewise, in human studies, AIT enhanced reuptake of  $\text{Ca}^{2+}$  in the sarcoplasmic reticulum in skeletal muscles while no changes were detected after CME [32, 33, 94]. For instance, compared with CME (i.e. 47 min at 70 - 75% of  $\text{HR}_{\text{max}}$ , 3 sessions a week), 3 months of AIT (i.e. 4 min at 90% of  $\text{HR}_{\text{max}}$ , 3 min at 70% of  $\text{HR}_{\text{max}}$ , 3 sessions a week) increased maximal rate of calcium uptake by sarcoplasmic reticulum calcium ATPase by 60% with no observed effect in the CME group and significant improvement to peak systolic mitral annulus velocity (i.e. an index of global contractility in the heart) by 22% with no observed effect in the CME group.. To the best of the authors' knowledge, no study has yet to compare the effect of SIT with CME on skeletal muscle calcium handling and this warrants further study. Comparison studies between AIT and CME on changes to PGC-1 $\alpha$  protein levels, showed that between the two protocols, PGC-1 $\alpha$  protein levels increased only after AIT and not CME [32, 33, 94] and in two of these studies, energy expenditure between AIT and CME was similar [32, 33]. Furthermore, 6 weeks of SIT, despite having a lower training volume (i.e. SIT: ~225 vs. CME: 2250 kJ/week) and time commitment (i.e. SIT: 1.5 vs. CME: 4.5 h/week) than CME, elicited similar marked increase in protein content of PGC-1 $\alpha$  to CME [65]. These findings suggest that with sufficiently high exercise intensity, training volume can be reduced to elicit similar mitochondrial biogenesis, therefore illustrates the time efficient characteristic of HIIT. In fact, the transcription of PGC-1 $\alpha$  is influenced by exercise intensity [139]. On this basis, improvement in intramuscular metabolic capacity should be greater after HIIT. Indeed, Tremblay, Simoneau and Bouchard (1994) observed that 20 weeks of AIT (i.e. 30 min at 70% heart rate reserve, 15 - 30 sec at 60% of maximal aerobic power and 60 - 90 sec at 70% of maximal aerobic power) up-regulated both phosphofructokinase and hexokinase activities, while CME (i.e. 30 - 45 min at 60 - 85% maximal heart rate reserve, 4 - 5 sessions a week) down-regulated and induced greater enzymatic activities in both lipid oxidative and citric-acid cycle pathways than CME [97]. Furthermore, total energy expenditure was different between the two protocols (i.e. CME: 120.4MJ vs. AIT: 57.9MJ) [97]. Difference in intramuscular adaptations between both HIIT and CME protocols, could be partly explained by the difference in substrate utilisation relative to

exercise intensity. Considering that exercising at a constant intensity of 65%  $\dot{V}O_{2max}$  for 30 min, would elicit maximal lipid oxidation rate, the glycolytic pathway may not be sufficiently stressed, therefore partly explaining the down-regulation of enzymatic activities in the glycolytic pathway observed after CME [140]. Comparison studies between SIT and CME reported similar increment in intramuscular metabolic capacity despite difference in total training volume [65, 141]. For instance, in a 6 week SIT versus CME comparison study, both training protocols induced similar increase in intramuscular glycolytic and lipid oxidative potentials, despite a substantial difference in total training volume (i.e. ~225kJ/week vs. ~2250kJ/week) [65]. Repeated sprints slowly increase energy derivation from the aerobic pathway, which likely explains the observed increase in both glycolytic and aerobic capacities [75, 142]. Therefore, both SIT and AIT are superior to CME in increasing intramuscular metabolic capacity, mediated by their ability to induce greater mitochondria biogenesis.

### **2.6.2 Cardiovascular adaptations and improvements in hemodynamic parameters**

CME is commonly prescribed to improve aerobic fitness, however, in comparison to AIT; most comparison studies reported that the latter is superior to CME in eliciting greater improvement in aerobic capacity [32-34, 63, 64, 94]. Comparison studies using an isocaloric design between AIT and CME which reported greater improvement in aerobic fitness after AIT suggests that improvement in aerobic fitness is likely to be exercise intensity dependent [32-34, 64]. On the contrary, others have also reported similar improvements in aerobic capacity after both AIT and CME [108, 143]. For instance, Wisloff et al. (2007) reported that AIT (i.e. 4 min at 90 – 95% of  $HR_{peak}$ , 3 min at 70%  $HR_{peak}$ , 3 sessions a week) elicited a greater improvement to  $\dot{V}O_{2peak}$  (i.e. AIT: 35% vs. CME: 16%) compared to CME (i.e. 47 min at 70 – 75%  $HR_{peak}$ , 3 sessions a week) [32]. However, Warburton et al. (2004) observed that both AIT (i.e. 2 min at 90%  $\dot{V}O_{2peak}$ , 2 min at 40%  $\dot{V}O_{2peak}$ , 3 sessions a week) and CME (i.e. 1% below anaerobic threshold with progressive increment from 30 – 48 min, 3 sessions a week) elicited similar increment in  $\dot{V}O_{2max}$  [144]. It is likely that the disparity between both findings is a result of the different training protocols, and is plausible that the duration

spent exercising in the high-intensity is also crucial to the improvement in aerobic capacity.

In addition to the improvement in calcium handling with enhance muscular contractility, AIT is also superior to CME in eliciting reverse left ventricle remodelling of the heart and improvement in endothelial function [32]. For instance, Wisloff et al. (2007) observed a decline in left ventricle systolic (i.e. by 15%), diastolic (i.e. 12%) diameters, a decline in pro-B-type natriuretic peptide level (i.e. marker of cardiac hypertrophy and severity of heart failure) by 40% and improvement in flow mediated dilation only after AIT and not CME [32]. Furthermore, the observed exercise intensity dependent balanced hypertrophy in cardiomyocytes in rats after AIT, supplements how AIT is superior to CME in enhancing cardiac function [138]. These studies indicate that AIT is likely to be superior to CME as an exercise intervention for cardiac rehabilitation. Pertaining to SIT and CME comparison studies, both protocols improved aerobic capacity [73, 110, 119], endothelial function [110] and peripheral artery distensibility [110] despite marked differences in exercise volume. For instance, despite difference in weekly workloads (i.e. AIT: 225 kJ vs. CME: 2250 kJ), both SIT and CME induced similar improvement in endothelial function and increment in distensibility in the popliteal artery [110]. It is likely that the unique feature of work – rest intervals allows for prolonged training periods at or near maximal oxygen consumption, thereby also partly explaining the greater improvement in aerobic capacity after AIT and SIT [145, 146]. However, despite AIT being superior over CME in eliciting greater improvements in vascular function, both AIT and CME have similar blood pressure lowering effect [33, 94]. For example, Tjønnå et al. (2008) reported that AIT (i.e. 4 min at 90% heart frequency max, 3 min at 70% heart frequency max) and CME (i.e. 47 min at 70% heart frequency max) reduced systolic (i.e. by ~ 10 mmHg), diastolic (i.e. by ~ 6 mmHg) and mean arterial (i.e. by ~ 7 mmHg) blood pressures, although reduction of diastolic blood pressure in the CME group did not reach statistical significance [33]. Also, Schjerve et al. (2008) observed a decrement in diastolic blood pressure in both AIT (i.e. 4 min at 85 – 95%  $HR_{max}$ , 3 min at 50 – 60%  $HR_{max}$ ) and CME (i.e. 47 min at 60 – 70%  $HR_{max}$ ) groups by 6 mmHg and 8 mmHg respectively, with no change in systolic blood pressure [94]. Similarly, 7 weeks of either SIT (i.e. progressive increment from 4 sets of 20

maximal sprint running interspersed with 20 – 30 sec of active recovery, 3 sessions a week) or CME (i.e. 20 min of running at 70%  $\dot{V}O_{2max}$ ) was reported to lower systolic blood pressure by ~ 5 mmHg in adolescents, although reduction in the CME did not reach statistical significance and there was no observable change in diastolic blood pressure in both protocols [119]. Therefore, extant studies demonstrated that AIT, SIT and CME can lower blood pressures however, reduction of blood pressure in CME usually does not reach statistical significance, hence comparison studies with greater statistical power may be needed to elucidate if CME in comparison to AIT and SIT can also significantly reduce blood pressure.

### **2.6.3 Metabolic risk factors**

Besides cardiovascular parameters, 4 months of AIT (i.e. 4 min at 90% of  $HR_{max}$ , 3 min at 70% of  $HR_{max}$ , 3 sessions in a week) has been reported to be superior to CME (i.e. 47 min at 70%  $HR_{max}$ ) in reducing overall metabolic risk factors (i.e. fasting glucose, HDL, triglycerides, body mass index, etc.), indicating that AIT is an effective intervention in reversing metabolic syndrome [33]. Furthermore, 3 - 4 months of AIT can attenuate lipogenesis in adipose tissue, while both AIT and CME are equal at reducing body weight and waist circumference [33, 94]. However, Tremblay et al. (1994) observed a more pronounced reduction in subcutaneous adiposity as assessed using skin fold measurements after AIT (i.e. 30 min at 70% heart rate reserve, 15 - 30 sec at 60% of maximal aerobic power and 60 – 90 sec at 70% of maximal aerobic power) in comparison to CME (i.e. 30 – 45 min at 60 – 85% maximal heart rate reserve, 4 – 5 sessions a week) [97]. Conversely, Nybo et al. (2010) observed that 3 months of AIT (i.e. 2 min at above 95% of  $HR_{max}$ , 1 min of passive rest, 2 sessions a week) was less effective than CME (60 min at 65%  $\dot{V}O_{2max}$ , 2.5 times a week) in reducing overall body fat percentage when assessed using the DEXA [63]. Due to the difference in training protocols between studies, direct comparison cannot be made and may account for the difference in findings. Therefore, while AIT can attenuate lipogenesis in adipose tissue, its ability to reduce body weight and improve body composition may be similar or inferior to that of CME and more studies are needed to elucidate it further. In a 7 week SIT (i.e. 20 m maximal sprints, 20 – 30 sec rest, 4 – 6 sets) and CME (i.e. 20 min

at 70%  $\dot{V}O_{2max}$ ) comparison study, while only BMI was improved after SIT, both BMI and body fat percentage were reduced after CME [119]. In another study, Macpherson, Hazell, Olver, Paterson and Lemon (2011) observed similar reduction in fat mass and increment in lean tissue mass after 6 weeks of either SIT (i.e. 30 sec maximal effort sprint, 4 min rest, 4 – 6 sets per session) or CME (i.e. 30 – 60 min at 65%  $\dot{V}O_{2max}$ ) in healthy individuals [73]. However, others have reported that 15 weeks of SIT is superior to CME in reducing both total body and fat masses and increasing trunk lean mass of sedentary but otherwise healthy women [111]. As such, it is likely that 6 – 7 weeks of SIT can elicit improvement to BMI and body composition, equal that observed after a similar period of CME. While, it is likely that SIT is superior to CME over longer training period of up to 15 weeks, more studies over the similar time frame are needed to substantiate it further.

In comparison with CME, 3 months of AIT is observed to have similar effect with reducing blood glucose during OGTT in healthy individuals while in one other study [63], 4 months of AIT had a greater effect in reducing fasting blood glucose levels, resulting in an improvement in both insulin sensitivity and beta-cell function using the HOMA model in those with metabolic syndrome [33]. In a comparison study between SIT and CME in adolescents over 7 weeks, authors observed that only CME marked reduced fasting insulin levels [119]. However, Trapp et al. (2008) reported that after 15 weeks of exercise intervention, only the SIT (i.e. progressive increment of 8 sec sprinting against 0.5kg followed by 12 sec of slow cycling over 15 weeks) group had a significant decrease in fasting insulin levels from the control group. These observations suggest AIT is superior if not similar to CME in improving glucose homeostasis. Few studies have compared the effect which both CME and SIT have on glucose and insulin homeostasis, the findings are equivocal, therefore further SIT and CME comparison studies are needed to investigate this further.

Finally, 4 months of AIT and CME did not elicit any changes in lipid profile (i.e. triglycerides, total cholesterol and LDL) [33]. Similarly, in a study by Buchan et al. (2011), both SIT and CME did not elicit any changes in HDL, LDL and total cholesterol levels but elicited an increase in triglycerides level in adolescents [119]. The present of

elevated triglycerides level is uncommon, however considering that physiological responses to exercise may differ between adults and adolescents, this may be an isolated effect in adolescents. [147]. There are limited studies comparing the effects of both AIT and SIT with CME on lipid profile, however present studies show that in comparison to CME, both SIT and AIT elicit negligible changes to lipid profile, therefore more studies are warranted.

In summary, both AIT and SIT are superior to CME in eliciting increment in both intramuscular glycolytic and oxidative capacities, despite marked difference in energy expenditure and time commitment. These enhancements can be attributed to the greater exercise intensity exposed during AIT and SIT. Furthermore, AIT is superior to CME in reducing overall metabolic risk factors. In fact, studies have demonstrated that both AIT and SIT have similar blood pressure lowering effect to CME. However, compared to CME, both AIT and SIT have divergent results on promoting weight loss and improvement to body composition. AIT seems to have similar if not inferior effects in these aspects as compared to CME while SIT is inferior to CME. In regards to glucose homeostasis, the literature indicates that AIT is superior to CME in maintaining fasting glucose levels and improving basal insulin sensitivity and beta-cell function. In addition, it is inconclusive if SIT is superior to CME in improving fasting plasma insulin levels or insulin sensitivity, since findings are equivocal; therefore more research is needed to ascertain it. Importantly, although isocaloric studies comparing between AIT and CME reported similar adaptations or improvements to health markers in both protocols, it must be highlighted that these favourable changes occurred over shorter exercise duration, meaning lower time commitment in the AIT protocols. These studies including those studies comparing between SIT and CME, underscores the time efficient quality of HIIT and that improvement to health, exercise tolerance and capacity can occur over shorter time using HIIT, as opposed to engaging in CME.

## **2.7 Gaps in the research**

In the previous sections, we have discussed about the intramuscular adaptations and health benefits observed after both short and long term HIIT. Furthermore, we also

examined these observations in relation to the conventional continuous moderate-intensity exercise in an attempt to determine its efficacy as a potential alternative exercise modal. However as mentioned previously, there are still gaps in the literature which call for more research before we can recommend its prescription to the general public. This study is designed to address some of these gaps and they will be discussed in the following sections.

### **2.7.1 Comparison of acute responses to a single high-intensity interval session versus a single continuous moderate-intensity session**

Long term study of training adaptations and the resulting health improvements from long term adherence to an exercise intervention is important to establish its long term efficacy. However, the study of acute responses to a particular exercise intervention is just as important. These acute responses or termed “last exercise bout” effects can be defined as the immediate and transient physiological responses elicited by a single bout of exercise, which can occur during or over a short period of time after exercise and can have major positive health related implications [37]. Furthermore, “last exercise bout” effects can confound results in long term training studies, making it difficult to distinguish them from training adaptations [148].

Research on acute responses to a single session of SI is scarce. A single SI session consisting of 4 sets of 30 seconds “all-out” sprints against 0.088kp/kg interposed with 4 min of active recovery with or without replacement of the energy deficit from the session has been reported to significantly reduce plasma level of triglycerides but not insulin and glucose levels in response to a high fat meal 24 h after the session; furthermore, fasting plasma triglycerides, insulin, glucose and NEFA levels were not affected [149]. Consistently, others have reported that a single bout of SI did not affect plasma glucose, insulin and NEFA levels between 12 h to 72 h post exercise [129, 150]. In comparison between a single SI (i.e.  $125\% \dot{V}O_{2peak}$  for 30 seconds interposed with 5 min of rest) versus CME (i.e.  $75\% \dot{V}O_{2peak}$  for 15 min interposed with 2 min of rest) session, only the CME resulted in significant improvement to both fasting and OGTT insulin sensitivity indices, possibly resulting from the observed lower fasting and OGTT at 60 min insulin levels [150]. However it must be highlighted that long term SIT can

improve insulin sensitivity as mentioned previously [1, 128, 129]. In adolescents, a single SI session consisting of 2 sets of 30 seconds “all-out” sprints against 7.5% of their body mass, interposed with 4 min of passive recovery can significantly reduce systolic blood pressure 90 min post-exercise and also increase resting energy expenditure over 30 min post exercise [151]. The health implication of “last exercise bout” effect can be illustrated using this finding, for example, we can posit that this protocol can be utilised by adolescents with pre-hypertension in accumulative daily 9 min bouts to regulate their daily average blood pressure.

The implication of the “last exercise bout” effects on confounding “actual” training adaptations can be illustrated in the following example. In a 2-week SIT intervention on overweight/obese sedentary individuals by Whyte, Gill and Cathcart (2010), they reported transient decrease in plasma insulin levels under glucose loading, resting energy expenditure, carbohydrate oxidation rate and systolic blood pressure, increase in lipid oxidation rate and improvement in insulin sensitivity, all of which were no longer significant against baseline levels at 72 h post intervention [1]. The authors postulated that such a finding could be due to the acute response from the last training bout [1]. Furthermore, specific to insulin sensitivity, Richards et al. (2010) measured change in insulin sensitivity 72 h after a single SI session, reported no significant difference [129]. Therefore, it is possible that the significant improvement in insulin sensitivity observed by Whyte et al. (2010) is an acute response to the last exercise bout. Kessler, Sisson and Short (2012), emphasised the importance of defining the timing of post training tests to avoid confounding adaptive effects from exercise training with “last exercise bout” effects [70].

In a recently published study, Bartlett et al. (2012) reported that both a single aerobic interval (AI) session involving 6 sets of 3 min work to rest ratio using workloads corresponding to 90%  $\dot{V}O_{2max}$  for work and 50%  $\dot{V}O_{2max}$  for rest intervals and single CME session of cycling for 50 min at a workload corresponding to 70%  $\dot{V}O_{2max}$ , when matched for average intensity, workload and duration, induced similar level of phosphorylation of the intracellular signalling pathways that elicit mitochondria biogenesis and consequentially matched level of PGC-1 $\alpha$  mRNA 3 hours post exercise



[152]. However, long term comparison studies have observed that long term AIT induced a greater increase in PGC-1 $\alpha$  protein content than CME [33, 94]. Despite this disparity, direct comparison cannot be made due to differences in the study designs, such as a longer work interval (i.e. 3 vs. 4 min) and the lack of control for both exercise duration and mean intensity in the long term studies. In fact, having a longer work interval means that the mean intensity for the AIT protocol in both the long term studies was greater than the CME protocol (i.e. ~80% vs. 70% of HR<sub>max</sub> or heart frequency max) while in the Bartlett et al. (2012), mean intensity (70% of  $\dot{V}O_{2max}$ ) was similar between protocols. Since transcription of PGC-1 $\alpha$  mRNA is likely to be exercise-intensity dependent and influenced by the increasing muscle fibres recruitment relative to exercise intensity limited at maximal exercise intensity, this could be a plausible explanation as to why Bartlett et al. (2012) did not find any difference in PGC-1 $\alpha$  mRNA content between both protocols while the long term comparison studies did [139, 153, 154]. In addition, Bartlett et al. (2012) also noted that elevation in plasma levels of both glycerol and NEFA were significantly greater 3 hours after CME and attributed this finding to lactate-induced inhibition of lipolysis, which levels were more elevated during the AI, possibly due to differential muscle fibres recruitment (i.e. both type I and II fibres during AI) [152]. The difference in substrate appearance is consistent with previous findings that low – moderate intensities resulted in greater rate of appearance ( $R_a$ ) of free fatty acids (FFA) with the  $R_a$  attenuating with increasing exercise intensity [155]. Interestingly, while increasing exercise intensity can enhance lipid oxidation and increase energy expenditure for over 90 min post exercise, an interval cycling session utilising 1:2, work to recovery ratio at 85%  $\dot{V}O_{2max}$  for work and 30%  $\dot{V}O_{2max}$  for recovery, in comparison with moderate-intensity cycling at 50%  $\dot{V}O_{2max}$ , with both protocols matched for mean exercise intensity, duration and total energy expenditure did not result in any significant difference in energy expenditure, RER kinetics or lipid oxidation rate over 90 min post exercise [74]. In addition, a single bout of CME can reduce 24 h systolic, diastolic blood pressures, night time systolic and diastolic blood pressures, while a single bout of AI can reduce 24 h systolic blood pressure and night time systolic blood pressure in medically treated hypertensive patients [156]. From this, it is clear that CME is superior to AI in eliciting favourable acute hemodynamic

responses however it also highlights that AI can be also used as an intervention to mitigate hypertension blood pressure.

In summary, research on the “last exercise bout” effects from HIIT modalities is scant. Considering that AIT in comparison to SIT, is more widely prescribed to both the general and especially the clinical populations, the need for more research into these acute responses to a single AI session is twofold: it provides exercise physiologists with greater understanding so as to improve the prescription of AI as both prophylactic and auxiliary treatment for lifestyle-related chronic diseases and allows for better discrimination against “true” training adaptations against “last exercise bout” effects. At present, the literature has provided some insights on the acute responses to a single AI session: it activates intramuscular signalling pathways that promote mitochondrial biogenesis, attenuate appearance of glycerol and NEFA, enhance lipid oxidation rate and lower RER kinetics for up to 90 min post exercise and in hypertensive patients it improves 24 h average blood pressure. However, an interesting perspective observed is that when all three exercise variables, namely mean exercise intensity, duration and expenditure are controlled for, the acute intramuscular responses and post exercise metabolism after either a single AI or CME session are both similar. Notably, more research is needed to provide for a more comprehensive understanding of “last exercise bout effects” after an AI session on other metabolic and health markers such as insulin sensitivity, central hemodynamic parameters and cortisol responses. Furthermore, the extent of the duration of the effects if any at all, measured over 72 h after the AI session to allow future long term intervention studies to discriminate between “true” training adaptations from “last exercise bout” effects.

### **2.7.2 Comparison of acute cortisol response immediately after a single session of AI versus CME**

Glucocorticoids, especially cortisol are part of the endocrine system adaptive response towards physiological and mental stress such as exercise or pre-competition anxiety [38]. In fact, literature suggests that cortisol may be responsible for several observed metabolic and physiological adaptations such as mitochondria biogenesis, improvement in beta-cell function and its proliferation resulting from exposure to stress

(i.e. exercise) and exercise induced enhance lipolytic action in adipose tissue [41, 157, 158]. During exercise, the stimulation of the hypothalamus-pituitary-adrenal, resulting in the secretion of glucocorticoids, plays an essential role in mobilising substrates for the increase metabolic demands [39, 159]. It is clear when individuals are placed under both emotional and physiological stress which can be elicited simultaneously through exercise, cortisol levels are raised, as seen during and after either exhaustive cycling at 156% of  $\dot{V}O_{2max}$  or prolonged cycling (i.e. 50 min) at anaerobic threshold with the latter having a more pronounced effect [160]. Therefore, both exercise duration and intensity mediate the secretion of glucocorticoids during and after exercise. Indeed when exercising at 55% of  $\dot{V}O_{2max}$ , a sustained duration of at least 80 min is required to elicit an elevation in cortisol levels during exercise [161]. Others have reported that a duration threshold of more than 40 min and high-intensity exercise at 76% of  $\dot{V}O_{2peak}$  is required to observe an elevation in cortisol levels during exercise [42]. Consistently, not only did 60 min of cycling at 50% of  $\dot{V}O_{2max}$  not elicit an increase in cortisol levels but further decreased it, however 4 sets of 30 seconds “all-out” supramaximal cycling bouts separated by 5 min of rest after each set elicited an increase in cortisol levels at 10 and 60 min post-exercise [44]. Separately, after a single 30 seconds “all-out” supramaximal cycling bout, cortisol levels were observed to be significantly higher than pre-exercise levels at 20 min post exercise, while cortisol levels were significantly increased immediately after 30 min of cycling at 70% of  $\dot{V}O_{2max}$  [43]. Taken together, we can infer that at low intensity (i.e. <55% of  $\dot{V}O_{2max}$ ), exercise duration needs to be longer than 80 min to elicit an increase in cortisol levels, however with increasing exercise intensity (i.e.  $\geq 70\%$  of  $\dot{V}O_{2max}$ ), this exercise duration threshold becomes shorter (i.e.  $\geq 30$  min).

Therefore, the observation of cortisol levels after an acute bout of exercise provides information on the extent of physiological stress induced by that particular exercise protocol. It is consistent from the extant studies on healthy young males in the literature that a delayed spike in cortisol levels occurring 10 – 20 min can be observed after a SI protocol [43, 44]. Furthermore, while cortisol responses after a CME protocol involving cycling at 70% of  $\dot{V}O_{2max}$  for 30 min is similar to a single sprint, cycling at 50% of  $\dot{V}O_{2max}$  for 1 hour is unable to elicit greater post exercise cortisol levels in comparison to after a SI session consisting of 4 maximal “all-out sprints” interposed

with 5 min of passive rest [44]. Unlike the SIT, the influence of a single AI on cortisol response has not been extensively studied. Furthermore, cortisol response towards exercise stress in obese individuals has been shown to be different to lean individuals, usually more pronounced [45]. In fact, overweight and obese individuals have been characterised with elevated basal cortisol than their lean counterparts [45, 162], however, it can also be lower than their counterparts as observed by others [163]. However, study has shown that 12 weeks of moderate intensity exercise (i.e. 60 – 65% HRR) reduced cortisol levels [164]. In fact, it has been shown that multiple bouts of high intensity interval exercise (i.e. 4 – 5 sets of 2 min at 100 – 110% of  $\dot{V}O_{2max}$ , 2 min at 40%  $\dot{V}O_{2max}$ ) during the day resulted in lower night cortisol levels than multiple bouts of continuous moderate-intensity exercise (i.e. 45 min and 60 min at 65% of  $\dot{V}O_{2max}$ ) [165]. Accordingly, it is likely AIT which can be greater in exercise intensity may be used to reduce elevated cortisol levels found in obese individuals. Therefore, in consideration that AIT is commonly advocated for both the general and clinical populations as it is better tolerated in comparison to SIT and that overweight and obese individuals who tend to be physically inactive and are at risk of developing metabolic diseases would be likely candidates for exercise prescription, more research is required to understand the cortisol response in these individuals elicited after a single AI session.

### **2.7.3 Comparison of exercise preference between HIIT versus CME**

HIIT is a time efficient exercise modality with the potential to be an alternative against the traditionally recommended CME. As mentioned previously, “lack of time” has been consistently proposed as a barrier against the engagement of physical activity and while HIIT can circumvent it, there are other barriers to be considered. Both perceived enjoyment of physical activity and physical activity preference are factors which have been frequently reported to influence physical activity participation and adherence [20, 166, 167]. Taken together, it is imperative that for any exercise modality to be successfully introduced to the general population, it has to be able to overcome these barriers. Drawing insights from the literature, HIIT may receive less aversion as compared to other forms of long-period type vigorous intensity activities, since the interposed low-intensity active or passive recovery bouts may attenuate the

unpleasantness from each high-intensity bouts [168]. At present, not much research has been done towards determining whether HIIT may be perceived as an enjoyable exercise intervention while extant reports are mostly anecdotal evidence. For example, in a previously mentioned SIT study, Richards et al. (2010) evaluated that compliance rate for the study was high due to two contributing factors, mainly the substantial verbal encouragement given and supervision during every training session [129]. In an AIT intervention study on patients with metabolic syndrome, Tjønnå et al. (2008) reported informal comments that participants performing AIT found the varying intensity to be motivating while those performing CME found it “quite boring” to walk continuously [33]. A study by Bartlett et al. (2011) comparing the perceived enjoyment of performing either high-intensity interval (i.e. 3 min at 90%  $\dot{V}O_{2max}$ , 3 min at 50%  $\dot{V}O_{2max}$ ) or continuous moderate-intensity (i.e. 50 min at 70%  $\dot{V}O_{2max}$ ) running using a previously validated “Physical Activity Enjoyment Scale” reported that recreationally active men experienced greater enjoyment performing the former to the latter [2]. However, one must be cautioned against applying this finding to other populations such as those who are morbidly overweight or obese. For example, Sim et al. (2014) found that overweight individuals perceived similar enjoyment from both AI (i.e. 1 min at 100%  $\dot{V}O_{2peak}$ , 4 min at 50%  $\dot{V}O_{2peak}$ ) and CME (60%  $\dot{V}O_{2peak}$ ). This disparity in reported perceived enjoyment between recreationally active and overweight individuals suggests that perception of enjoyment from AIT cannot be generalised between both cohorts. Studies have indicated overweight individuals find physical activity less pleasurable and their level of enjoyment can be further diminished if exercise intensity is above which that they prefer [47]. Therefore, it is imperative that research be invested into determining the optimal AIT protocol such that perceived enjoyment is not compromised for these at risk individuals.

#### **2.7.4 Conclusion: Addressing the gaps**

Physical activity is a modifiable risk factor for obesity and its accompanying comorbidities. It also augments other risk factors which are consequences of unfavourable metabolic changes from adopting a sedentary lifestyle. The clustering of these risk factors is associated with the term “metabolic syndrome”. Recently, HIIT has been

proposed as an alternative exercise modality to the conventional CME. The work to rest intervals, a hallmark feature of HIIT forms the premise for its use in individuals with reduced capacity to exercise since it allows sustained exposure to increase cellular stress, thereby allowing greater physiological adaptations which otherwise would require greater volume and duration for the similar effect to be achieved in convention CME. Importantly, both AIT and SIT have been safely prescribed to various clinical populations despite the increased risk of cardiovascular accident that accompanies the participation of vigorous physical activity. In fact among the different HIIT protocols, reviews in the literature advocates the prescription of AIT to both the general and especially clinical populations. However, there are several gaps pertaining to AIT which need to be addressed. There is an exigency for studies to look at the acute physiological effects from a single AI session to allow for better understanding and discrimination between the “last exercise bout effects” versus training adaptations. Furthermore, comparison between a single AI and CME session is required to highlight the difference, if any on the acute physiological effects on health and metabolic markers, especially since single bout of exercise accumulated over several sessions is just as important as long term adherence to an exercise programme on health. In addition, the observation of cortisol response can provide an illustration of the extent of physiological and emotional stress elicited by an exercise protocol. In fact, the literature indicates that both exercise intensity and duration play important role in mediating cortisol response during and after an exercise bout. Extant studies have examined and compared the effects from a single SI session and CME session on cortisol response; however, the influence from a single AI session and in comparison to a single CME session on cortisol response has not been examined. Further to that, the acute cortisol responses elicited by overweight or obese individuals after a single AI session has not been examined, which is important since this population group is at risk of developing metabolic diseases and thus are prime targets for exercise prescription. Lastly, both exercise intensity and exercise preference play important roles in determining exercise adherence. For overweight or obese individuals, the prescription of a pre-determined exercise intensity that is above their preferred exercise intensity threshold can have negatively affect exercise preference and consequentially reduce exercise adherence. Overweight and obese individuals are prime

targets for exercise prescription as prophylactic measure against metabolic diseases since they are usually physically inactive, however AIT involves brief escalation into high exercise intensities and therefore may affect exercise preference, thus exercise preference for AIT needs to be examined in this particular group of individuals and comparison to be made against CME to determine which exercise modality is more preferred.

In conclusion, we can confidently prescribe AIT to both the general and clinical populations; some of these gaps need to be addressed. By closing these gaps, there can be positive implications for both the research and clinical communities.

## **CHAPTER THREE      METHODS**

### **3.1      Subjects**

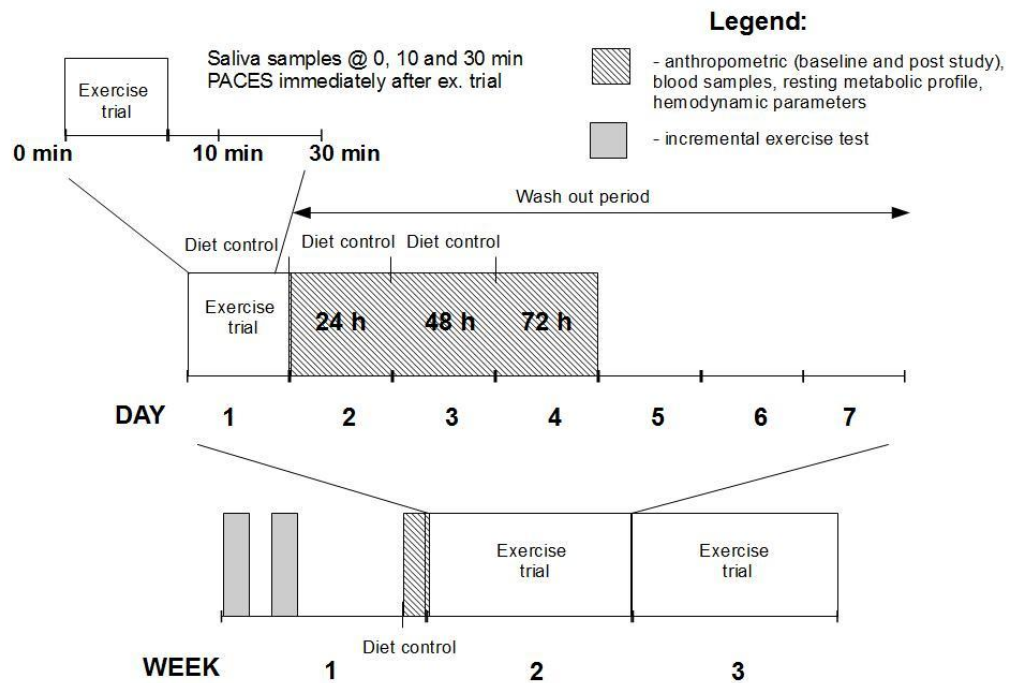
Twelve overweight/obese sedentary males were recruited to participate in this study, however, four dropped out for personal reasons ( $n = 8$ ). Participants were recruited on the basis that they were sedentary (i.e. engaging in less than 1 h/week of structured exercise), aged between 19 to 55 years and had a BMI greater than 25 kg/m<sup>2</sup>. Each participant readiness to participate in physical activity was assessed using the “Physical Activity Readiness Questionnaire” (PAR-Q), created by the Canadian Society for Exercise Physiology [169]. Participants who answered positively to any questions in the PAR-Q were required to fill out a medical questionnaire and obtain medical clearance from a medical practitioner before inclusion into the study. Participants were excluded if they were smokers, currently taking medications that could confound the results of the study and were unable to receive medical clearance from a medical practitioner. The study protocol and procedures were approved by Edith Cowan University’s human research ethics committee. Participants provided written informed consent prior to participation in the study. Throughout the study, participants were requested to maintain their sedentary lifestyle and to refrain from participating in any strenuous physical activity. Furthermore, participants were asked to avoid the consumption of caffeinated and alcoholic beverages 6 h prior to all trials.

### **3.2      Study design**

Participants attended the laboratory on 11 separate occasions to perform, two maximal aerobic capacity tests, two exercise trials and seven measurement sessions (Figure 1). On the first and second visits, participants performed two incremental exercise tests to exhaustion, interposed by 2 days for determination of their peak aerobic capacity. On the third visit, anthropometric and baseline measurements were assessed (described below) and dietary intake 24 h prior was recorded by participants and replicated in the subsequent measurement sessions. In a randomised and



counterbalanced order, participants then performed two exercise trials with a seven days washout period interposed between each trial. These exercise trials involved an AI session and a CME session (described below). After the completion of each exercise trial, participants completed a “Physical Activity Enjoyment Scale (PACES)” questionnaire, to evaluate their perceived level of enjoyment of the exercise trial. The PACES is a valid [170] 18-item questionnaire with each item having a 7-point scale (see appendix 2). Saliva samples were also collected prior to, at 10 and 30 min post exercise trial. At 24 h, 48 h and 72 h following each trial, participants returned to the laboratory for measurements which were similar to that performed at baseline (described below). Anthropometric measurements (only DEXA assessments) were taken again at post 72 h of the last exercise trial.



**Figure 1.** Schematic illustration of the study design.

### **3.2.1 Incremental exercise test**

Participants performed the incremental cycling tests on an electronically braked cycle ergometer (Velotron Pro, RacerMate, Seattle, WA). Each participant performed the test twice and the highest power output elicited between both tests was used to determine the workloads for both exercise trials (described below). The test commenced at a workload of 30 W which gradually increased by 3 W every 12 s (i.e. 15 W every min). Participants continued cycling against the increasing workload until either volitional exhaustion or a cadence greater than 50 rpm could no longer be maintained. Verbal encouragement was given throughout the protocol. Gas exchange was assessed throughout the test using a TrueOne® gas analyser (Parvo Medics, Sandy, Utah USA) which was previously verified [171]. Before all testing sessions, the gas analyser was calibrated using alpha gases of known concentration and the volume transducer was calibrated using a 3 L syringe (Hans Rudolph, Kansas, MO).  $\dot{V}O_{2\text{peak}}$  was established as the highest  $\dot{V}O_2$  value recorded over 15 s average. The peak power output (PPO) was defined as the workload corresponding  $\dot{V}O_{2\text{peak}}$ . Heart rate (HR) (Polar S610, Polar, Finland) and rating of whole body perceived exertion (RPE) (Borg's RPE scale, 6 - 20) were recorded at 2 min intervals during the test.

### **3.2.2 Exercise trials**

Participants performed the two exercise trials on the Velotron cycle ergometer. Both were designed to match for trial duration (CME: 40 vs. AI: 42 min) and total work (CME:  $256.5 \pm 42.6$  vs. AI:  $253.3 \pm 42.1$  kJ), while allowing differences in exercise intensity between trials. Briefly, in the CME bout; participants cycled for 40 min at 50% of their individual PPO, determined during the incremental exercise test. The intensity and duration for this trial was based on previous research and chosen to mirror the minimum intensity and duration of conventional exercise prescription guidelines [74]. During the AI bout, participants completed a total of 13 interval repetitions of 1 min at 85% of their individual PPO. Each repetition was interposed with 2 min of low intensity cycling at 30% of their individual PPO. Prior to the high-intensity efforts participants performed a 3 min warm up at 30% of their individual PPO. The total exercise time for this trial was 42 min.

Total work performed during each trial was calculated based upon the following formula:

$$\text{Work (J)} = P \times t$$

Where P is the power output calculated from the percentage of the PPO (W) and t (min) is the total time during which the power output was elicited. Total energy expenditure for the high-intensity bout included the warm-up.

Throughout both exercise trials, HR (Polar S610, Polar, Finland) and RPE (Borg's RPE scale, 6-20) were recorded at rest, 4 min and every 6 min thereafter (i.e. the end of every second interval). Throughout both trials, participants were provided with continuous feedback on time and their power output. Participants were also allowed to consume water ad libitum.

### **3.2.3 Measurements**

#### **3.2.3.1 Blood sampling and analysis**

Venous blood samples were collected by certified phlebotomist at a commercial pathology laboratory following a 12 h fast. Fasting blood samples were collected between 8:00am and 10:00am. During blood sampling, blood was drawn into fluoride oxalate and serum separator vacutainers (Vacutainer; BD, Oxford, United Kingdom). Serum separating tubes were left to clot for 30 min at room temperature. All samples were centrifuged at 3000 rev/min for 10 min at ambient temperature. Plasma and serum samples were immediately analysed for glucose, triglycerides and cholesterol, while additional serum samples were frozen at -80°C and later analysed for insulin. Commercially available kits were used for the analysis for triglycerides, total cholesterol and HDL (ADVIA Chemistry; Siemens, Munich, Germany) on an automatic analyser (ADVIA Chemistry 2400; Siemens, Munich, Germany). LDL was estimated from measured triglycerides and total cholesterol levels using the Friedewald equation, however where triglycerides are > 4.5 mmol/L, derived values are unreliable and therefore were omitted as was for one participant [172]. Insulin was determined using an

enzyme-linked immunoassay (EIA) (Immulite® 2000 Insulin, Siemens, Munich, Germany) on an automatic analyser (Immulite XPI, Siemens, Munich, Germany). Insulin values below the instrument limits of detection were reported as the lowest possible level of detection (2 mU/L). All analyses for each sample were performed in duplicate.

### **3.2.3.2 Saliva collection and analysis**

Participants did not have any dental surgery or wearing gum shields 48 hrs prior to saliva collection and refrained from brushing or flossing their teeth, 45 min prior to their arrival at the exercise laboratory [173-175]. There were no oral lesions present during the time of testing [173]. At least 10 min before sampling, participants rinsed their mouth with water and swallowed the first pool of accumulated saliva [173]. The passive drool method was utilised to collect unstimulated saliva, which involved directing pooled saliva through a polypropylene drinking straw into 2 mL cryovials (Salimetrics, State College, PA, USA) by getting participants to tilt their head slightly forward with eye open while making minimal orofacial movement [176]. Collected saliva samples were immediately stored at -80°C until further analysis [173-177]. Salivary cortisol was analysed using commercially available EIA kits (Salimetrics, State College, PA, USA).

### **3.2.3.3 Insulin resistance**

Insulin resistance (IR) was estimated using the HOMA2-IR. Assuming healthy normal-weight subjects who are <35 years old, with 100% β-cell function and an insulin-resistance of 1, approximation of the insulin resistance and β-cell function in a patient can be calculated using the plasma concentration of both glucose and insulin at fasting basal level using the following formulas [178]:

$$HOMA - IR = \frac{Glucose \times Insulin}{22.5}$$

The model has been previously validated against euglycaemic hyperinsulinaemic clamp protocol and has been shown to correlate with insulin sensitivity in both healthy

( $R_s=0.83$ ,  $p<0.01$ ) and diabetic individuals ( $R_s=0.92$ ,  $p<0.01$ ) [178]. Similarly, the HOMA-IR index also correlates well with the euglycaemic hyperinsulinaemic clamp protocol in obese and older (i.e. 57 years old) individuals [179]. The coefficient of variation for HOMA-IR function has previously been reported to be 34% [178]. The HOMA2 model provides non-linear solutions, accounts for variations in hepatic and peripheral glucose resistance, with the insulin secretion curve modified to allow for an increase in insulin secretion in response to a plasma glucose concentration of  $>10$  mmol/L and allows for use with newer assays [180].

### **3.2.3.4 Anthropometric assessment**

Both body mass (measured to the nearest 0.01 kg) and height (measured to the nearest 1.0 cm) were measured using an electronic scale and stadiometer (seca763, seca, Germany). Waist circumference was measured in a horizontal plane, midway between the inferior margin of the ribs and the superior border of the iliac crest [127]. Waist circumference was measured to the nearest 1.0 cm. Body fat percentage, lean tissue mass and body fat mass were determined from a whole body scan using dual-energy X-Ray absorptiometry (DEXA) (Hologic Discovery A, Waltham, MA).

### **3.2.3.5 Resting metabolism and substrate utilisation**

Participants rested in a supine position on a bed, in a private and dimly illuminated room with temperature maintained between 22°C to 25°C for 10 min. Thereafter, pulmonary gas exchange was continuously measured and averaged each min over 20 to 40 min using a TrueOne® gas analyser (Parvo Medics, Sandy, Utah USA) [1, 181]. A 5 min steady state interval was used for the determination of energy expenditure. Only data with respiratory quotient within the physiological range of 0.67 to 1.3 was used [182]. Heart rate was measured simultaneously using telemetry (Polar S610, Polar, Finland). Resting metabolic rate (RMR), estimated resting energy expenditure (REE), CHO oxidation rate ( $M_{CHO}$ ) and lipid oxidation rate ( $M_{lipid}$ ) were derived from the following equations [181-184]:

$$RMR \text{ (kcal/min)} = (3.941 \times VO_2) + (1.106 \times VCO_2) - (2.17 \times uN_2)$$

$$\text{REE (kcal/day)} = \text{RMR} \times 1440 \text{ min/day}$$

$$\text{M}_{\text{CHO}} = (4.55 \times \text{VCO}_2) - (3.21 \times \text{VO}_2) - (2.87 \times \text{uN}_2)$$

$$\text{M}_{\text{Lipid}} = (1.67 \times \text{VO}_2) - (1.67 \times \text{VCO}_2) - (1.92 \times \text{uN}_2)$$

Urinary nitrogen ( $\text{uN}_2$ ) was assumed to be 0.11 mg/kg/min for all metabolic calculations, with reference to previous studies [183, 185].

### **3.2.3.6 Resting hemodynamic parameters**

Following the resting energy expenditure measurement, blood pressure (BP) was measured while participants were still in a supine position. Blood pressure was measured using a cuff sphygmomanometer (Medical Industries Australia, Land Cove, NSW) twice on the right arm with a 5 min interval between assessments, during which pulse wave analysis was performed [186]. Pulse wave analysis was performed using a sphygmocardiogram (SphygmoCor; AtCor Medical, Sydney) and involved the derivation of the central aortic BP through the non-invasive reconstruction of the aortic pulse wave from the radial pulse wave. The radial pulse wave was recorded using a handheld high fidelity tonometer (SPT-301B; Millar Instruments, Houston, TX) placed on the surface of the skin superficial to the radial artery with the application of a light pressure to appanate the radial artery against the radius bone [187, 188]. Both brachial BP measurement and pulse wave analysis were performed twice and the average was reported. The indices generated from the estimated central aortic pulse wave provide an illustration of the hemodynamics between the ventricle-vascular interaction [187]. Briefly, the central augmentation index is adjusted to a HR of 75 beats per min (bpm) ( $\text{Alx@HR75}$ ) is calculated as the difference between the area under the late systolic peak and early systolic peak of the aortic pulse wave as a percentage of the pulse pressure [186]. The subendocardial viability ratio (SEVR) is defined as the percentage of the ratio between the area under during diastole and systole [187]. Derived aortic pressure and augmentation index using SphygmoCor has been previously validated [189-192] and measurements have been shown to be highly reproducible [188, 193, 194].

### **3.3 Statistical analysis**

Statistical analysis was performed using the Predictive Analytic Software (PASW) version 19 (Chicago, IL, USA). All results are presented as mean  $\pm$  standard deviation (SD), unless otherwise stated. Significant differences between exercise trials (CME and AI) and over time were assessed using a two-way repeated measures analysis of variance (ANOVA). Where significance between main effects interaction were identified, the Fisher's LSD post-hoc test was performed. A dependent student's t-test was used to assess for significance in the PACES scores and total work performed between exercise trials. Significance was defined as  $p < 0.05$  for all analyses.

## CHAPTER FOUR RESULTS

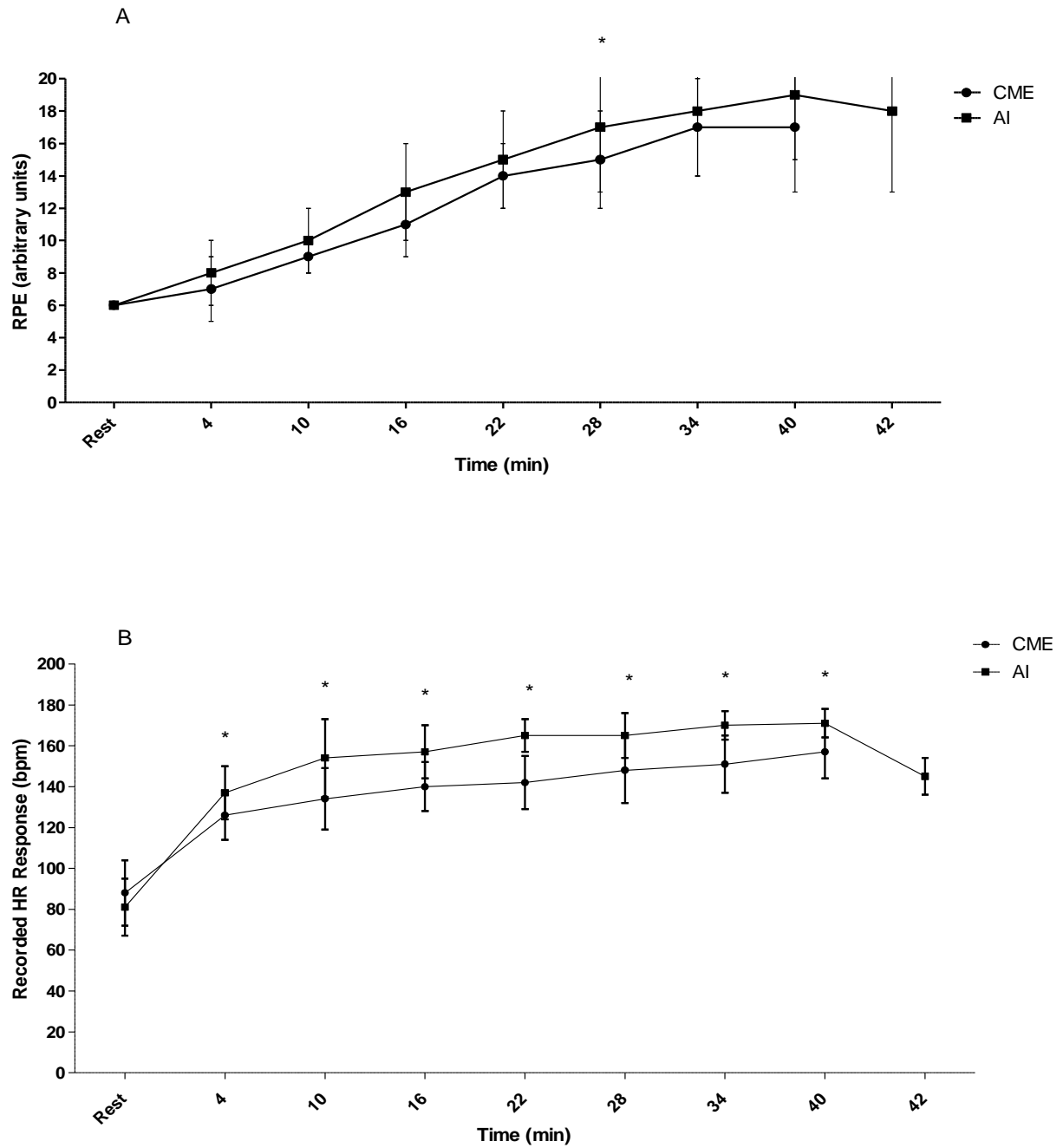
### 4.1 Exercise responses during trials

The demographics for the 8 overweight/obese participants are presented in Table 1. There was a significant difference in the total work performed between exercise trial (CME:  $256.5 \pm 42.6$  vs. AI:  $253.3 \pm 42.1$  kJ,  $p < 0.05$ ), however, the difference is  $3.2 \pm 0.5$  kJ. The RPE was only higher in the AI trial, compared with the CME trial, only at the 28 min time point ( $p < 0.05$ ) (Figure 2A). The HR response to exercise was higher during the AI trial, compared with the CME trial ( $p < 0.05$ ) (Figure 2B).

**Table 1.** Demographics of participants (n = 8)

	Baseline	Post study
	Mean $\pm$ SD	Mean $\pm$ SD
Age (y)	$37 \pm 12$	-
Height (cm)	$174 \pm 6$	-
Weight (kg)	$94 \pm 12$	-
BMI ( $\text{kg}/\text{m}^2$ )	$31 \pm 3$	-
Waist circumference (cm)	$103 \pm 10$	-
Hip circumference (cm)	$105 \pm 6$	-
Waist-hip ratio	$0.99 \pm 0.06$	-
Lean tissue mass (kg)	$65 \pm 8$	$65 \pm 8$
Fat mass (kg)	$27 \pm 6$	$27 \pm 6$
Fat-free mass (kg)	$68 \pm 8$	$67 \pm 8$
Bone mineral content (g)	$2.66 \pm 0.46$	$2.65 \pm 0.46$
Body fat percentage (%)	$28 \pm 4$	$28 \pm 4$





**Figure 2.** Whole body rating of perceived exertion (RPE; A) and heart rate (HR; B) measured during bouts of continuous moderate-intensity (CME) and aerobic interval (AI) exercise. \* denotes significant difference ( $p < 0.05$ ) between trials.

	Baseline	CME			AI		
		24 h post	48 h post	72 h post	24 h post	48 h post	72 h post
<b>Blood metabolites and insulin</b>							
Fasting plasma glucose (mmol/L)	5.36 ± 0.75	5.26 ± 0.54	5.29 ± 0.53	5.56 ± 0.72	5.33 ± 0.54	5.40 ± 0.60	5.34 ± 0.57
Fasting plasma insulin (mU/L)	10.13 ± 6.53	9.75 ± 6.56	7.63 ± 5.18	9.88 ± 8.44	8.88 ± 6.10	10.00 ± 5.71	9.38 ± 5.24
Fasting plasma triglycerides (mmol/L) (n = 7)	1.87 ± 0.91	1.91 ± 1.03	1.66 ± 0.93	1.71 ± 0.93	1.76 ± 0.75	1.51 ± 0.72	1.60 ± 0.75
Fasting plasma total cholesterol (mmol/L)	5.09 ± 0.92	5.31 ± 1.24	5.24 ± 1.03	5.25 ± 1.04	5.16 ± 0.93	5.11 ± 1.04	5.34 ± 1.03
Fasting plasma HDL (mmol/L)	1.25 ± 0.64	1.43 ± 0.71	1.33 ± 0.64	1.16 ± 0.26	1.14 ± 0.28	1.18 ± 0.29	1.16 ± 0.28
Fasting plasma LDL (mmol/L) (n = 7)	3.21 ± 0.84	3.36 ± 1.19	3.39 ± 1.02	3.33 ± 0.90	3.19 ± 0.81	3.26 ± 1.09	3.46 ± 1.02
HOMA2-IR	1.3 ± 0.9	1.3 ± 0.8	1.0 ± 0.7	1.3 ± 1.1	1.2 ± 0.8	1.3 ± 0.7	1.2 ± 0.7
<b>Hemodynamic parameters</b>							
Brachial systolic pressure (mmHg)	133 ± 17	132 ± 19	131 ± 17	131 ± 20	131 ± 20	130 ± 18	130 ± 16
Central aortic systolic pressure (mmHg)	120 ± 20	118 ± 22	118 ± 20	118 ± 23	118 ± 23	116 ± 22	117 ± 21
Subendocardial viability ratio index (%)	173 ± 16	168 ± 19	174 ± 26	170 ± 16	176 ± 21	172 ± 25	172 ± 24
Augmentation index @HR75 (%)	9 ± 12	5 ± 14	8 ± 13	6 ± 14	5 ± 14	4 ± 15	8 ± 11
<b>Resting metabolism</b>							
Resting metabolic rate (kcal/min)	1.24 ± 0.17	1.29 ± 0.07	1.35 ± 0.11	1.35 ± 0.16	1.36 ± 0.11	1.38 ± 0.16	1.36 ± 0.13
Resting energy expenditure (kcal/day)	1787 ± 252	1852 ± 105	1945 ± 151	1949 ± 224	1952 ± 161	1983 ± 237	1959 ± 193
Lipid oxidation rate (g/min)	0.06 ± 0.03	0.06 ± 0.02	0.07 ± 0.02	0.06 ± 0.03	0.07 ± 0.03	0.07 ± 0.03	0.04 ± 0.02

**Table 2.** Blood metabolites and insulin, hemodynamic parameters and resting metabolism measured at baseline, post 24, 48 and 72 h after both aerobic interval (AI) and continuous moderate-intensity (CME) exercise trials. No significant differences in these variables were detected after either trial.

## **4.2 Anthropometric measurements**

Participants' body composition remained similar to baseline at the end of the study. Lean mass, fat mass, fat-free mass, body fat percentage and bone mineral content were similar to baseline (Table 1).

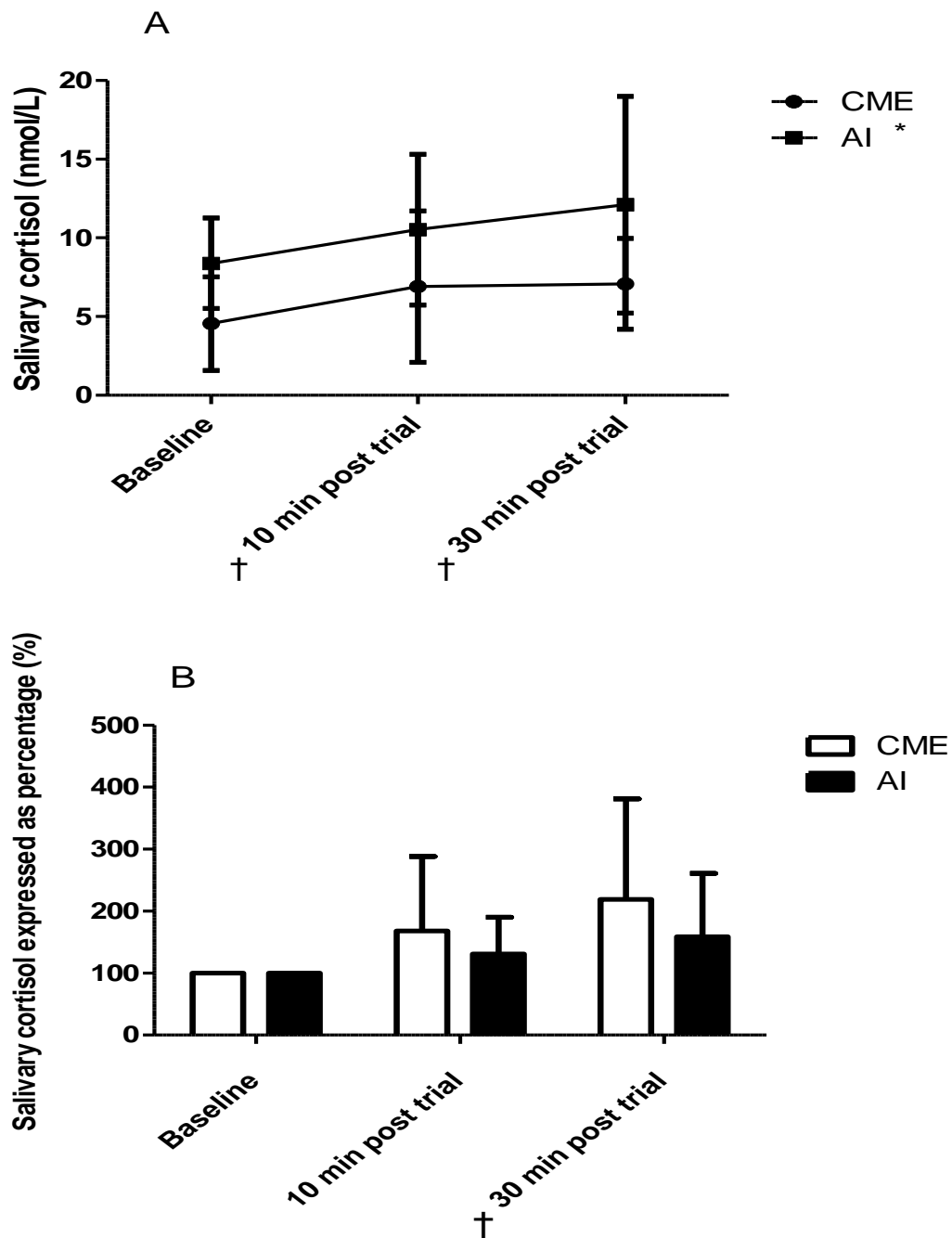
Both exercise trials did not elicit any statistically significant effect on anthropometrical measurements or body composition (Table 1).

## **4.3 Blood metabolites and insulin resistance**

Both exercise trials did not significantly change fasting plasma glucose, insulin, triglycerides, total cholesterol, HDL and LDL. Furthermore, HOMA2-IR index was not significantly different after either exercise trials. The data for these variables is presented in Table 2.

## **4.4 Salivary Cortisol**

Preliminary examination of absolute salivary cortisol response (Figure 3A) showed there was a main effect of trial on salivary cortisol with greater concentration observed in the AI trial compared to CME trial ( $10.06 \pm 1.4$  vs.  $6.17 \pm 1.0$  nmol/L,  $p < 0.05$ ). There was also a main effect of time with absolute salivary cortisol increasing from baseline in both trials (Baseline:  $6.05 \pm 0.67$ , post 10:  $8.71 \pm 1.36$ , and post 30:  $9.59 \pm 1.39$ ,  $p < 0.05$ ). There was no significant interaction between both exercise trials and time points. However, when salivary cortisol was expressed as a percentage relative to baseline level (Figure 3B); it was greater than baseline at 30 post exercise trial ( $189 \pm 35\%$ ,  $p < 0.05$ ) however no significant trial or interaction effects were observed.

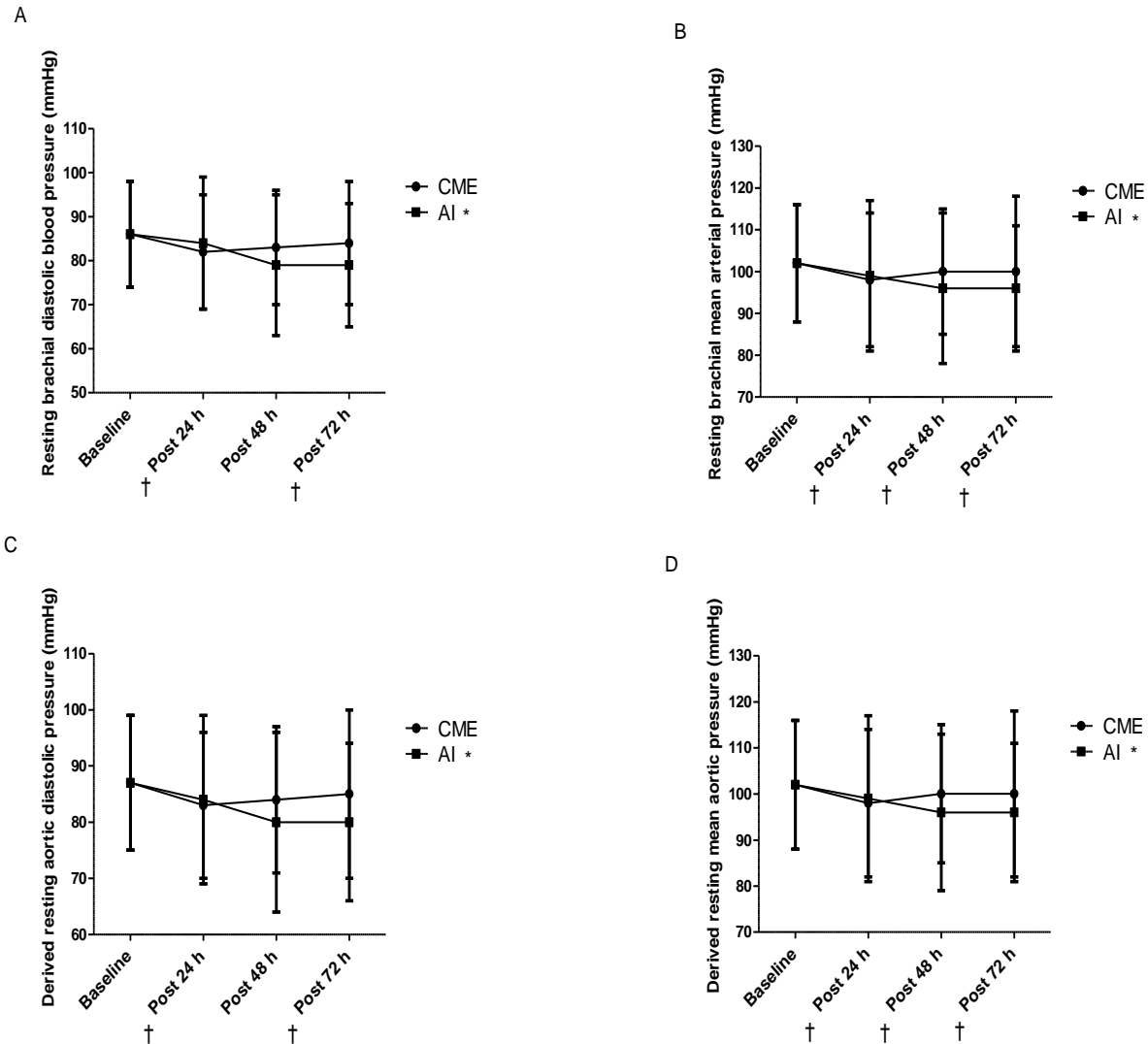


**Figure 3.** Absolute salivary cortisol (A) and salivary cortisol presented as percentage change from baseline (B) measured at post 10 and post 30 min after bouts of aerobic interval, (AI) and continuous moderate-intensity (CME) exercise. No significant interaction effects between trials and time points were observed in both A and B. \* denotes significance ( $p < 0.05$ ) between trials. † denotes significance ( $p < 0.05$ ) between time points. (see text for further illustrations)

## 4.5 Resting hemodynamic parameters

There was a main effect for trial and time in resting brachial diastolic BP (DBP). Resting brachial DBP was lower in the AI trial, compared with the CME trial (AI:  $82 \pm 5$  vs. CME:  $84 \pm 5$  mmHg,  $p < 0.05$ ) (Figure 4A). Compared with baseline, resting brachial DBP was lower at 24 h (baseline vs. 24 h:  $86 \pm 4$  vs.  $83 \pm 5$  mmHg,  $p < 0.05$ ) and 72 h (baseline vs. 72 h:  $86 \pm 4$  vs.  $82 \pm 5$  mmHg,  $p < 0.05$ ) post exercise, in both AI and CME trials. No significant interaction effects were observed for resting brachial DBP ( $p = 0.07$ ). There was a main effect for trial and time for resting brachial mean arterial pressure (MAP). Resting brachial MAP was lower in the AI trial, compared with the CME trial (AI:  $98 \pm 6$  vs. CME:  $100 \pm 5$  mmHg,  $p < 0.05$ ) (Figure 4B). Resting brachial MAP was lower compared with baseline at 24 h (baseline vs. 24 h:  $102 \pm 5$  vs.  $99 \pm 6$  mmHg,  $p < 0.05$ ), 48 h (baseline vs. 48 h:  $102 \pm 5$  vs.  $98 \pm 6$  mmHg,  $p < 0.05$ ) and 72 h (baseline vs. 72 h:  $102 \pm 5$  vs.  $98 \pm 6$  mmHg,  $p < 0.05$ ) post exercise, in both AI and CME trials. No significant interaction effects were observed for resting brachial MAP ( $p = 0.07$ ). There was a main effect for trial and time for derived resting aortic diastolic BP (CDBP). Derived resting CDBP was lower in the AI trial, compared with the CME trial (AI:  $83 \pm 5$  vs. CME:  $85 \pm 5$  mmHg,  $p < 0.05$ ) (Figure 4C). Compared with baseline, derived resting CDBP was lower at 24 h (baseline vs. 24 h:  $87 \pm 4$  vs.  $83 \pm 5$  mmHg,  $p < 0.05$ ) and 72 h (baseline vs. 72 h:  $87 \pm 4$  vs.  $82 \pm 5$  mmHg,  $p < 0.05$ ) post exercise, in both AI and CME trials. No significant interaction effects were observed for resting CDBP ( $p = 0.10$ ). There was a main effect for trial and time for derived resting mean aortic pressure (CMAP). Derived resting CMAP was lower in the AI trial, compared with the CME trial (AI:  $98 \pm 6$  vs. CME:  $100 \pm 5$  mmHg,  $p < 0.05$ ) (Figure 4D). Compared with baseline, derived resting CMAP was lower at 24 h (baseline vs. 24 h:  $102 \pm 5$  vs.  $99 \pm 6$  mmHg,  $p < 0.05$ ), 48 h (baseline vs. 48 h:  $102 \pm 5$  vs.  $98 \pm 6$  mmHg,  $p < 0.05$ ), 72 h (baseline vs. 72 h:  $102 \pm 5$  vs.  $98 \pm 6$  mmHg,  $p < 0.05$ ) post exercise, in both AI and CME trials. There was a trend towards interaction effects for derived resting CMAP, however significance was not reached ( $p = 0.063$ ). There were no significant changes to other resting hemodynamic parameters such as resting

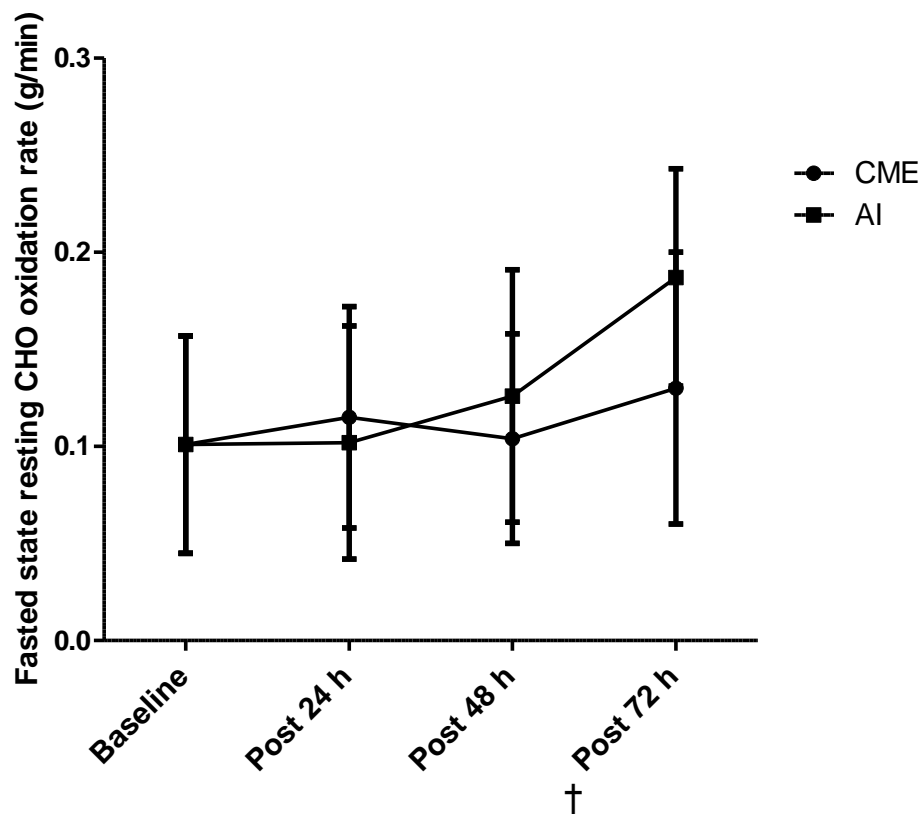
HR, both resting brachial and derived central systolic blood pressure and both SEVR and  $AIx@HR75$  indexes (Table 2).



**Figure 4.** Resting brachial diastolic blood pressure (DBP; A), resting brachial mean arterial pressure (MAP; B), derived resting aortic diastolic BP (CDBP; C), derived resting mean aortic pressure (CMAP; D) measured at baseline, 24 h, 48 h and 72 h post exercise after bouts of aerobic interval, (AI) and continuous moderate-intensity (CME) exercise. No significant interaction effects between trials and time points were observed in A, B, C and D. \* denotes significance ( $p < 0.05$ ) between trials. † denotes significance ( $p < 0.05$ ) at time point vs. baseline. (see text for further illustrations)

#### 4.6 Resting metabolism and substrate utilisation

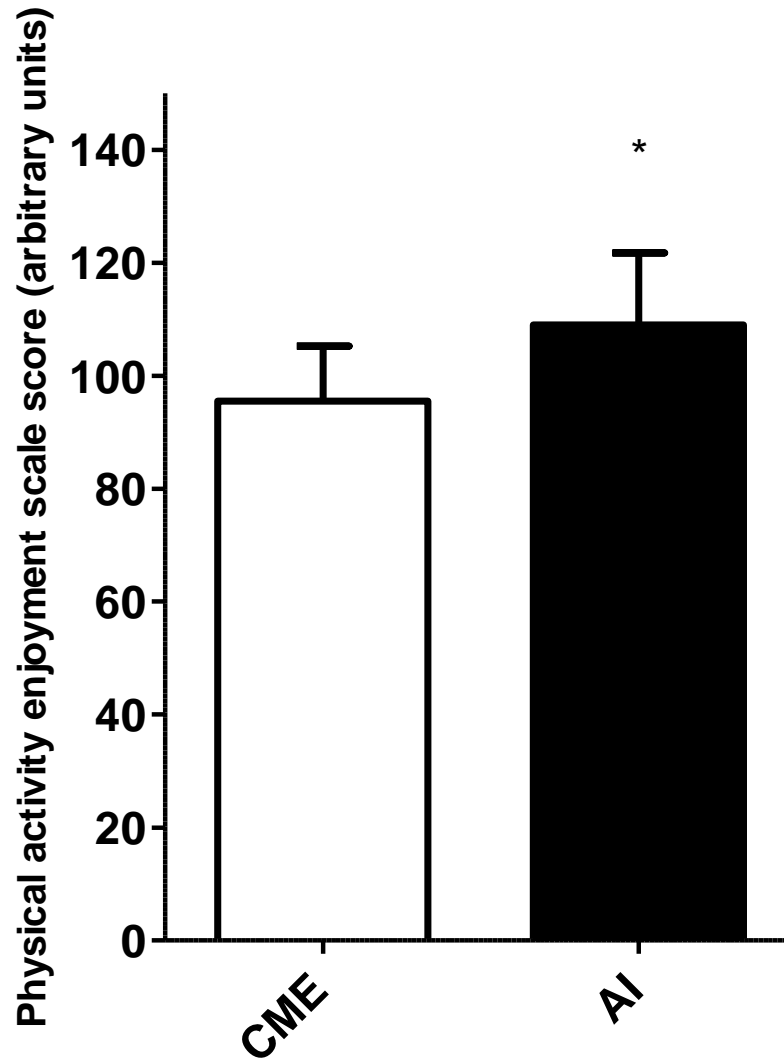
Compared with baseline and 24 h post exercise, fasted state  $M_{\text{CHO}}$  was higher at 72 h post exercise (baseline vs. 72 h:  $0.10 \pm 0.02$  vs.  $0.16 \pm 0.02$  g/min, 24 h vs. 72 h:  $0.11 \pm 0.01$  vs.  $0.16 \pm 0.02$  g/min,  $p < 0.05$ ) in both trials (Figure 5). No significant interaction effects were observed for fasted state  $M_{\text{CHO}}$ . There were no significant changes to resting metabolic rate, resting energy expenditure and lipid oxidation rate (Table 2).



**Figure 5.** Fasted state resting CHO oxidation rate ( $M_{\text{CHO}}$ ) measured at baseline, 24 h, 48 h and 72 h post exercise after bouts of aerobic interval, (AI) and continuous moderate-intensity (CME) exercise, no significant trials and interaction effects were observed. † denotes significance ( $p < 0.05$ ) at time point vs. baseline and post 24 h.

#### 4.7 PACES scores

Participants perceived greater enjoyment in the AI trial as reflected by the higher PACES score, compared with the CME trial ( $109 \pm 13$  vs.  $96 \pm 10$ ,  $p < 0.05$ ) (Figure 6)



**Figure 6.** The physical activity enjoyment scale (PACES) score awarded by participants immediately after bouts of aerobic interval, (AI) and continuous moderate-intensity (CME) exercise. \* denotes significance ( $P < 0.05$ ) between trials.



## **CHAPTER FIVE      DISCUSSION**

There is currently an increase in the incidence of overweight and obesity in developed nations. Obesity can lead to an undesirable clustering of cardiometabolic risk factors leading to metabolic syndrome, which increases the risk of type 2 diabetes and cardiovascular diseases [127, 195]. Aerobic interval training has been suggested to be a prospective alternative to the time intensive continuous moderate-intensity exercise. While the health benefits conferred by long term aerobic interval exercise has been established in both the general and certain clinical populations, its acute physiological effects in overweight and obese individuals is not well known. Understanding these effects is important in the discrimination between training effects and/or the effects of the last/final exercise bout. Furthermore, while recreationally active individuals perceive greater enjoyment from an aerobic interval session compared to continuous moderate-intensity exercise, perceived enjoyment between the two exercise modals does not differ in overweight individuals [2, 48]. In fact, recovery duration between work intervals may also negatively influence perceived enjoyment as previously reported [196]. Such information is likely to be important in improving exercise program adherence and further research is warranted on defining an optimal AIT protocol that does not compromise perceived enjoyment in this population.

### **5.1 Study objectives and primary findings**

The main objectives of this study were to examine and compare: i) the acute physiological effects after a single bout of AI session versus a single bout of matched work and duration CME session on cardiometabolic risk factors such as anthropometric variables, blood metabolites and insulin levels and resting hemodynamic parameters and metabolism and substrate utilisation and finally, ii) the immediate cortisol response after a single AI session versus a single CME session and iii) to determine which of the two exercise modalities is perceived more enjoyable by overweight and obese individuals.

The primary findings from this study are as follows. While we found a difference in total work performed between exercise trials, it was an extremely small (i.e. 3.2 kJ) and therefore unlikely to be responsible for the observed physiological differences between conditions. Both AI and CME trials reduced resting brachial DBP, at post 24 h and 72 h after an acute session. The AI trial elicited a greater reduction in resting brachial DBP. Similarly, derived resting CDBP was lower at post 24 h and 72 h than baseline following AI and CME, however; AI had a more pronounced effect. Both resting brachial MAP and derived resting CMAP were reduced over post 24 h, 48 h and 72 h following both CME and AI trials, however the effect was greater following the AI trial. Fasted-state  $M_{CHO}$  was higher post 72 h versus baseline and post 24 h in both CME and AI trials. Despite HR being higher during the AI trial, RPE scores were similar throughout the trials, except at the 28 min time point. The increase in salivary cortisol at 30 min post exercise was not different between AI and CME trials. Finally, PACES scores were significantly higher in the AI trial, indicating that overweight/obese individuals found the AI session to be more “enjoyable”.

## **5.2 Hemodynamic parameters**

Raised blood pressure has been attributed to 13% of global mortality [4]. Indeed mortality from both stroke and ischemic heart disease doubles with every 20 mmHg increase in systolic or 10 mmHg increase in diastolic BP [197]. The pathophysiological link between abdominal obesity and cardiovascular diseases is well established. Obesity causes left ventricle remodelling, leading to ventricular dysfunction [198]. This can result in diastolic dysfunction (i.e. elevation of diastolic BP) which can happen independently, simultaneous or prior to systolic failure [198]. Indeed, it has been found that mean arterial, systolic and diastolic BP are robust predictors of cardiovascular disease in young men below the age of 50 years old [199, 200]. The cohort in our study had baseline brachial systolic and diastolic BP of  $133 \pm 17$  mmHg and  $86 \pm 12$  mmHg, respectively. Accordingly, they can be classified as pre-hypertensive (i.e. systolic BP: 120 – 139 mmHg, diastolic BP: 80 – 89 mmHg) and are predisposed towards a greater risk of developing cardiovascular diseases [200].

Within the present study we found that a single session of CME or AI can acutely reduce resting DBP, MAP, derived resting CDBP and CMAP for up to 72 h following exercise. Post exercise hypotension can be described by a transient decreased in blood pressure following an acute bout of exercise, which underlying mechanisms have yet to be determined [201]. The magnitude of this phenomenon is more pronounced and consistent in borderline hypertensive [202, 203] and hypertensive [156, 204, 205] than in normotensive [206, 207] individuals [201]. While studies have found that such post exercise hypotension which can last up to more than 12 h [156, 201, 206, 208], this is the first study to our knowledge that has observed such changes for up to 72 h. However, since there was an absence of post exercise hypotension observed at 48 h post exercise, caution should be taken when interpreting these findings. Indeed, the variability of diastolic BP can be mediated by the participants' emotional state [209]. It is also possible that participants may have intermittently slept during resting metabolic measurements, which could have lowered their BP [210-212].

Previously, Ciolac et al. (2009) found that CME (i.e. 40 min at 60% of heart rate reserve) reduced both mean 24 h systolic (i.e.  $-2.6 \pm 6.6$  mmHg) and diastolic (i.e.  $-2.3 \pm 4.6$  mmHg) BP post exercise and night time systolic and diastolic BP (i.e.  $-4.8 \pm 6.4$  mmHg and  $-4.6 \pm 5.2$  mmHg, respectively). However, AI (i.e. 40 min of 1 min at 80% of heart rate reserve, 2 min at 50% of heart rate reserve) only reduced 24 h mean systolic BP post exercise and night time systolic BP (i.e.  $-2.8 \pm 6.5$  mmHg and  $-3.4 \pm 7.2$  mmHg, respectively) without any significant change in diastolic BP [156]. Within the present study both CME and AI were found to reduce BP. However, we observed a significant reduction in diastolic and mean arterial and aortic BP and not in resting systolic and derived systolic BP, which is somewhat contrary to previous studies [156, 206, 213]. Indeed, studies have reported an average reduction in systolic BP by 14 mmHg in borderline hypertensive populations [201]. However, since post-exercise hypotension is negatively correlated with BMI [214], the discrepancies between our study and others may partially be explained by difference in the sampled populations. Our participants had a mean BMI of  $31 \pm 3$  kg/m<sup>2</sup> while other studies have recruited participants with BMI of  $< 25$  kg/m<sup>2</sup> [206, 213]. Although, Taylor Tolbert et al. (2000) observed a decrease in 24 h mean systolic BP (i.e.  $-7.4 \pm 1.8$  mmHg) and which was lower up to 16

h after 45 min of running at 75% of  $\dot{V}O_{2\max}$  in older obese males [205]. Alternatively, a small sample size of 8 could have limited the ability to detect significance in the present study. Further research is needed to better understand the factors that influence post exercise alterations in BP in overweight and obese individuals.

Results of the present study indicate that AI elicited a greater reduction in BP. These findings are supported by the study of Jones, Taylor, Lewis, George and Atkinson (2009) who reported that intermittent exercises exerts a more pronounced post-exercise hypotension effect compared with a single bout of isocaloric CME [213]. This suggests that AI may be considered as an alternative exercise intervention to CME in mitigating hypertension on a daily basis. However, more studies are required to examine blood pressure changes during AI and further substantiate our findings before recommendation. Taken together, these results have implications for the measurement and interpretation of post training changes in BP and exercise prescription for reducing risk for cardiovascular diseases.

### **5.3 Blood metabolites, insulin, HOMA2-IR and resting metabolism**

Metabolic syndrome is usually characterised by dyslipidaemia and impaired glucose homeostasis, all of which increases cardiometabolic risks. Exercise is a cost-effective intervention that can mitigate hyperglycaemia. In the present study however, we did not observe a significant change in HOMA2-IR, fasting insulin and glucose following either the AI or CME trials. These data indicate that both exercise protocols had little influence on acute insulin resistance fasting glucose homeostasis. Jarmurtas et al. (2006) reported that in overweight males, glucose, insulin and insulin resistance were not different from baseline at 24 h and 48 h after an acute bout of aerobic exercise (i.e. 65% of  $\dot{V}O_{2\max}$  for 45 min) [215]. Despite the difference in exercise intensity, our findings are also similar. Interestingly, Jarmurtas et al. (2006) detected a significant decrease in insulin and insulin resistance immediately post exercise [215]. Together with our findings, we can infer that while an acute bout of exercise can immediately reduce insulin and insulin resistance, both these variables will likely return to pre-exercise levels within 24 to 48 h after exercise. Contrary to this, a previous study found that

aerobic exercise (i.e. 75% of  $\dot{V}O_{2peak}$  for 45 min with two bouts of 2 min rest interspersed after every 15 min) is superior to an acute bout of sprint interval session in eliciting significant improvement in both the HOMA and composite whole-body insulin sensitivity indexes, 24 h after exercise [150]. Similarly, Whyte et al. (2010) found significant improvement in whole-body insulin sensitivity index following 2 weeks of SIT in overweight and obese participants. However, in this study this change did not persist beyond 24 h and therefore is likely to be associated with an acute change in insulin sensitivity resulting from the ‘last exercise bout’ effect. It is possible that differences between the present study and the findings of previous research are the result of differences in exercise protocols. In the present study participants performed matched work and duration bouts of AI and CME, whereas the aforementioned studies have performed exercise for longer durations at a high-intensity or shorter but at supramaximal intensities. As such, further research is needed in order to understand the acute effects of exercise intensity and duration on insulin sensitivity.

In addition to improving insulin sensitivity, it has also been found that acute exercise can alter blood lipid profile. For instance, it was previously observed that in men with hypercholesterolemia, following acute bouts of isocaloric high (i.e. 80% of  $\dot{V}O_{2max}$ ) and moderate (i.e. 50% of  $\dot{V}O_{2max}$ ) intensity exercise bout, total and LDL (i.e. 4%) dropped and subsequently rose (i.e. 5 – 8%) by 48 h [216]. In the similar study, triglycerides were lower at 24 h (i.e. -18%) and 48 h (i.e. -15%) and HDL rose (i.e. 8 – 9%) following exercise bouts [216]. In another study, Cullinane, Siconolfi, Saritelli and Thompson (1982) also reported a decrease in triglycerides levels in both trained and sedentary individuals, although values did not reach statistical significance, after 1 h of exercise bout [217]. In our study, after removal of an outlier, triglycerides decreased over 24 to 72 h (Table 2), however, changes were not statistically significant. Likewise, neither AI nor CME significantly influenced HDL, LDL or total cholesterol. The small sample size in our study, could have limit the power to detect significance. Therefore, future study would need to take into account a larger sample population if they are examining the influence of exercise on lipid metabolites.

Impaired lipid oxidation is typically observed in obese individuals and exercise has been postulated to be an effective way in improving lipid oxidation in these individuals since previous studies have demonstrated increase lipid oxidation during post-exercise recovery in healthy adults [218, 219]. Indeed, two weeks of sprint interval training in obese individuals has been shown to increase lipid and decrease carbohydrate oxidation rates, however changes did not persist past 24 h, therefore, authors alluded that it may be due to the “last exercise bout” effect as opposed to being a training adaptation [1]. Therefore, if assuming that the change in substrate oxidation rates observed by Whyte et al. (2010) were “last exercise bout” effects, then their observations are contrary to our findings since we observed a significant increase in carbohydrate oxidation rate only at 72 h after the last exercise bout with no effect on lipid oxidation rate [1]. Exercise can acutely increase lipid oxidation rate. For instance, Warren, Howden, Williams, Fell and Johnson (2009) observed similar increase in lipid oxidation rate (i.e. over 90 min) and total fat oxidation between CME (i.e. 90 min at 50% of  $\dot{V}O_{2max}$ ) and AI (i.e. 1 min at 85%  $\dot{V}O_{2max}$ , 2 min at 30% of  $\dot{V}O_{2max}$ ) [74]. Our findings indicate that any increment in lipid oxidation rate will not last up to 24 h. In addition, previous studies have observed a significant shift towards greater carbohydrate oxidation rate, 15 to 20 min post exercise (i.e. 50% of  $\dot{V}O_{2max}$ ) in overweight boys and a pronounced increase in carbohydrate oxidation rate but not lipid oxidation after 16 weeks of aerobic (i.e. 70% of  $\dot{V}O_{2max}$ , 3 days per week) in obese women [220, 221]. Together with our findings, which are somewhat similar, it is plausible that in overweight and obese individuals, there is an exercise induced shift towards preferential carbohydrate utilisation which is independent of exercise intensity. Furthermore, we have shown that this can occur after an acute bout of exercise and can become evident 72 h later. Future studies examining exercise induce changes in substrate utilisation in overweight and obese individuals should take into account a longer observation period.

Our observation that neither a single session of AI or CME has a significant effect on resting energy expenditure over 72 h is consistent with that observed in the study by Saris and Schrauwen (2004), who reported that either isocaloric low (i.e. 60 min at 38% maximal aerobic power) or high (i.e. 2.5 min of 80% and 50% maximal aerobic power using 1:1 work to rest intervals) intensity protocols did not significantly

affect 24 h energy expenditure or respiratory quotient in obese men [222]. Together, these findings indicate that despite the high intensity bouts in AI, compared with a matched work and duration bout of CME, both exercise modalities do not affect energy expenditure and respiratory quotient 24 h following exercise in overweight and obese individuals.

## 5.4 Cortisol response

Glucocorticoids play an important role in facilitating substrate utilisation as an adaptive response when under either physical or emotional stress [39]. In this study, we examined and compared cortisol response over 30 min post exercise in overweight and obese individuals after they have performed a single bout of AI and CME on separate occasions. We measured cortisol response using salivary cortisol as it detects biologically active free fraction cortisol and is not limited by the saturation point of cortisol binding globulin (CBG), since exercise stress can raise absolute cortisol above CBG saturation point [223, 224]. Absolute cortisol was significantly greater in the AI trial and was significantly greater than baseline at 10 and 30 min post exercise in both AI and CME. However, since baseline cortisol in the AI trial was greater than the CME trial, we decided to analyse the change in cortisol relative to the baseline of each respective trial. We then observed that both an acute bout of CME and AI similarly elicited pronounced elevation of cortisol 30 min post exercise with no difference between protocols. It should also be noted that cortisol level was not significantly elevated at 10 min post exercise. Jamurtas et al. (2006) reported that an acute bout of CME bout (i.e. 65% of  $\dot{V}O_{2max}$  for 45 min) in overweight males did not significantly affect cortisol levels immediately post exercise [215]. The CME protocol used in our study involved a lower intensity and shorter duration (i.e. 50% of PPO for 40 min) compared to the protocol employed by Jamurtas et al. (2006), yet we found significant increase in salivary cortisol levels 30 min post exercise. While it is plausible that measuring for serum cortisol may have affected the likelihood of detecting any significant changes due to its lower sensitivity, it is also likely that if cortisol was measured over a longer time period post exercise, the authors would have detected

significant fluctuations in cortisol levels. Furthermore, the findings from our study is consistent with that observed by Wong and Harber (2006) who reported that cortisol levels in obese individuals after performing an acute exercise bout (i.e. 30 min CME at power output corresponding to ventilatory threshold) were elevated over time, peaking significantly between approximately 30 to 50 min post exercise [45]. Therefore, we suggest that studies intending to examine exercise induced cortisol response should consider measuring for cortisol fluctuation over a time course of up to 30 min at least to allow for the delayed spike in cortisol response immediately post exercise.

Exercise-induced cortisol response is predominantly mediated by exercise intensity and duration, specifically over extended duration [42]. When matched for work output (i.e. 120 – 180 kJ) and duration (i.e. 20 min) between exercise types, Vanhelder, Radomski, Goode and Casey (1985) reported that only both resistance exercise and intermittent cycling elicited pronounced increase in cortisol levels, while cortisol levels were not affected after continuous cycling [225]. Our findings are contrary to that of Vanhelder et al. (1985) such that both CME and AI trials elicited similar increases in cortisol levels [225]. Rudolph and Mcauley (2010) reported that RPE and post exercise cortisol responses are positively correlated while McGuigan, Egan and Foster (2004) observed that both salivary cortisol and RPE responses correctly reflect the prescribed exercise intensity of resistance training protocols [226, 227]. In our study, participants reported almost similar RPE responses except at the 28 min time point in both CME and AI trials. From this alternative perspective, we examined the mean exercise intensity of the AI trial, in accordance to Saltin, Essen and Pedersen (1976), and found that it is 48% of PPO [228]. This is close to the exercise intensity we prescribed for the CME (i.e. 50% of PPO) trial. Therefore, if exercise intensity modulates cortisol levels, the similarity in exercise intensity between protocols could explain why we did not find any difference in post exercise cortisol response between both trials.

## **5.5 Perceived “enjoyment” of the exercise trials**

It is clear that despite significant differences in HR responses between CME and AI trials in the present study (Figure 2), participants’ ratings of perceived exertion did



not differ throughout the majority of the trials. Therefore, we can infer that the AI trial is well tolerated in obese or overweight individuals despite eliciting greater HR response in these individuals. Thereby, AI may provide an exercise mode that allows overweight or obese participants to train “comfortably” at higher exercise intensity intermittently. Furthermore, perceived enjoyment of activity collected from PACES questionnaire was significantly higher for the AI trial, indicating a preference for it over the CME trial. This is consistent with a recently published study, which also reported that recreationally active men preferred high-intensity interval (i.e. 3 min at 90%  $\dot{V}O_{2max}$ , 3 min at 50%  $\dot{V}O_{2max}$ ) over continuous moderate-intensity (i.e. 50 min at 70%  $\dot{V}O_{2max}$ ) [2]. However in a study by Sim et al. (2014), authors reported that perceived enjoyment in overweight individuals did not differ between either AI (i.e. 1 min at 100%  $\dot{V}O_{2peak}$ , 4 min at 50%  $\dot{V}O_{2peak}$ ) or CME (60%  $\dot{V}O_{2peak}$ ) [48]. We postulate that the disparity in findings could be due to the difference in mean exercise intensity between our AI protocol and that of Sim et al. (2014) (i.e. 48% PPO vs. 60%  $\dot{V}O_{2peak}$ ), however further research is warranted to determine to AI protocols with different mean exercise intensities have a negative influence on perceived enjoyment in overweight and obese individuals [48].

Indeed, adherence to physical activity can be reduced in overweight or obese individuals when exercising at higher than preferred exercise intensity; however this study has corroborated with a previous finding that having lower intensity recover bouts interspersed between high-intensity work bouts can reduce perceived aversion of exercise [168]. The finding from this contributes to the literature by suggesting that AI is well tolerated in overweight and obese individuals and further substantiates previous anecdotal reports that high-intensity interval exercise is more preferred. These results have implications for the prescription of exercise in such populations.

## 5.6 Limitations

A major limitation of this study is its small sample size. A small sample due to pragmatic considerations such as both budget and time constraints would have decreased the power of the study. This holds true especially for blood metabolites and HOMA-IR2 measured in this study, most of which the observed powers were less than 0.1 for

between trials analysis, which could partly explain the inconsistencies in detecting significant changes compared to previous studies. Furthermore, it has also resulting in the inability to detect interaction between the main effects, making interpretation of the results challenging. Therefore, future prospective studies should consider the use of a larger sample size. In addition, the rationale for employing the AI protocol used in our study was so that it is tolerable and safe, since we recruited untrained and sedentary overweight and obese individuals to perform an acute bout of exercise. However as mentioned previously, this resulted in the mean exercise intensity of the AI and exercise intensity of the CME trials to be somewhat similar. This could have partly contributed to why we did not detect significant differences in many of the metabolic and health markers we measured. It is possible that greater differences in exercise intensity resulting from SI interval training or higher intensity AI are needed in order to observe substantial differences in physiological response compared to CME. As such, further research is warranted to determine if a higher mean exercise intensity in the AI trial could elicit greater changes in both health and metabolic markers in comparison to a lower exercise intensity CME protocol.

## **5.7 Practical implications**

This study has attempted to elucidate the “last exercise bout” effect arising from a single session of CME and AI. Pertaining to the findings from this study, either CME or AI can elicit a “last exercise bout” effect on both diastolic or its derived central parameters and carbohydrate oxidation rate up to 72 h, therefore, future training studies should consider scheduling more than 72 h for post training measurements especially if both the abovementioned variables are measured. In addition, cortisol responses should be sampled over a time period of at least 30 min for an accurate determination of post exercise cortisol response. Furthermore, the findings in our study have corroborated with extant studies that RPE and post exercise salivary cortisol response can be used to determine the intensity of an exercise protocol. Most importantly, we have demonstrated that aerobic interval cycling is preferred over continuous moderate-intensity cycling and

thus can be prescribed as an alternative to these individuals, with the likelihood of possibly higher adherence rate.

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## CHAPTER SEVEN

## APPENDICIES

	<b>Appendix</b>	<b>Page</b>
A	Physical activity readiness – questionnaire	74
B	Physical activity enjoyment scale	75
C	Daily food and physical activity diary	76
D	Informed consent form	77
E	Medical questionnaire form	84

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

# PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

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

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

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

YES	NO	
<input type="checkbox"/>	<input type="checkbox"/>	1. Has your doctor ever said that you have a heart condition <u>and</u> 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 <u>any other reason</u> 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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**Physical Activity Enjoyment Scale**

Subject Tag no.: \_\_\_\_\_

Exercise Protocol: \_\_\_\_\_

Please rate how you feel at the moment about the physical activity you have been doing.

I enjoy it	1	2	3	4	5	6	7	I hate it
I feel bored	1	2	3	4	5	6	7	I feel interested
I dislike it	1	2	3	4	5	6	7	I like it
I find it pleasurable	1	2	3	4	5	6	7	I don't find it pleasurable
I am very absorbed in this activity	1	2	3	4	5	6	7	I am not at all absorbed in this activity
It's no fun at all	1	2	3	4	5	6	7	It's a lot of fun
I find it energizing	1	2	3	4	5	6	7	I find it tiring
It makes me depressed	1	2	3	4	5	6	7	It makes me happy
It's very pleasant	1	2	3	4	5	6	7	It's very unpleasant
I feel good physically while doing it	1	2	3	4	5	6	7	I feel bad physically while doing it
It's very invigorating	1	2	3	4	5	6	7	It's not at all invigorating
I am very frustrated by it	1	2	3	4	5	6	7	I am not at all frustrated by it
It's very gratifying	1	2	3	4	5	6	7	It's not at all gratifying
It's very exhilarating	1	2	3	4	5	6	7	It's not at all exhilarating
It's not at all stimulating	1	2	3	4	5	6	7	It's very stimulating
It gives me a strong sense of accomplishment	1	2	3	4	5	6	7	It doesn't give me a strong sense of accomplishment
It's very refreshing	1	2	3	4	5	6	7	It's not at all refreshing
I felt as though I would rather be doing something else	1	2	3	4	5	6	7	I felt as though there is nothing else I would rather be doing

### Daily Food and Physical Activity Diary

Name: \_\_\_\_\_

Date: \_\_\_\_\_



#### Dietary Intake

Morning		Afternoon		Evening - Supper	
Serving	Item	Serving	Item	Serving	Item



#### Physical Activity

Type of Physical Activity	Duration

Physical Activity -

Any bodily movements produced by skeletal muscles that results in energy production (Includes structured exercise and any daily activities eg. Walking to working or gardening)

**INFORMATION LETTER TO PARTICIPANTS**



**Comparison of the acute effects between aerobic interval versus moderate-intensity continuous cycling bouts**

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**Chief Investigator: Derek Koh Fujie**

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Thank you for your expression of interest. This document provides you with details relevant to the study in which you may participate in. Please read all information contained within this letter carefully, and feel welcome to contact the chief investigator if you have any questions or concerns you wish to raise. You will be required to fill out the informed consent form attached at the end of this letter before you can begin participation in this study.

The study is being conducted by researchers from the School of Exercise and Health Sciences, in the Faculty of Computing, Health and Science at Edith Cowan University:

- Mr Derek Koh Fujie (Masters student)
- Prof. Rob Newton, Professor of Exercise and Sports Science
- Dr. Chris Abbiss, Senior Lecturer
- Dr. Michael Baker, Postdoctoral Research Fellow in Exercise and Chronic Disease.

**Description of study**

The main aims of this study are to compare the effects between a single bout of aerobic interval training and moderate-intensity continuous cycling and to understand the post-exercise effects of each of these exercise protocols over 72 hours. A better understanding of these effects will help inform future studies on how to better manage and prevent chronic diseases such as diabetes and heart disease. The whole time course for this study is approximately 3 weeks where you will visit the Joondalup campus of Edith Cowan University on 11 occasions (refer to figure 1 in the "Appendix" for a schematic description of the study):

- Pre-data collection and familiarisation phase (Week 1)
- Baseline testing, commencement of first exercise protocol followed by washout period (no structured exercise) (Week 2)
- Second exercise protocol and laboratory testing (no structured exercise) (Week 3)

\* Washout period – Period which participants are asked to refrain from participating in any structure exercise or straining physical activity and would not be participating in any testing or exercise trials in the study. This is to "clear" up any residual effect from previous exercise protocol.

#### Required participant involvement

You have been identified as a potential participant for the study because:

- you are between the ages of 19 - 55 years old
- you have a BMI of  $\geq 25 \text{ kg/m}^2$  or waist circumference of  $>94 \text{ cm}$
- you are leading a sedentary lifestyle (participating in  $<1 \text{ h/wk}$  of structured exercise)

You will be excluded from the study however if:

- you are a smoker;
- you are taking medications or have any medical conditions that could prevent participation in the exercise protocol.
- you have answered positively to any question in the Physical Activity Readiness Questionnaire (PAR-Q) or are unable to receive medical clearance for physical activity by a medical practitioner.

As a participant you will be required to attend 1 familiarisation, 1 exercise testing, 2 exercise protocols and 7 data collection sessions throughout the course of the study. Furthermore, you will be required to standardise your diet on days prior to fasting measurements and exercise trials and to refrain from participating in any physical activity throughout the study. You will also be required to refrain from alcohol and caffeine consumption 6 hours prior to any exercise or laboratory sessions.

We will assess your height and weight, fasting blood plasma content (insulin, glucose, and lipid profile), salivary steroid levels, aerobic fitness, body composition, condition of your arteries and fasting metabolic (lipid and carbohydrate) changes. Your dietary and physical activity throughout the study will be monitored before each visit using a food and physical activity diary.

The following instruments or equipments will be used during the study:

- heart rate monitor
- sphygmomanometer (measures blood pressure)
- sphygmocardiogram (measures arterial vessel stiffness)
- dual-energy x-ray absorptiometry (DEXA) (measures body composition)
- gas analyser (measures volume and concentration of inspired and expired gases)
- perceptual scales
- questionnaires (PAR-Q, medical, Physical Activity Enjoyment Scale and food and activity diary)
- venipuncture (see below)
- saliva collection kit

All exercise and laboratory sessions will be performed within ECU sports physiology laboratory located in 19.150. All blood sampling sessions will be performed by certified phlebotomists at either ECU sports physiology laboratory or Western Diagnostic Pathology collection centre located within Joondalup Health Campus.

#### Exercise Protocols

##### Maximal Aerobic Capacity Test

An incremental ramp cycling protocol will be used for this test, where the workload will be progressively increased during the test. You will commence cycling at a workload of 60 W, which will subsequently be increased 15 W every minute (3 W every 12 s). You will be required to cycle against the increasing workload until volitional exhaustion or when you cannot continue at the required pace.

##### Continuous moderate-intensity cycling

You will be required to cycle continuously for 40 minutes on an electronically-braked (workload remains constant regardless of the cadence) cycle ergometer with the workload set to a constant equivalent to that of  $\sim 50\%$  of the maximal work output at maximal aerobic capacity ( $\text{VO}_{2\text{max}}$ ).

#### Aerobic interval cycling

An electronically-braked cycle ergometer will be used for this protocol. You will commence by cycling for 3 minutes at ~30% of your work output at  $VO_{2max}$ , followed by 13 interval repetitions of cycling at 85% of  $VO_{2max}$  for 1 minute, interspersed with an active recovery bout of cycling at ~30% of your work output at  $VO_{2max}$  for 2 minutes. The whole session would last for 42 minutes including warm up.

#### **Potential Risks**

By participating in this study, you will be exposed to risk of:

- muscle soreness
  - minimal exposure to radiation
  - physical and mental fatigue
  - increased likelihood of an adverse cardiovascular related event
  - infection, bruising and bleeding
- 1) You may experience both discomfort and muscle soreness after training and testing sessions which may last for a few days. Additionally, the likelihood of experiencing a cardiovascular related emergency during high intensity exercise is increased. However, your cardiac activity will be monitored for any irregularities with an ECG during exercise testing prior to the training intervention. You will be removed from the study if there is any irregularity in your cardiac activity. In addition, the investigator has a degree in sports and exercise science and is first-aid and automated external defibrillator certified and will be closely monitoring all exercise and testing sessions.
  - 2) As the Dual-energy x-ray absorptiometry (DEXA) provides an accurate estimation of your body composition, it will be used to assess your body composition (body fat percentage, fat-free mass and fat mass). Each scan takes about 3 minutes. The DEXA machine emits radiation during each scan. This will expose you to very low-level radiation. It is important to understand that DEXA scanning is routinely performed in the clinical setting, and produces extremely low levels of radiation dosages per scan. To assist your understanding, a single DEXA scan produces ~2.6  $\mu Sv$ ; a standard seven hour aeroplane flight exposes an individual to ~50  $\mu Sv$ ; and daily radiation levels (at sea-level) expose us to ~4  $\mu Sv$ . This study will only require you for seven scans, which will only expose you to a total low-level dosage of ~18.2  $\mu Sv$  distributed over seven separate assessments.
  - 3) Giving blood may cause some discomfort to you. The possible risks from this procedure are bleeding, dizziness, bruising and a slight risk of infection. Additionally, due to the difficulty of locating veins beneath fat tissues underneath the skin, the likelihood of requiring repetitive punctures is increased, which may result in further discomfort. An estimated 7.5 ml of blood will be extracted during each blood sampling session and a total of 52.5 ml of blood will be extracted from you over the course of the study (7 extractions). However, blood extraction will only be performed by certified phlebotomists and strict laboratory extraction procedures will be adhered to.

#### **Potential Benefits**

Apart from the contribution of your effort through this study to the current research understanding of the acute effects between moderate-intensity continuous and high-intensity interval cycling, as a participant of this study, you will receive complimentary health and fitness assessment and results will be furnished to you upon request. In addition you will participate in two supervised exercise sessions and you will be advised on how to integrate these into your subsequent exercise sessions.

#### **General**

##### **Privacy and Confidentiality**

All information collected during this research is private and confidential, and will not be disclosed to any third parties without your written consent, except to meet government, legal or other regulatory authority requirements. A de-identified copy of this data may be used for other research purposes. Your anonymity will, at all times, be safeguarded. Further to this, all data collected during this study will be de-identified to all

individuals, except the investigators, and will be stored on a password protected computer, and in a locked cabinet. Original data will be kept for a period of five years, and subsequently destroyed at the end of this time.

#### **Voluntary Participation**

Involvement in this study is strictly voluntary. Your decision to accept or decline participation in this project will not prejudice you in any way. During your involvement, you are free to withdraw your consent and discontinue at any time without any prejudice.

#### **Contacting Us**

If you have any questions or concerns regarding this study, you are always welcome to contact the chief investigator, Mr Derek Koh Fuijie: 6304 5097 (office); 0432 118 789 (mobile); [f.koh@ecu.edu.au](mailto:f.koh@ecu.edu.au) (email). Alternatively, you may also contact the project principal supervisor, Prof. Rob Newton: 6304 5037 (office); [r.newton@ecu.edu.au](mailto:r.newton@ecu.edu.au) (email).

If you wish to speak to an independent person regarding any concerns or complaints about this research project, you may contact the Research Ethics Officer: 6304 2170 (office); [research.ethics@ecu.edu.au](mailto:research.ethics@ecu.edu.au) (email).



#### Requirements of Study Participation – Checklist

Once you have read the information letter have you should have a clear understanding of what you will be asked of you upon your consent. Please carefully read the bullet points below and check each one that you clearly understand. If there is a point that is unclear please consult the chief investigator and they will answer any questions, clarifying the study procedures. Once each point is explained to your satisfaction you will be asked to sign the informed consent form.

☐ I am aware that the course of study will run for approximately 3 weeks which will include 1 familiarisation, 1 exercise testing, 2 exercise protocols and 7 data collection sessions.

☐ I understand that my physical activity and dietary habits will be monitored throughout the course of the study and that my diet will be standardised to my diet as recorded on baseline measurement prior to any fasting measurements. I also am clear that I must report to the laboratory early in the morning after a 12 h overnight fast for blood collection and gas analysis.

☐ I am aware that the integrity of my arterial vessels and systemic blood pressure will be monitored during the course of the study. It will involve the use of the standard sphygmomanometer and sphygmocardiogram (a non-invasive procedure involving the use of a tonometer to measure pulse pressure waves on the surface location of the radial artery located on the wrist.)

☐ I am aware that for this study, a total of 52.5ml of blood (7.5ml per sampling session) will be extracted from me over 7 sampling sessions and that each session will be performed by a trained personnel in phlebotomy. I also know that although the adverse risks are minimal, there is still a possibility of bruising, bleeding and infection from the site of collection. I have also been made aware that blood collection would be taken at either ECU sports physiology laboratory or Western Diagnostic Pathology collection centre located within Joondalup Health Campus.

☐ I am aware that my saliva will be collected on both days of the two exercise trials at three time intervals. It will involve a non-invasive procedure whereby I will have to direct my pooled saliva through a polypropylene drinking straw into a 2ml vial tube. I have also been informed that a total of 12ml of saliva will be collected from me throughout the study.

☐ I understand that I will be performing 1 exercise testing and 2 exercise protocols. The exercise testing will involve a maximal aerobic capacity test which will measure oxygen consumption and quantify the amount of work under maximal oxygen consumption. The 2 exercise protocols will involve single bouts of moderate-intensity continuous and aerobic interval cycling.

☐ I am aware that I will have my body composition accessed through DEXA scan procedures which exposes me to a total low-level dosage of ~18.2  $\mu$ Sv distributed over seven separate assessment sessions.

☐ I understand all the possible risks and discomforts that accompany all the procedures involved in this study.

☐ I understand of the benefits I will receive in participating in this study and that my involvement will contribute to the fight against chronic disease development.

## Consent

I, \_\_\_\_\_, have read the information provided and any questions I have asked have been answered to my satisfaction. I consent to become a participant in the research project, **"Comparison of the acute effects between aerobic interval versus moderate-intensity continuous cycling bouts"** and understand that I may withdraw at any time without reason and without prejudice.

I also understand that all information provided will be treated as strictly confidential and will not be released by the investigator unless required to by law. I have been advised with regards to the purpose of the research, the type of data that will be collected, and the way the data will be used upon completion of the research.

I agree that any research data gathered for the study may be published provided my name or other identifying information is not used.

\_\_\_\_\_  
Name of participant

\_\_\_\_\_  
Signature of participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature of researcher

## Appendix

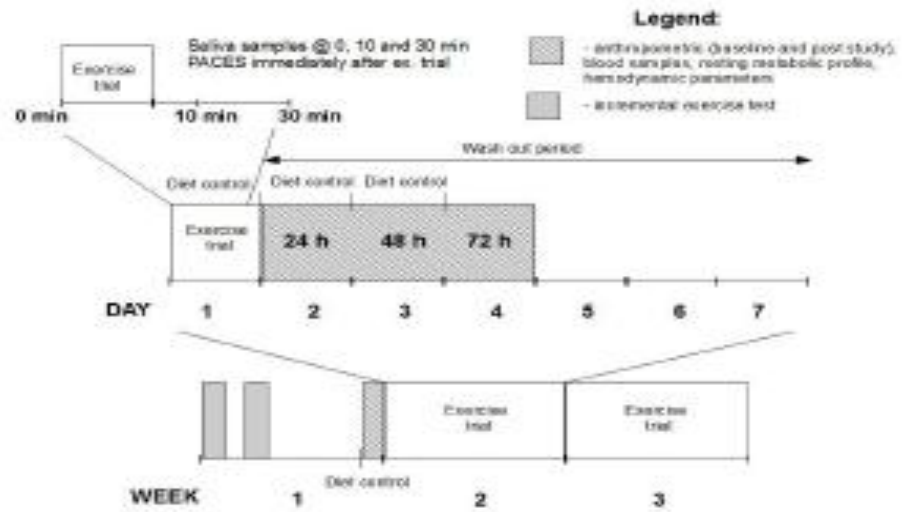


Figure 1. Schematic design of the study



### Medical Questionnaire

The following questionnaire is designed to establish a background of your medical history, and identify any injury and/ or illness that may influence your testing and performance.

Please answer all questions as accurately as possible, and if you are unsure about any thing please ask for clarification. All information provided is strictly confidential. If you answer "yes" to any non-exercise related question that may contraindicate you from completing a testing or training session a clearance from a qualified medical practitioner may be required prior to participation.

#### Personal Details

Name: \_\_\_\_\_

Date of Birth (DD/MM/YYYY): \_\_\_\_\_ Gender: Male

#### Emergency Contact Details (Next of Kin)

Name: \_\_\_\_\_

Relation: \_\_\_\_\_

Mobile: \_\_\_\_\_ Residential: \_\_\_\_\_

Workplace: \_\_\_\_\_

#### Medical History

Have you ever had, or do you currently have any of the following? If YES, please provide details

##### Cardiovascular-related conditions

(e.g high blood pressure, heart abnormalities, atherosclerosis)

Y N \_\_\_\_\_

High cholesterol Y N \_\_\_\_\_

Rheumatic fever Y N \_\_\_\_\_

Asthma Y N \_\_\_\_\_

Diabetes	Y	N _____
Epilepsy	Y	N _____
Recurring back pain	Y	N _____
Recurring neck pain	Y	N _____
Severe allergies	Y	N _____
Any infectious diseases	Y	N _____
Any neurological disorders	Y	N _____
Any neuromuscular disorders	Y	N _____
Have you had flu in the last two weeks?	Y	N _____
Have you recently injured yourself?	Y	N _____
Do you have any recurring muscle or joint injuries?	Y	N _____
Is there any other condition not previously mentioned which may affect your ability to perform exercise?	Y	N _____

Are you currently taking medications for any of the conditions as stated above (those that you have circled "yes")?      Y      N

If yes, please list the medications that you have been prescribed with:

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#### Family Medical History

Do any of your parents have complications related to blood sugar, cholesterol and blood pressure?

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Do you smoke tobacco?	Y	N
If YES, how much per day?		

Do you consume tea or coffee?	Y	N
If YES, how many cups per day?		

## Participant

Signature: \_\_\_\_\_

Signature: \_\_\_\_\_