

EFFECTS OF HIGH INTENSITY INTERVAL EXERCISE VERSUS STEADY STATE
EXERCISE WITH SIMILAR ENERGY EXPENDITURES ON EPOC

by

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A thesis submitted in partial fulfillment
of the requirement for the degree of
Master of Human Kinetics

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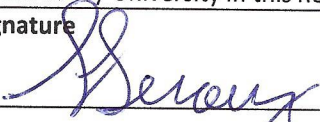
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Abstract

The purpose of the study was to determine whether steady state exercise (SSE) or high intensity interval exercise (HIIE) would better improve energy expenditure (EE) during 90 minutes of excess post exercise oxygen consumption (EPOC) while attempting to match EE between both exercise protocols. We also wanted to examine physiological changes during post exercise measurements, which included VO_2 , RER, VE and HR. Twelve males aged between 19 and 24 were assigned to the SSE and HIIE conditions. A $\text{VO}_{2\text{max}}$ and a 30s-all-out sprint set at 150% of maximum workload was performed on a cycling ergometer interspersed by 5 minutes to ensure sufficient recovery time. Participants randomly completed SSE or HIIE followed by 90 minutes of EPOC. A gross efficiency (GE) of 18% was used in order to best quantify the anaerobic attributable EE during the HIIE in order to estimate total EE. Our results indicate that the HIIE expended less EE than SSE and from our pre-test EE estimations ($p < 0.05$). With that being said, HIIE was able to generate a greater EE during EPOC in comparison to SSE, while utilizing more grams of fat during post exercise measurements ($p < 0.05$). There was no significant difference between both protocols when adding exercise and EPOC EE. Physiological markers such as VO_2 ($\text{L}\cdot\text{min}^{-1}$), VE ($\text{L}\cdot\text{min}^{-1}$) and HR were significantly greater in HIIE during EPOC. To conclude, our findings indicate that HIIE is a time efficient workout able to expend more EE, utilize more fat and have greater physiological responses during EPOC when compared to SSE.

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List of Abbreviations

AOD	accumulated oxygen deficit
BLC	blood lactate concentration
BLC0	blood lactic acid measured immediately after the high intensity interval exercise
BLC3	blood lactic acid measured 3 minutes after the high intensity interval exercise
BLC5	blood lactic acid measured 5 minutes after the high intensity interval exercise
BLCH	blood lactic acid measured halfway through the high intensity interval exercise
BLCP	blood lactic acid measured prior to high intensity interval exercise
BMI	body mass index
CP	critical power
CSEP	Canadian society for exercise physiology
EE	energy expenditure
EE _{EPOC}	energy expenditure from excess post exercise oxygen consumption
EE _{HIE}	energy expenditure from a 30s sprint & the 60s recovery (1 repetition)
EE _{sprint}	energy expenditure from the 30s sprint post VO_{2max}
EE _{SSE}	steady state exercise energy expenditure
EPOC	excess post exercise oxygen consumption
GE	gross efficiency
HIAE	high intensity aerobic exercise
HIIE	high intensity interval exercise
HIIT	high intensity interval exercise
LT	lactate threshold
MAOD	maximal accumulated oxygen deficit
MLC	muscle lactate concentrations
OBLA	onset of blood lactate accumulation
PI	power input
PO	power output
RER	respiratory exchange ratio

SSE	steady state exercise
SST	steady state training
VE/VO ₂	ventilatory equivalent
VO	oxygen consumption
VO ₂ drift	cardiovascular drift (oxygen consumption drift)
VO _{2max}	maximal oxygen consumption
W _{max}	work max

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1. Introduction

Physical activity has been positively correlated with physical and mental health-related quality of life (Bize, Johnson, & Plotnikoff, 2007). However, evidence suggest that 85% of Canadians fail to meet the physical activity recommendations (Colley et al., 2011a). As of 2017, as many as 49.2% and 67.7% of adults ranging from 18-34 and 35 to 49 years of age, respectfully, self-reported and categorized themselves as obese or overweight (Statistics Canada, 2018). This lack of exercise increases the risk of mortality, which progressively increases with rising BMI values that range above 30 (Calle, Thun, Petrelli, Rodriguez, & Heath, 1999; Gu, He, & Duan, 2006; Jee et al., 2006). Physiological repercussions in response to obesity include metabolic inflammatory markers that can give rise to atherosclerosis, hypertension, type-II diabetes, impaired glucose tolerance as well as dyslipidemia characterized by elevated triglycerides, low HDL-cholesterol and a small LDL particle phenotype (Lamarche, Lemieux, & Després, 1999; Lee, Blair, & Jackson, 1999; Tchernof et al., 1996). Obesity has also been associated with various forms of cancers (Renehan, Tyson, Egger, Heller, & Zwahlen, 2008), psychological and social burdens (Colles, Dixon, & O'Brien, 2008; Dixon, Dixon, & O'Brien, 2003; Wadden et al., 2006) as well as depression in older individuals with poor health, chronic disease and functional disabilities (Onyike, Crum, Lee, Lyketsos, & Eaton, 2003; Roberts, Deleger, Strawbridge, & Kaplan, 2003).

The Canadian Society for Exercise Physiology and the U.S. Department of Health and Human Services both recommend that adults should achieve 150 minutes of moderate to vigorous exercise per week in order to obtain health benefits (Donnelly et al., 2009; Tremblay et

al., 2011). However, many Canadians fail to meet these recommendations (Colley et al., 2011b) with lack of time (Kimm et al., 2006; Stutts, 2002) and access to facilities being the two major factors for inactivity (Booth et al., 2000; Sherwood & Jeffery, 2000). Alternatively, short but very intense exercise can potentially provide health benefits in a time efficient manner (Keating et al., 2014). Compared to traditional continuous exercise, high intensity interval training (HIIT) has been shown to have similar or greater cardiovascular and metabolic benefits in healthy populations despite having a smaller training volume (Keating et al., 2014). These benefits include an increase in cardiorespiratory fitness (Babraj et al., 2009; Burgomaster et al., 2008; Ciolac, Bocchi, Bortolotto, Carvalho, & Greve, 2010; MacPherson, Hazell, Olver, Paterson, & Lemon, 2011; Nybo et al., 2010; Trapp, Chisholm, Freund, & Boutcher, 2008), improved work capacity (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005) and insulin sensitivity (Babraj et al., 2009; Ciolac et al., 2010; Trapp et al., 2008). The benefits of HIIT are reportedly similar for overweight/obese men, notably an increase in VO_{2max} and resting fat oxidation, improvement in insulin sensitivity and decrease in systolic blood pressure (Whyte, Gill, & Cathcart, 2010). Interestingly, this particular type of training had beneficial results in populations with coronary artery disease, congestive heart failure, middle aged adults with metabolic syndrome and obese individuals as it revamped their cardiorespiratory fitness (Moholdt et al., 2009; Munk, Staal, Butt, Isaksen, & Larsen, 2009; Warburton et al., 2005; Wisloff et al., 2007).

Energy intake that exceeds energy expenditure (EE) results in a positive energy balance that can consequently lead to weight gain (Thyfault et al., 2004). Therefore, EE that occurs from exercise can lead to a daily negative energy balance hence, weight loss. Measuring EE during steady state exercise is precisely done with the use of a gas analyzer as it measures energy derived from the aerobic system. On the other hand, high intensity exercise above 100% work

W_{\max} relies mainly on the anaerobic system making it difficult to quantify its EE since there is no gold standard method to confidently quantify it. Different methods have been introduced over the years in order to estimate anaerobic attributable EE, such as the blood lactate concentrations (BLC), the O_2 deficit more commonly known as the Accumulated Oxygen Deficit (AOD), the Excess Post Exercise Oxygen Consumption (EPOC) and the Gross Efficiency (GE) method.

This review of literature will elaborate on the health benefits of exercise, the different energy systems that are mobilized during physical activity, measures by which energy expenditure can be quantified whether it is derived from the aerobic or anaerobic pathway and the physiological benefits that emanate from continuous and high intensity interval training/exercise. It will also review recent literature to explore which of these two exercise protocols (i.e. continuous vs interval) is optimal for post exercise energy expenditure.

2. Literature review

2.1 Physical activity

Physical activity is defined as “bodily movement that is produced by the contraction of skeletal muscle and that substantially increases energy expenditure” (Physical activity and health: a report of the Surgeon General, 1996). Increasing EE is crucial to maintain a healthy lifestyle as it influences energy balance and body composition. It can also decrease the risk of cardiovascular disease, stroke, type II diabetes, as well as colon and breast cancers (Miles, 1996). On the other hand, physical inactivity, which is defined as “a state in which bodily movement is minimal and energy expenditure approximates the resting metabolic rate” (Bianchini, Kaaks, & Vainio, 2002) leads to a positive energy balance. To clarify, energy intake exceeding EE over a period of time is the foundation of weight gain (Thyfault et al., 2004). Being overweight or obese is categorized as having a body mass index (BMI) ranging from 25-29.9 kg.m⁻² and 30 kg.m⁻² +, respectively (Donnelly et al., 2009). Detrimental health consequences can arise from having excessive levels of body fat such as; heart disease, hypertension, diabetes, some forms of cancer along with psychosocial and economic difficulties (Gortmaker, Must, Perrin, Sobol, & Dietz, 1993; Mokdad et al., 2003; Must et al., 1999). A large waist circumference along with high visceral fat has been strongly associated with increased risk of mortality in middle-aged men and women (De Koning, Merchant, Pogue, & Anand, 2007; Reis et al., 2009), whereas a greater proportion of fat free mass is correlated with a lower risk of all-cause mortality (Biggaard et al., 2005; Heitmann, Erikson, Ellsinger, Mikkelsen, & Larsson, 2000).

The prevalence of obesity over the last several decades has been steadily rising at unprecedented levels (Flegal, 2012). One underlying cause behind this escalation is the greater

consumption of high caloric (sugar) and fat-laden foods, which are not only affordable but also easily accessible (Rolls, 2003). Physical inactivity has also been shown to be a relevant cause of obesity. Of concern is the fact that many adults do not meet the recommended levels of exercise (Macera et al., 2003). According to the Canadian Society for Exercise Physiology and the U.S Department of Health and Human Services, 150 minutes of moderate to vigorous exercise per week should be completed if one wishes to obtain health benefits (Donnelly et al., 2009; Tremblay et al., 2011). However, lack of time is often cited as the most common reason for inactivity in patients with chronic conditions as well as those at risk of cardio-metabolic disease (Gibala & Little, 2010; Gibala, Little, Macdonald, & Hawley, 2012). A potential solution to overcome the confines of time is high intensity interval training/exercise (HIIT/HIIE), which condenses a full workout into 15-20 minutes of exercise. This genre of exercise will be discussed in more detail later in this literature review.

2.2 Exercise

2.2.1 Anaerobic

Physical fitness cannot be merely summarized by maximal oxygen consumption alone. Speed, strength and maximal power are all determinant factors of athletic activities derived from the anaerobic metabolism. This powerful yet limited energy system is divided into alactic and lactic components (Vandewalle, Péérès, & Monod, 1987). The alactic system creates the shortest and strongest muscle contraction and predominates during the first 10 seconds of high intensity exercise (Serresse, Lortie, Bouchard, & Boulay, 1988). The energy source of this immediate

muscle contraction is generated from ATP and phosphocreatine, which are directly stored in the muscle cell (Jacobs, Tesch, Bar-Or, Karlsson, & Dotan, 1983). A non-invasive biopsy to visualize ATP and creatine phosphate that is also based on muscle energetics has been developed from the application of ^{31}P magnetic resonance spectroscopy (Meyer, Kushmerick, & Brown, 1982; Chance et al., 1986; Blei, Conley, & Kushmerick, 1993a; Blei, Conley, Odderson, Esselman, & Kushmerick, 1993b; McCully, Fielding, Evans, Leigh, & Posner, 1993), which emits a radio-frequency within a magnetic field providing a unique view of ATP turnover and creatine phosphate (Conley, Cress, Jubrias, Esselman, & Odderson, 1995).

Anaerobic glycolysis is the second system which breaks down carbohydrates, mainly in the form of muscle glycogen, resulting in pyruvic acid followed by lactic acid (Gastin, 2001). The glycolytic system is mobilized when the exercise demand exceeds ATP-PC stores and has been found to peak between the 16th and 30th second of intense exercise (Jacobs et al., 1983; Serresse et al., 1988). Maximal anaerobic power has been found in exercise lasting as much as 90 seconds when performed on a cycling ergometer with a resistance set at 0.1 kp/kg (Serresse, Simoneau, Bouchard & Boulay, 1991).

During high intensity exercise (e.g. HIIE) fast twitch motor units are recruited for relatively short durations (Enoka & Duchateau, 2008; Scott, 2011), however, these muscles fibres are less efficient and easily fatigued (Sargeant, 1994). The result is the attenuation of the anaerobic capacity through depletion of ATP-PC stores in the muscle and blood lactate accumulation (Hermansen & Vaage, 1977; Hultman & Sjoholm, 1983; Karlsson & Saltin, 1970; Medbø & Tabata, 1989; Sahlin, Harris, & Hultman, 1975). It is imperative to note that all three energy systems (i.e. alactic, glycolytic and aerobic) are sequentially interconnected in order to

efficiently deliver the necessary energy into working muscles (Gastin, 2001). Single bouts of dynamic high intensity exercise lasting 6s (Péronnet & Thibault, 1989), 30s (Calbet, Havarren, & Dorado, 1997), 60s (Withers, Ploeg, & Finn, 1993) and 90s (Gastin & Lawson, 1994) have all shown to have aerobic contributions ranging from 5%, 23%, 47% and 58%, respectively. Not only is the oxidative pathway relevant during intense exercise but it can also be utilized for the removal of lactic acid and for the resynthesis of phosphocreatine (Linossier, Denis, Dormois, Geysant, & Lacour, 1993).

2.2.2 Aerobic

Aerobic capacity is the product of the body's ability and efficiency to supply oxygen to the skeletal muscles via the cardiorespiratory system in order to create energy (McArdle, Katch & Katch, 2010). This process uses carbohydrates, fats and in some circumstances proteins when oxygen is present and provides large amounts of ATP (Gastin, 2001) however, it is rate-limited by the oxidative phosphorylation efficiency and by the respiratory and cardiovascular systems' ability to deliver the oxygen to the working muscles (Gastin, 2001). The energy produced by the aerobic system can be calculated by steady-state O₂ uptake measurements and is considered the gold standard in the estimation of exercise-energy expenditure (Scott, Leighton, Ahearn, & McManus, 2011). Energy released from the breakdown of carbohydrates and fats in the presence of oxygen is directly related with whole-body aerobic production of ATP (Åstrand, 1981). For every litre of oxygen consumed (standard temperatures, pressure and density) roughly 20kJ of energy is produced (Gastin, 2001; Scott, 1998). The respiratory exchange ratio (RER) enables us to determine the proportion of carbohydrates and fats that are utilized to provide the indirect

measurement of aerobic energy generated (Gastin, 2001). Lactate production can occur during light aerobic exercise where it is oxidized and utilized in the Krebs cycle. When lactate production is equivalent to its removal, and its concentration remains constant, VO_2 can represent whole body EE (di Prampero & Ferretti, 1999).

Metabolic and physiological adaptations in response to exercise depend on the intensity, duration and frequency of exercise training sessions. Endurance training causes adaptations in the pulmonary, cardiovascular and neuromuscular system, which improve the delivery of oxygen to the mitochondria, consequently enhancing aerobic metabolism within the muscle cells (Jones & Carter, 2000). Training programs on recreationally active participants involving as little as 3-5 weekly sessions of 20-30 minutes of aerobic training have been shown to increase adult male and female's $\text{VO}_{2\text{max}}$ by 10% over a 6 week period (Carter, Jones, & Doust, 1999). Gormley et al. (2008) had 61 young adults free of cardiovascular disease matched for their sex and $\text{VO}_{2\text{max}}$. They were randomly assigned to perform at 50%, 75% 95% of their VO_2 reserve for 6 weeks while having their EE controlled throughout. They found that each group had a significantly greater $\text{VO}_{2\text{max}}$ after the training period, but more importantly, the higher intensity exercise was more effective for improving $\text{VO}_{2\text{max}}$. Another adaptation resulting from aerobic training is change in skeletal muscle substrate metabolism (Holloszy & Coyle, 1984). For example, Chesley, Heigenhauser, & Spriet (1996) and Green et al. (1992) found that cycling at 65-67% $\text{VO}_{2\text{max}}$ for 2 hours over the course of 5-7 days decreased the rate of glycogen degradation and lactate production when exercise intensities were consistent throughout the training program.

Aerobic exercise has also been shown to increase resting fat oxidation in young healthy, trained, and elderly individuals (Calles-Escandon, Goran, O'Connell, Nair, & Danforth Jr, 1996;

Poehlman, Gardner, Arciero, Goran, & Calles-Escandon, 1994; Romijn, Klein, Coyle, Sidossis, & Wolfe, 1993). There is evidence suggesting a greater post exercise energy expenditure when subjects exercised at 70% $\text{VO}_{2\text{max}}$ (20, 40 & 80 minutes) where longer exercise durations led to a greater post exercise EE (Bahr, Inghes, Vaage, Sejersted, & Newsholme, 1987). Although exercise duration is a key factor when measuring EPOC (Bahr et al., 1987), relative intensity of exercise has been shown to influence physiological responses (Jones & Poole, 2005; Poole, Ward, Gardner, & Whipp, 1988; Whipp & Wasserman, 1972; Wilkerson, Koppo, Barstow, & Jones, 2004).

2.2.3 VO_2 Slow Component

Steady state exercise performed at a constant resistance, below the lactate threshold (LT), will result in a stable VO_2 within 3 minutes (Whipp & Wasserman, 1972; Whipp, Ward, Lamarra, Davis, & Wasserman, 1982). Once exercise intensity surpasses LT, additional O_2 requirements are necessary and continue to rise until they reach a peak value (i.e. $\text{VO}_{2\text{max}}$) or exhaustion. The difference in VO_2 found between exercise exceeding 3 minutes above LT values and $\text{VO}_{2\text{max}}$ is characterized as the VO_2 slow component, not to be confused with the more modest VO_2 drift (Barstow, 1994; Casaburi, Storer, Ben-Dov, & Wasserman, 1987; Gaesser, 1994; Poole et al., 1991; Whipp, 1994). LT is identified by an increase in blood lactate and abrupt changes in the respiratory exchange ratio (RER), which is closely related but not interchangeable with the ventilatory threshold (Coyle, Coggan, Hopper, & Walters, 1988; Ivy, Withers, Van Handel, Elger, & Costill, 1980; Power, Dodd, & Garner, 1984). If the workload is performed above the LT and below the critical power (CP), steady VO_2 is delayed (Poole et al.,

1988; Whipp & Wasserman, 1972; Wilkerson et al., 2004), and the VO_2 value would be greater than the predicted VO_2 gathered from the sub-LT VO_2 . CP is known as the maximal amount of sustained exercise for a prolonged duration (Jones, Vanhatalo, Burnley, Morton, & Poole, 2010), whereas work done above CP would not result in a steady state, but rather, a continuous rise until $\text{VO}_{2\text{max}}$ is attained (Poole et al., 1988; Whipp & Wasserman, 1972). Exercise intensity domains such as “moderate” (<LT), “heavy” (>LT yet < CP), and “severe” (>CP) were named from VO_2 fluctuations (Poole et al., 1988; Wilkerson et al., 2004). Numerous studies have indicated that muscular contractions are a contributing factor to the pulmonary VO_2 slow component (Koga et al., 2005; Krustup, Jones, Wilkerson, Calbet, & Bangsbo, 2009; Poole et al., 1991). More specifically, the additional muscle fiber recruitment and the transition towards type II muscle fiber recruitment has a significant role on the VO_2 slow component during constant work exercise (Jones et al., 2011).

Cardiovascular drift (VO_2 drift) also ensues roughly 10 minutes into prolonged moderate intensities ranging from 50-75% $\text{VO}_{2\text{max}}$ in a neutral or warm environment (Ekelund, 1967; Johnson & Rowell, 1975). This process is characterized by a gradually decreasing stroke volume, pulmonary and systemic mean arterial pressure accompanied with an increase in heart rate in order to maintain an appropriate cardiac output (Ekelund, 1967; Rowell, 1987).

2.2.4 High Intensity Interval Exercise

As many as 85% of Canadians fail to meet the minimum physical activity recommended by the Canadian Society for Exercise Physiology (CSEP) (Colley et al., 2011b) with lack of time

being the predominant reason (Troost, Owen, Bauman, Sallis, & Brown, 2002). A time efficient solution to this problem is high-intensity interval training/exercise (HIIT/HIIE), which can be defined as brief, repeated bursts of intense exercise close to VO_{2max} (i.e., $\geq 90\%$) separated by periods of low intensity exercise. Low volume HIIE is characterised by training sessions that consist of ≤ 10 minutes of intense exercise within a 30-minute window including warm-up, recovery periods and a cool down (Gibala & McGee, 2008; Gillen & Gibala, 2014). This type of exercise may be as effective as the traditional prolonged continuous training while having similar health benefits in addition to having a reduced time commitment (Gillen & Gibala, 2014). The health benefits that arise from interval training include the improvement of muscular strength (Kraemer, Ratamess, & French, 2002; Hass, Feigenbaum, & Franklin, 2001), muscular endurance (Mazzetti et al., 2000), an increase in bone mass along with a reduction in blood pressure and body fat (Kraemer et al., 2002).

Different variations of HIIE protocols can be applied in order to obtain health benefits. Trapp et al. (2008) conducted a study on young women where they trained for 15 weeks performing 8-second all-out sprints followed by 12 seconds of active recovery for 20 minutes. Their findings included a decrease in whole-body abdominal and trunk fat mass accompanied with an increase in their VO_{2max} , whole-body leg and trunk fat free mass. Overweight young men completed this same training regimen in a 12-week period, which led to whole-body fat mass reduction along with an increase in fat oxidation, and lean mass in the legs and trunk (Heydari, Freund, & Boutcher, 2012).

One of the most common HIIE protocols is the Wingate Test, which consists of 30s all-out cycling set with a high resistance. Repeating this workout six times, varying from 4 to 7

repetitions, over the course of two weeks has been shown to increase skeletal muscle oxidative capacity (Burgomaster, Heigenhauser, & Gibala, 2006; Burgomaster et al., 2005; Gibala et al., 2006) in healthy subjects who were previously sedentary or physically active. The Wingate protocol also improved subjects VO_{2max} (Astorino, Allen, Roberson, & Jurancich, 2012; Hazell, MacPherson, Gravelle, & Lemon, 2010; Whyte et al., 2010) and resting fat oxidation 24h post-exercise (Whyte et al., 2010). Babraj et al. (2009) and Metcalfe, Babraj, Fawkner, & Vollaard (2012) have shown that short-term Wingate-based HIIT protocols improved insulin sensitivity as measured using an oral glucose tolerance test in young healthy men. Similar results were found in overweight and obese individuals (Whyte et al., 2010), along with recreationally active men and women using the gold standard hyperinsulinemic euglycemic clamp method with the same exercise protocol (Richards et al., 2010). Prolonging this same workout to 6-weeks induced an increase in the individuals' VO_{2max} (Astorino et al., 2012; Burgomaster et al., 2007, 2008), as well as an elevation in whole body fat oxidation, resting muscle glycogen content (Burgomaster et al., 2008) and cardiovascular and skeletal muscle remodelling that is observed with traditional endurance training programs. In other words, short-term HIIT was found to produce outcomes similar to those resulting from prolonged exercise despite having a 90% difference in training volume (Burgomaster et al., 2007, 2008).

Although all-out HIIT protocols can be very effective, it can be impractical for some individuals, as it requires a sizable amount of motivation. For this reason, studies were done to examine participants' physiological adaptations to HIIT at submaximal intensities. Multiple studies report training protocols using 60 seconds of cycling at 85-90% of HR_{max} interspersed with 60 seconds of active recovery. Physiological improvements from this type of training were found in young healthy individuals (Little, Safdar, Wilkin, Tarnopolsky, & Gibala, 2010),

overweight/obese individuals (Gillen, Percival, Ludzki, Tarnopolsky, & Gibala, 2013), in previously sedentary adults (Hood, Little, Tarnopolsky, Myslik, & Gibala, 2011), in patients with coronary artery disease (Currie, Dubberley, McKelvie, & Macdonald, 2013) and in subjects with type II diabetes (Little et al., 2011a). Furthermore, two weeks of HIIT has been shown to increase time trial performance, maximal workload and glycemic control in type II diabetes patients (Little et al., 2011a). Hood et al., (2011) Little, Gillen, & Percival (2011b), and Little et al. (2010) have all shown that an increase of maximal activity of citrate synthase and cytochrome c oxidase can occur from only 2 weeks of HIIT training. “Citrate synthase is one of the key regulatory enzymes in the energy-generating metabolic pathway that catalyzes the condensation of oxaloacetate and acetyl coenzyme A to form citrate in the tricarboxylic acid cycle” (Siu, Donley, Bryner, & Alway, 2003, p.555). Little et al. (2010) found that cycling training increased citrate synthase, which is likely to improve exercise performance as it would enable individuals to work at a higher metabolic rate. The physiological response to an increase of cytochrome c levels is an augmentation in the capacity to oxidize pyruvate and fatty acids and to generate ATP (Freysenet, Di Carlo, & Hood, 1999; Holloszy & Coyle, 1984; Swallow, Garland, Carter, Zhan, & Sieck, 1998).

Findings from Gillen, Little, Punthakee, Tarnopolsky & Gibala (2012) suggest that 6 weeks of 10 x 60 seconds of HIIT at 90% HR_{max} led to a reduced 24 hour blood glucose concentration immediately after a single session of exercise. They also found that subjects' VO_{2max} , work load max, maximal activity of citrate synthase, glucose transporter 4 content, leg and gynoid fat-free mass all increased after the training program. Individuals suffering from coronary artery disease took part in a cycling training regimen that consisted of 10 x 60 second sessions twice a week for 12 weeks. In this case, low-volume HIIT led to an increased VO_{2max}

and brachial artery flow mediated dilation (Currie et al., 2013). The results from the HIIT group were similar to the continuous group (cycled at 58% of peak power output (PO) for 30-50 minutes) despite having a smaller training volume. Improvements in body composition and fitness were found in overweight/obese women during a 6-week program that consisted of 10 x 60s cycling bouts set at $\sim 90\%HR_{max}$ (Gillen et al., 2013). Whyte et al. (2010) found that 6 sessions of sprint training over the course of 2 weeks led to an increase in subjects VO_{2max} , mean PO, insulin sensitivity, resting fat oxidation and reduced systolic blood pressure in overweight/obese men. Prolonging the exercise to 12 weeks led to an increase in VO_{2max} , trunk and leg fat-free mass and a decrease in total, abdominal, trunk and visceral fat of young overweight males (Heydari et al., 2012).

Aerobic EE is easily quantified because there is a direct correlation between VO_2 measured at the mouth and whole-body ATP production through the combustion of carbohydrates and fats (Åstrand, 1981). However, methods to quantify anaerobic energy production are less precise considering anaerobic ATP production is an intracellular process (Gastin, 2001). Methods that have been introduced to estimate anaerobic energy release include blood lactate measurements, oxygen debt, oxygen deficit and gross efficiency.

2.2.5 Blood Lactate

During relatively intense exercise, there is an inadequate oxygen supply to the mitochondria, which initiates anaerobic glycolysis in order to provide the muscle with sufficient contractile energy. The by-product of this physiological mechanism results in lactic acid

(Wasserman, Beaver, & Whipp, 1990). As the intensity increases, lactate production surpasses its excretion and/or its utilisation in the muscle partly due to blood flow deviations from lactate removal sites such as non-exercising muscles, liver, kidneys and the heart (Brooks et al., 1991; Karlsson & Jacobs, 1982). Working muscles naturally contribute to the elevation of circulating BLC, but as the exercise continues, these muscles are able to consume and oxidize the lactate (Brooks et al., 1991). This process is known as the lactate shuttle mechanism (Brooks, 1985), which suggest that lactic acid increment in the blood is reciprocal with the total number of muscle fibers recruited in conjunction with the intensity of muscle activation (Stainsby & Brooks, 1990). The lactate shuttle mechanism assumes that lactate production occurs more in fast twitch muscles fibers, which then circulates to other fast-twitch or slow twitch fibers where it is converted into pyruvate. This substrate is then able to convert into acetyl-CoA enabling its entry into the citric acid cycle for aerobic energy metabolism. The lactate shuttle is an efficient system considering it can use the by-product of glycogenolysis from one cell in order to create fuel for another cell. Therefore, muscles are not only a site of lactate production, but also play an essential role in its removal via oxidation (Brooks, 2000; Gladden, 2008; Hashimoto & Brooks, 2008). The Cori cycle, on the other hand, is able to gather lactate from working muscles, transport it to the liver where it is transformed into glucose or stocked as glycogen following intense exercise (Sumida, Urdiales, & Donovan, 1993).

Lactate production increases due to anaerobic energy metabolism initiated by deficiencies in aerobic energy metabolism, known as anaerobic threshold (Davis, Vodak, Wilmore, Vodak, & Kurtz, 1976). Contrary to popular belief, lactate can be utilized under fully aerobic conditions, most notably when there are high levels of glycogenolysis and glycolysis (Brooks, 2002; Richardson, Noyszewski, Leigh, & Wagner, 1998). As previously mentioned, the lactate

threshold occurs once there is an increment in exercise intensity where BLC increases accordingly (Svedahl & MacIntosh, 2003). The onset of blood lactate accumulation (OBLA), also known as individual anaerobic threshold, occurs once BLC reaches 4mmol/L (Sjödín & Jacobs, 1981; Svedahl & MacIntosh, 2003). Muscle lactate concentrations (MLC) and BLC have been found to be similar near rest and during submaximal exercise until ~60% VO_{2max} (Knuttgen & Saltin, 1972). Exercise above this intensity leads to a sudden increase in MLC two to three times that of BLC, notably known as the anaerobic threshold (Chwalbinska-Moneta, Robergs, Costill, & Fink, 1989). The degree of changes in post-exercise measurements of BLC is dependent on the intensity and duration of the exercise (Ali, Bhatti, Khan, & Jan, 2004). That being said, Gollnick, Bayly, & Hodgson (1986) found that BLC peaked 5 minutes after the termination of intense exercise indicating that BLC and MLC are not interchangeable measures.

Although BLC may represent the extent of glycolysis, it cannot be utilized to estimate lactate in the working muscles since BLC is the product of various exchanges and transport processes occurring between blood and numerous tissues and organs during exercise (Cabrera, Saidel, & Kalhan, 1999). Gathering lactate information from working muscles would be quite difficult as it would require biopsies every few seconds. This method would still fall short of providing any information from the alactic energy system (Jacobs & Kaiser, 1982; Karlsson, 1971; Medbø, 1993; Tesch, Daniels, & Sharp, 1982). Blood samples, on the other hand, can be taken from the participant pre-or post-exercise making it quite convenient and also affordable to measure lactate levels. Monitoring the BLC during training sessions can indicate the level of physical stress individuals are experiencing during exercise (Norton, Norton, & Sadgrove, 2010).

Although it is quite evident that lactate production increases with exercise intensities, its removal from the blood is dependent of the fitness level as well as the metabolic rates making it inappropriate to use BLC to estimate lactate production (Donovan & Brooks, 1983). Bangsbo et al. (1990) suggests that by the time lactate is evenly distributed in various aqueous compartments of the body, a large portion of it is already metabolized. Despite the fact that BLC reflects the anaerobic metabolism during intense exercise, it is inadmissible to use this method in order to compare the anaerobic capacity between different subjects (Gastin, 1994). Specifically, BLC should not be used to presume an increase in the glycolytic potential following sprint-training (Gastin, 1994).

2.2.6 O₂ debt/EPOC

Another method used to assess anaerobic energy expenditure is to measure EPOC, previously known as oxygen debt. EPOC represents the amount of O₂ consumed during post-exercise measurements that exceed the resting values. The oxygen debt hypothesis predicts that the volume of O₂ consumed after the exercise is correlated to the metabolism of lactate during post-exercise recovery (Hill & Lupton, 1923). Margaria, Edwards, & Dill (1933) modified the hypothesis by dividing O₂ debt into two categories alactic and lactic components. However, the use of oxygen debt has been questioned and discredited by several authors (Hermansen, 1969; Vandewalle et al., 1987). Bangsbo et al. (1990) found that O₂ debt, whether it was measuring an active muscle or the entire body, markedly overestimated the anaerobic energy release. The by-products of exercise consists of elevated VO₂, lactate production as well as numerous factors that stimulate mitochondrial respiration, consequently impairing EPOC's ability to precisely estimate

the anaerobic EE (Gaesser & Brooks, 1984; Rieu, Duvallet, Scharapan, Thieulart, & Ferry, 1988). An increase in post exercise VO_2 (i.e. EPOC) is the result of ATP and PCr resynthesis, lactate to glycogen resynthesis, the oxidation of lactate into energy metabolism, the restoration of oxygen into myoglobin and hemoglobin. Thermogenic effects of elevated core temperatures and hormones (catecholamines), effects of an elevated heart rate, an increment of ventilation as well as the body's energy needs to return to homeostasis can also increase EPOC (Bangsbo et al., 1990; Gaesser & Brooks, 1984; McArdle et al., 2010). Many factors are related to EPOC but the exact underlying mechanisms remain unclear (Tahara et al., 2008). Gaesser & Brooks (1984) have recommended the use of the term EPOC instead of oxygen debt to avoid any confusion between the elevations in metabolic rate above resting levels after exercise. Moderate exercise intensities rely mainly on the aerobic system whereas high intensity exercise (supramaximal exercise) exceeds the rate of ATP produced by the oxidative system and is therefore heavily dependent of the anaerobic system (Medbø & Tabata, 1989). In essence, contributions of different energy systems are dependent on the exercise intensity and duration. In relation to EPOC, intensity and duration of the exercise both influence the magnitude of the VO_2 during post-exercise recovery (Børsheim & Bahr, 2003). The energy that is borrowed from energy expenditure reserves are paid back in extensive O_2 uptake during the recovery phase (Brooks, Fahey, & White, 1996; Gaesser & Brooks, 1984). Oxidative EE is measured from the VO_2 ($L \cdot min^{-1}$) multiplied by the RER, which amounts to an energy equivalent value measured in KJ. The RER is the CO_2 produced divided by steady state O_2 consumed and typically results in values ranging between 0.70 (total fat oxidation) and 1.00 (total glucose oxidation) {Appendix 10.1} (Ferrannini, 1988; Scott, 2011).

2.2.7 O₂ deficit

As previously mentioned, there are several methods to estimate the maximal anaerobic capacity during various types of exercise. One of the most frequently used methods is the maximal accumulated oxygen deficit (MAOD). The MAOD is based on the linear relationship between treadmill speed or cycling PO and VO₂ (Medbø et al., 1988; Medbø & Tabata, 1989, 1993). The accumulated VO₂ demand is calculated by multiplying the supramaximal exercise duration (roughly 2-3 minutes) by the predicted VO₂ demand (Noordhof, Skiba, & de Koning, 2013a). Subsequently, the MAOD can be determined by subtracting the oxygen uptake measured during the supramaximal exercise from the accumulated VO₂ demand (Medbø et al., 1988). A review done by Noordhof, de Koning, & Foster (2010) evaluated the procedures and outcomes of studies that used oxygen deficits as a method to determine the anaerobic capacity. Even though MAOD is the most commonly used approach to determine anaerobic capacity, that does not imply that it is a flawless method (Bangsbo, 1992, 1996; Noordhof et al., 2010). Findings from Bangsbo (1992, 1996) states that using a linear relationship between exercise intensity and VO₂ in order to determine anaerobic capacity may be unwise. The MAOD method must begin with relatively short submaximal exercise in order to construct a linear PO and VO₂ relationship (Bangsbo, 1996; Buck & McNaughton, 1999; Noordhof, Vink, de Koning, & Foster, 2011). Non-linear relationships between the PO and VO₂ could easily occur if the submaximal exercises are too intense, which would provoke a secondary VO₂ spike also known as VO₂ slow component. Based on Maxwell & Nimmo (1996), 8-10 minutes of submaximal exercise is the appropriate time to properly assess the treadmill speed-VO₂ relationship. Noordhof et al. (2010) proposed 10x4 minute submaximal exercise bouts distributed between 30-90% VO_{2max} along with a fixed y-intercept in order to construct a linear

PO-VO₂ relationship. Using horizontal running or cycling at a fixed PO and low pedalling frequency is advised as it minimizes the VO₂ slow component. This would then enable the determination of the MAOD during a supramaximal exercise protocol, thus creating a more valid PO-VO₂ relationship resulting in reliable data (Noordhof et al., 2010).

Tabata et al. (1997) concluded that HIIE can be used to determine the anaerobic capacity. The MAOD of HIIE was calculated by subtracting the difference between the VO₂ demand at rest and the VO₂ during active resting periods from the VO₂ deficit of each exercise bout. The determination of anaerobic capacity depends on the duration of the intervals along with the duration of resting periods (Tabata et al., 1997). However, is the MAOD method a valid protocol in determining the anaerobic capacity? Bangsbo et al. (1990) assessed the validity of the MAOD method as they compared the MAOD attained by untrained males during supramaximal, constant intensity, one-legged knee-extensions where only the quadriceps muscles were activated. A muscle biopsy of the activated muscle was used to help determine changes in ATP, creatine phosphate, inosine monophosphate and lactate concentrations. In addition, lactate efflux and VO₂ of the quadriceps was estimated from measurements of blood flow and the femoral arterial-venous difference for lactate and oxygen. Bangsbo et al. (1990) concluded that the MAOD is a quantitative expression of the anaerobic capacity when exercising a single muscle group. Green, Dawson, Goodman, & Carey (1996) also examined the validity of the MAOD method in well-trained male cyclist by examining the relationship between MAOD, the amount of anaerobic ATP produced, and measures of the anaerobic potential of muscles. They concluded that the estimates of muscle anaerobic ATP production or anaerobic potential were not related to the accumulated O₂ deficit. The use of the VO₂ power regression to estimate the energy demand of intense cycling is the predicted primary source of error in the O₂ deficit method. Medbø &

Tabata (1993) found a strong correlation between the rates of anaerobic ATP turnover in active muscles along with the anaerobic energy production in the entire body measured by the MAOD method. Medbø & Tabata (1993) took a similar approach to Green et al. (1996) methods, where they gathered ATP, PCr and lactate concentrations from a muscle biopsy in the *M. Vastus Lateralis* after intense cycling bouts. However, Medbø & Tabata (1993) and Green et al. (1996) both used different equations, which did not account for the amount of lactate released in the blood. Considering lactate release can represent between 5% to 38% of total anaerobic ATP production, Bangsbo (1996), Green et al. (1996) and Medbø & Tabata (1993) all underestimated the amount of anaerobic ATP produced during their exercise protocols. It is also unknown if the measured muscle metabolite concentrations represent a single or a whole group of muscles (Medbø, 1996), which weakens Green et al. (1996) and Medbø & Tabata's (1993) results as the active muscle mass is unknown during whole-body exercise. According to Noordhof et al. (2010), the most precise assessment of anaerobic energy yield has been conducted by Bangsbo et al. (1990) as they used an intense dynamic knee-extension exercise. Their results conclude that isolating a specific group of muscles is the most valid way of determining the anaerobic capacity when using the MAOD method.

Relatively short submaximal exercise bouts are ideal for a construction of linear PO and VO₂ relationship, as a secondary increase will arise after 3 minutes of exercise at the highest submaximal exercise intensities. Noordhof et al. (2010) recommend ten submaximal exercise tests at intensities evenly distributed between 30% and 90% VO_{2max}. Increasing the duration of submaximal exercise bouts results in increasing values of VO₂ demand and MAOD (Bangsbo, 1992, 1996; Buck & McNaughton, 1999; Maxwell & Nimmo, 1996). Using a linear relationship between exercise intensity and VO₂ for the determination of anaerobic capacity is not suggested

according to the conclusions of Bangsbo (1992) and Bangsbo (1996). With that being said, 10x4 minutes of submaximal exercise bouts with a fixed y-intercept have been found to construct a solid PO and VO₂ relationship as it enables a steady-state VO₂ and excludes the effect of the VO₂ slow component at intensities above lactate threshold (Bickham, Le Rossignol, Gibbons, & Russell, 2002; Noordhof et al., 2010). Despite these explicit methods, Bangsbo (1996) and Noordhof et al. (2010) have concluded that it is difficult to validate the proper use of linear relationship between exercise intensity and VO₂ in order to establish the anaerobic capacity.

2.2.8 Gross Efficiency

Gross efficiency (GE) has been defined as the ratio of work produced to the total metabolic energy cost (Joyner & Coyle, 2008). In other words, mechanical work (kJ) divided by total metabolic cost (kJ) results in a GE ratio. In order to establish someone's GE, the subject performs a steady-state submaximal exercise where their GE can be determined before the start of a supramaximal exercise bout (Noordhof et al., 2013a). The metabolic power input (PI) can be calculated from the VO₂ (l/min⁻¹) and the oxygen equivalent (Garby & Astrup, 1987; van Ingen Schenau & Cavanagh, 1990) $PI = VO_2 \times (4940 \times RER \times 16,040)$. Anaerobic EE can be estimated, if GE is known, as it uses the aerobically attributable mechanical power and subtracts it from the total power produced, resulting in the anaerobically attributable mechanical power (de Koning, Noordhof, Lucia & Foster, 2012). In essence, mechanical work (kJ) divided by GE (%) equals total metabolic cost (kJ). Anaerobic EE can then be estimated by subtracting aerobically attributable EE from total metabolic cost. Keep in mind that PO, VO₂, RER and GE must be known variables. High intensity exercises such as time trials (Foster et al., 2004; Foster et al.,

2003) and/or constant PO bouts (Noordhof et al., 2011) can be used to estimate the anaerobic power. Recent research has shown that a linear relationship between work rate and EE appears to be consistent, (Ettema & Lorås, 2009; de Koning et al., 2012) and that GE decreases during submaximal (60% $\text{VO}_{2\text{max}}$) (Passfield & Doust, 2000) and supramaximal exercise (de Koning et al., 2012). The decrease in GE during prolonged submaximal and supramaximal exercise is correlated with fatigue and appears to be dependent on the length and intensity of the exercise protocol (Noordhof, Mulder, Malterer, Foster, & de Koning, 2013b). The GE can be underestimated if it is determined at an intensity significantly lower than the ventilatory threshold leading to an overestimation of the anaerobically attributable energy (Noordhof et al., 2013b). When the GE is predicted during graded exercise, it seems that the length of the exercise stages is relevant as the GE is significantly overestimated during the 1st minute of the exercise stages in comparison with the GE measured during 3 or 6-minute stages (de Koning et al., 2012). Repeated sprints can alter the GE since efficiency is also influenced by the duration of the recovery (Bailey, Vanhatalo, Wilkerson, Dimenna, & Jones, 2009; Burnley, Doust, & Jones, 2006). Constant GE during heavy exercise may be questionable as it can easily underestimate the anaerobic EE (de Koning et al., 2013; Noordhof et al., 2013a). With the use of back-extrapolation, de Koning et al. (2013) found that GE diminished from 18.3% to 15.8% after 4 minutes of cycling at 100% of peak PO. Near the end of the exercise, the 15.8% GE lead to a 32% larger anaerobic contribution than GE set at 18.3%. During 30s Wingate, efficiencies of 16.2% (Serresse et al., 1988), 18.5% (Smith & Hill, 1991), 22% (Davies & Sandstrom, 1989) and 25% (Gaesser & Brooks, 1975; Kavanagh & Jacobs, 1988) have been used. Any value utilized is a best guess since efficiency or economy can only be precisely measured during SS exercise (Smith & Hill, 1991). With that being said, previous studies (Davies & Sandstrom,

1989; de Koning et al., 2013; Gaesser & Brooks, 1975; Kavanagh & Jacobs, 1988; Serresse et al., 1988; Smith & Hill, 1991) suggest that 18% is an acceptable GE value to use during 30s sprints.

2.3 Steady State vs High Intensity Interval Exercise

HIIE is defined as brief and repeated burst of intense exercise with low intensity resting periods, whereas low volume HIIE consists of less than 10 minutes of intense exercise within a 30 minute window (Gillen & Gibala, 2014). Benefits that come from this type of training includes increase in whole-body fat oxidation during exercise (Rakobowchuk et al., 2008), VO_{2max} (Gillen et al., 2013), resting fat oxidation (Trapp et al., 2008) along with reductions in whole-body and abdominal fat mass (Gillen et al., 2013), decreases in arterial stiffness, systolic and diastolic blood pressure (Heydari et al., 2012). Steady state exercise (SSE), also known as aerobic exercise, is defined as intensities varying from 60% of one's heart rate max for a duration exceeding 20 minutes. This type of training leads to multiple physiological adaptations, which include the enhancement of the individuals physical fitness and recovery rate (Sloan et al., 2011), VO_{2max} (Hottenrott, Ludyga, & Schulze, 2012), endurance capacity (Green, Ball-Burnett, Symon, Grant, & Jamieson, 1995) along with a decrease in body fat (Hottenrott et al., 2012).

There has been some controversy in recent years when comparing the physiological adaptations from HIIT and steady state training (SST). Results from Keating et al. (2014) suggest that SST is more effective for improving fat oxidation compared to HIIT in overweight adults. Hazell, Olver, Hamilton, & Lemon (2012) focused their study on young men and had them complete either four 30s sprints on a cycling ergometer (25-minute total duration) or 30

minutes of SS cycling at ~70% of their VO_2 max (37-minute total duration) three times per week for six weeks. Their findings indicate that EE of a single HIIT is typically lower than SST due to the total exercise duration, in other words, less oxygen was consumed during the HIIT protocol. Nybo et al. (2010) compared interval running to SS running in thirty-six untrained physically inactive men. The HIIT group completed five sets of 2 minute intervals above 95% of HR_{max} over a 12 week period while the SS group performed 60 minutes of running at 80% of HR_{max} (~65% VO_{2max}). Participants were supposed to complete three exercise sessions per week; however, the HIIT and SS group averaged 2 and 2.5 weekly sessions, respectively, due to injuries (i.e. shin splints). On the other hand, Burgomaster et al. (2008) and Nalcakan (2014) both had very similar approaches and found that HIIT and SST lead to similar adaptations such as an increment in mitochondrial markers for skeletal muscle CHO and lipid oxidation (Burgomaster et al., 2008), increase in VO_{2max} and decrease in body fat and waist circumference (Nalcakan, 2014). This is interesting considering that results come from 6 weeks of training where weekly time commitment differed from 1.5h to 4.5h in the HIIT and SS group, respectively (Burgomaster et al., 2008). No differences were found in obese children, as both HIIT and SS increased their VO_{2max} , peak velocity during maximal graded cardiorespiratory test, improved insulinemia levels and BMI (Corte de Araujo et al., 2012). Unlike Keating et al. (2014), Hazell et al. (2012) and Nybo et al. (2010) found that HIIT is a more productive method for obtaining health benefits when compared to SST. Trapp et al. (2008) compared SST and HIIT in young woman during a 15-week workout program on a cycling ergometer. The HIIT group performed 8s sprints followed by 12s of active recovery, repeated for 20 minutes compared to the 40-minute SS exercise performed at 60% VO_{2max} . Although both exercise protocols led to cardiovascular adaptations, HIIT was more effective for decreasing fasting insulin levels, reducing total body mass, fat mass and trunk fat, but it did so in half the time. A significant rise of catecholamines is

found during intense exercise unlike SST, which results in an increase of epinephrine and norepinephrine (Zouhal, Jacob, Delamarche, & Gratas-Delamarche, 2008). The release of catecholamines, but more importantly epinephrine, has been shown to drive lipolysis, which is responsible for fat release from subcutaneous and intramuscular fat stores (Issekutz & Recent, 1978). HIIT was also compared to SST in a sample of diabetic participants (16 males, 29 females) for a duration of 12 weeks (Mitranun, Deerochanawong, Tanaka, & Suksom, 2014). Although both protocols resulted in significant improvements, HIIT was more effective for improving VO_{2max} , vascular functions along with peak diameter of the brachial artery. Matsuo et al. (2014) wanted to determine which exercise protocol could improve the participants' cardiorespiratory capacity and/if it would have an effect on their left ventricular mass. Their approach was unique as they compared sprint interval training with the traditional continuous exercise, but integrated high intensity aerobic exercise. Forty-two sedentary men took part in 8 weeks of training five times per week and were divided into one of three groups. The sprint interval, high intensity aerobic and SS group spent on average 5 minutes (100kcal), 13 minutes (180kcal) and 40 minutes (360kcal), respectively, on a cycling ergometer during each of their exercise sessions. Despite having an inferior EE and time commitment, sprint interval training and high intensity aerobic training both led to an increase in left ventricular mass. Matsuo et al., (2014) suggest that exercise intensity rather than duration and volume can improve VO_{2max} , considering the aerobic interval group in their study had a significant increase in oxygen consumption when compared to the SS protocol. O'Donovan et al. (2005) compared interval and continuous training protocols to assess the effect of intensities on cardiorespiratory fitness and coronary heart disease in sedentary men. They randomly assigned the subjects into a control, SS or HIIT group while matching EE between both exercise protocols. EE was determined from VO_2 , assuming an energy cost of 5kcal/L of oxygen with an end goal of 400 kcal per session.

For example, SS would take 44 minutes while HIIT would require 30 minutes of exercise to reach 400kcal. Participants performed their exercise three times per week for 24 weeks at 60% and 80% of their VO_{2max} in either the SS or HIIT group, respectively. O'Donovan et al. (2005) found that HIIT was more effective for improving cardiorespiratory fitness while having equal energy cost.

Williams et al. (2013) analyzed an acute response of 60 minutes of SSE compared to four 30s Wingate test interspersed by 4.5 minutes of active recovery within young men. They concluded that there was no significant difference between SSE and HIIE during 3 hours of EPOC. However, with the use of a gas analyzer, they found that SSE had a greater EE above baseline levels when combining exercise and EPOC (~560 Kcal) compared to HIIE (~85 Kcal). Malatesta, Werlen, Bulfaro, Chenevière, & Borrani (2009) also compared HIIE with SSE and gathered 3 hours of post exercise data. The interval training consisted of 60s of cycling at 80% W_{max} with 60s of active recovery at 40% W_{max} while SSE was performed at a relative intensity of 45% VO_{2max} for 60 minutes. This protocol was unique considering they matched the mechanical work output between both exercise protocols and found that fat oxidation during EPOC did not differ but was significantly greater than the control group. Both isoenergetic exercises varied in intensity but had a similar shift towards fat utilization during EPOC. Williams et al. (2013) and Malatesta et al. (2009) findings support the notion that EE from the actual exercise is the primary factor that will lead to a decrease in body mass.

Studies completed by Keating et al. (2014), Nybo, Sundstrup, Jakobsen, Mohr, Hornstrup, Simonsen, et al. (2010), Burgomaster et al. (2008), Nalcakan, (2014), Matsuo et al. (2014), Corte de Araujo et al. (2012) and Williams et al. (2013) all compared either interval training/exercise with SS training/exercise but did not match EE between both exercise groups. Other authors

such as, Trapp et al. (2008), Mitranun et al. (2014), O'Donovan et al. (2005), Malatesta et al. (2009) all matched the EE between the HIIT/HIIE and SST/SSE protocol but did not take into account the anaerobic attributable EE.

3. Purpose and Hypothesis

The question remains, which exercise would lead to a greater EE during post exercise measurements when both protocols are equicaloric. The purpose of this research is to determine whether SSE or HIIE will better improve EE during EPOC when both exercise protocols are matched for exercise EE. This will allow us to parcel out the effect of exercise intensity. We hypothesized that HIIE set at 150% of W_{\max} will result in greater EPOC than SSE lasting 45 minutes performed at 60% of $VO_{2\max}$ with both protocols having matched exercise EE.

4. Methods

4.1 Participants:

The study consisted of 12 males free of heart and lung disease aged between 19 and 24. All elite athletes were excluded from this study considering they would likely respond differently to the exercise protocols. Prior to any testing, each participant completed a PAR-Q test and provided informed consent. The design and methods of this study were approved by the University Research Ethics Board.

4.2 Experimental design

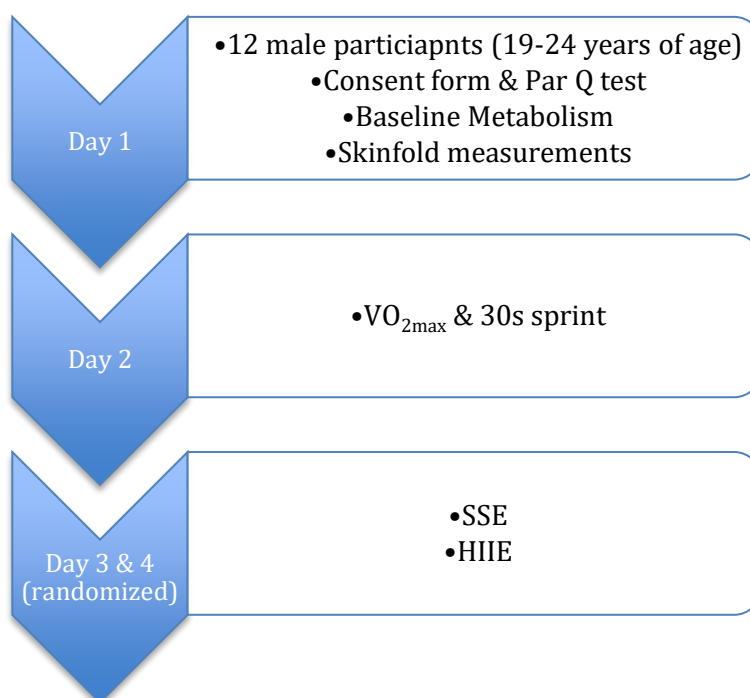


Figure 1: Experimental design

4.2.1 Day 1: Preliminary measurements

Subjects were advised to avoid any strenuous exercise two days prior to collecting preliminary measurements and exercise protocols. Participants were asked to fast 10 hours prior to any basal metabolic rate testing. After completing the consent form and PAR-Q test, the participant's resting heart rate, blood pressure, weight (kg) and height (cm) were measured. The basal metabolism was then measured using a gas analyser (VIASYS) while participants laid in a supine position for 30 minutes. Data from the last 10 minutes was utilized for analysis since it better represents the participants' basal metabolism. A skinfold caliper was used to measure skinfold in triplicate at each of the following sites: right abdomen, iliac crest, triceps, and the thigh. The mean value of each site was used and applied to the *Jackson & Pollock 4-site Caliper* in order to estimate the percentage of body fat (Jackson & Pollock, 1978).

4.2.2 Day 2: VO_{2max} & 30s sprint

Prior to the test to exhaustion, participants were informed that they could stop cycling at any time. They were also informed to maintain a 70 to 80 rpm pace and if they failed to do so, the test to exhaustion would be terminated. The resistance was set at 75W and increased by 25W every 5 minutes. These 5 minute durations are required according to Noordhof et al. (2010) to construct a solid PO and VO₂ relationship. When the subjects' HR approached 75-80% of their estimated HR_{max} (using the Karvonen formula), the intervals were minimized to 2 instead of 5 minutes in order to reach maximal PO. With the use of the *Breeze Suite* software, a *MGC Diagnostics* gas analyzer, a heart rate monitor and a facemask to support the gas analyzer, we

were able to collect data for numerous variables such as; VO_2 ($\text{ml.kg}^{-1}.\text{min}^{-1}$), VO_2 (ml.min^{-1}), VCO_2 (ml.min^{-1}), Respiratory Exchange Ratio {RER}, VE (L.min^{-1}) and HR (bpm). Following the $\text{VO}_{2\text{max}}$, the participants kept cycling at 40% of their W_{max} . Five minutes post $\text{VO}_{2\text{max}}$, participants performed a 30s-all-out sprint at 150% W_{max} . This exercise protocol had a duration of 4 minutes, where the first two minutes consisted of cycling at 40% W_{max} at a rpm between 70-80. Subjects then cycled at 150% W_{max} for 30s while keeping the rpm no higher than 145. We began to increase the resistance 5 seconds prior to the sprint since the ergometer could only increase the resistance by 25W. For the following 1.5 minutes, the participants cycled at 40% W_{max} while keeping their rpm between 70 and 80.

4.2.3 Days 3 & 4: Exercise protocols

Following day 2, subjects randomly performed either SSE or HIIE protocols interspersed by at least 2 days. Both exercise protocols had a 5-minute warm up and cool down set at 40% W_{max} . Data from the 5-minute cool down was integrated as a part of the 90 minutes of EPOC.

SSE was conducted at a resistance corresponding to the participant's 60% $\text{VO}_{2\text{max}}$. For example, if 60% $\text{VO}_{2\text{max}}$ occurred at 125W during the $\text{VO}_{2\text{max}}$, the SSE protocol would begin at 125 watts. Taking into account the effects of cardiovascular drift, we had to diminish the resistance during the course of SSE in order to keep participants' VO_2 at 60%. The mean resistance for 60% $\text{VO}_{2\text{max}}$ was 124 ± 15 Watts. We estimated the SSE EE (EE_{SSE}) by taking VO_2 (l/min^{-1}) and RER values from the $\text{VO}_{2\text{max}}$ exercise protocol. By multiplying the VO_2 (l/min^{-1}) by the equivalent kilojoules found at a given RER and then multiplying by 45 minutes we were

able to quantify the estimated EE (e.g. $2.1 \text{ (LO}_2\text{.min}^{-1}) * 20.25 \text{ KJ/LO}_2 \text{ (RER} = 0.83) * 45 \text{ minutes} = 1,913 \text{ Kilojoules}$) {Annexe 1}.

The HIIE protocol consisted of 30s all-out sprints with 60 seconds of active recovery. The intensities were set at 150% and 40% of the W_{max} established from the $VO_{2\text{max}}$ (Annex 2). If the test to exhaustion was terminated at 300 watts, the HIIE protocol would consist of 30s sprints at 450 watts with 60s of active recovery set at 120 watts. The mean resistance during the 30s sprints was $346 \pm 32 \text{ Watts}$. The 30s all out sprint performed 5 minutes after the $VO_{2\text{max}}$ helped to inform the EE attributable to aerobic pathways. We were able to estimate the anaerobic and aerobic energy pathway contributions during the 30s all out sprint with the use of a GE set at 18% along with the AOD at a relative resistance (EE_{sprint}). Total metabolic energy equals mechanical work ($450\text{w} * 30\text{s} = 13.5 \text{ kJ}$) divided by 18% (GE). By combining the EE_{sprint} with that from the active recovery we could estimate the EE of one HIIE repetition (EE_{HIIE}). The number of repetitions that each participant had to complete in a HIIE protocol was found by dividing the EE_{ss} by the EE_{HIIE} , which was performed after the $VO_{2\text{max}}$. Only the GE method was used to quantify the number of HIIE repetitions.

4.3 Measurements

4.3.1 EPOC

After both exercise protocols, each individual remained seated while the ‘breath by breath’ measurements were collected for 90 minutes EPOC. With the use of the gas analyzer

(MGC Diagnostics) attached to the face, all the dependant variables (VO_2 (relative/absolute), VCO_2 , RER, VE and HR) were recorded. The EE from EPOC (EE_{EPOC}) was calculated by averaging the data in 5 minute intervals and multiplying the mean of the VO_2 and energy equivalent derived from RER. The sum of all these averages was used to find the EE_{EPOC} for 90 minutes. To analyze the subsequent changes over time, EPOC was divided into 18 time intervals (5 minute averages over 90 minutes).

4.3.2 Blood lactic acid

The blood lactic acid was measured prior to (BLCP) and halfway through (BLCH) the exercise protocol as well as immediately after (BLC0), 3 minutes (BLC3) and 5 minutes (BLC5) following the HIIE protocol. We pricked either the index or the digitus medius with a BD Microtainer Contact-Activated Lancet to collect the blood drops. The blood was then collected with a Lactate Pro Test Strip and analyzed using a ARKRAY Blood Lactate Test Meter.

4.3.3 Substrate utilization

The average VO_2 and RER during EPOC for each individual were determined. The mean RER value determined the quantity of grams of fat and carbohydrates utilized per liter of O_2 /minute. The energy equivalent for a given RER was multiplied by the mean VO_2 then again by 90 minutes to determine how much fat and carbohydrates each individual expended during EPOC. For example, a mean VO_2 of 0.85L and RER of 0.84 would entail the use of 537mg of carbohydrates and 280mg of fat per L/ O_2 . Multiplying the mg of sugar and fat by 0.85,

multiplying it again by 90 minutes and dividing it by 1000 would translate to a usage of 41.08g of carbohydrates and 21.42g of fat during 90 minutes of EPOC.

5. Results

Statistical analysis was completed with the IBM SPSS Statistics 20 software. A paired samples T-test was used to compare gross and net exercise EE during EPOC, the combined exercise and post exercise EE along with net exercise fat and carbohydrate utilization during EPOC. A one-way repeated measures ANOVA was used to compare exercise EE, anaerobic and aerobic pathways during HIIE (14 intervals), BLC, gross fat and carbohydrate utilization during EPOC as well as VO_2 , RER, HR, VE and EE, which were further divided into 5 minute intervals during EPOC. A Bonferroni post-hoc test was then used to identify where the differences occurred during measurements.

Table 1: Subject characteristics

Variables	MEANS	Standard Deviation
Age	23	1.21
Height (cm)	176.33	7.35
Weight (kg)	82.89	12.82
BMI	26.61	3.37
Resting HR	60.25	8.7
Maximal HR	188.67	11.65
Systolic Blood Pressure (mmHg)	113.83	9.96
Diastolic Blood Pressure (mmHg)	74.83	8.42
Body fat %	15.14	4.55
VO2 max (l/min-1)	3.55	0.46
VO2 max (ml/kg-1/min-1)	43.12	4.00

A significant difference between the exercise protocols in regards to energy (kJ) was identified { $F(1.294, 14.229) = 8.454, p < 0.01$ }. These differences occurred between the HIIE protocol (M = 2040 kJ, SD = 212 kJ) and SSE (M = 2173 kJ, SD = 244 kJ) where the mean

differed by 133 kJ (Figure 2). Relatively speaking, SSE expended roughly 6% more energy compared to HIIE.

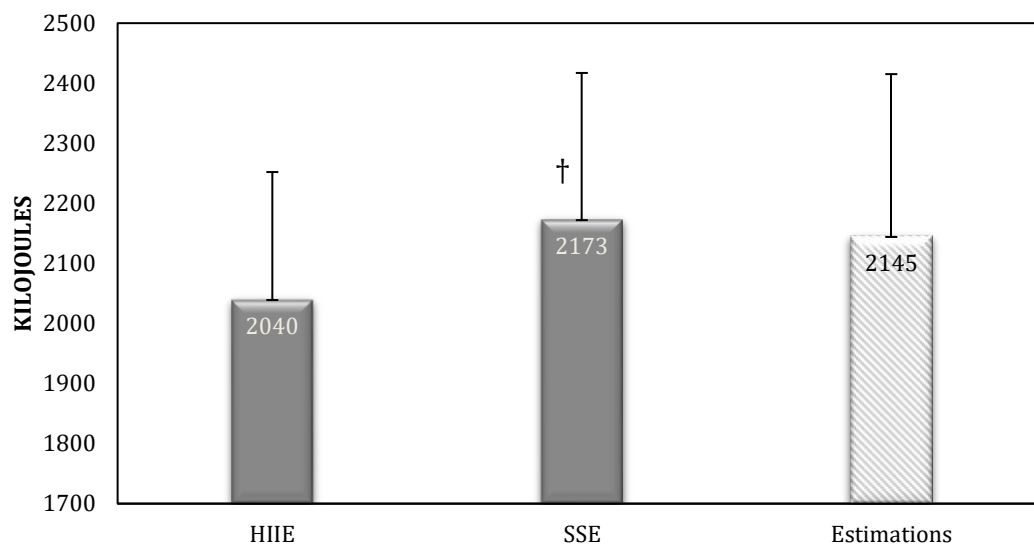


Figure 2: Exercise energy expenditure during HIIE, SSE and theoretical EE estimations.

† = significantly different from HIIE ($P < 0.05$)

Significant differences between the anaerobic and aerobic systems in regards to absolute and relative energy contributions was identified { $F(1.000, 11.000) = 35.724, p < 0.01$ } (Figure 3A) and { $F(1.000, 11.000) = 37.352, p < 0.01$ } (Figure 3B), respectively. These differences occurred at each repetition during the HIIE protocol. The anaerobic system provided 60% of the energy during the first interval but rapidly declined as the aerobic system was the predominant source of energy for the remaining 13 intervals.

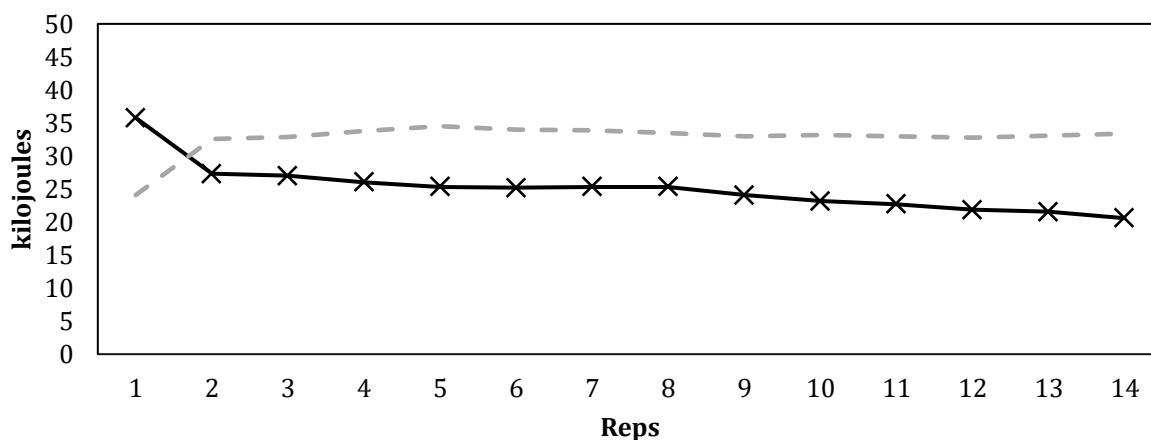
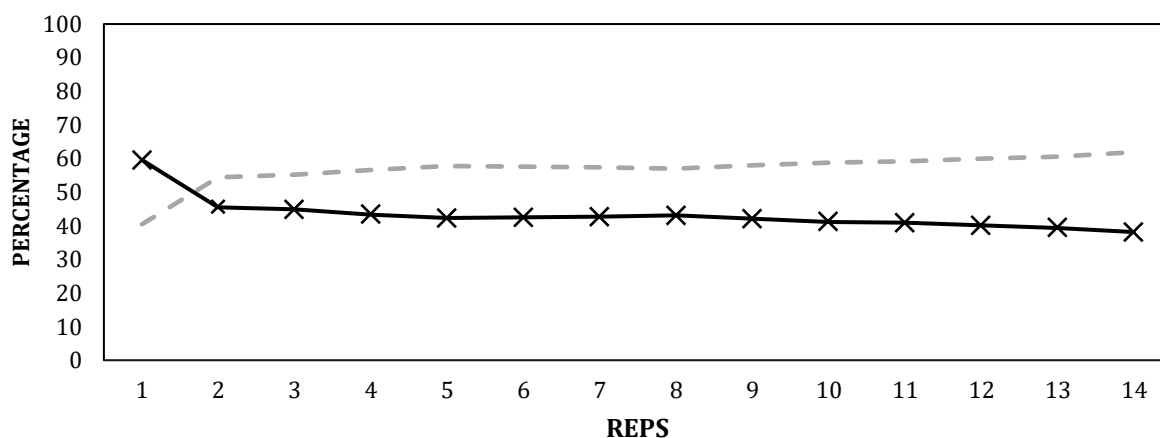
A**B**

Figure 3: The absolute (A) and relative (B) value of aerobic (--) EE and anaerobic (-) EE for each HIIE repetition.

X = Anaerobic contributions are significantly different from aerobic contributions ($P < 0.05$).

A significant difference was found between BLC measurements { $F(1.764, 14.113) = 87.492, p < 0.01$ } (Figure 4). Pre-Test BLC differed from the other four measurements ($P < 0.05$). These results indicated a large portion of the energy was derived from anaerobic pathways during HIIE. Another significant difference was identified between 0 Min Post and 5 Min Post BLC ($p < 0.05$). BLC gathered immediately after the HIIE protocol was significantly greater than the BLC gathered 5 minutes post exercise.

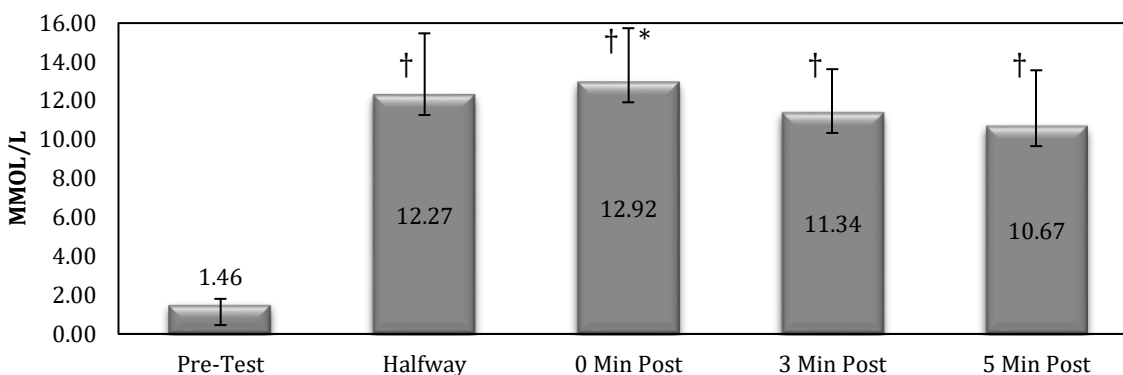


Figure 4: Blood lactate concentrations measured at pre HIIE, halfway through, 0-minute post, 3-minute post and 5-minute post exercise.

† = significantly different from Pre-Test ($P < 0.05$)

* = significantly different from 5-minute post ($P < 0.05$)

A significant differences between gross and net EE during post exercise measurements following HIIE and SSE. The interval exercise had a greater gross EE (993 kJ) during 90 minutes of EPOC as opposed to SSE (848 kJ) , { $t(11) = 4.221$, $p < 0.01$ } (Figure 5). The net effect of exercise (total EE – basal metabolism) remained significantly greater for HIIE, { $t(11) = 4.221$, $p < 0.01$ }.

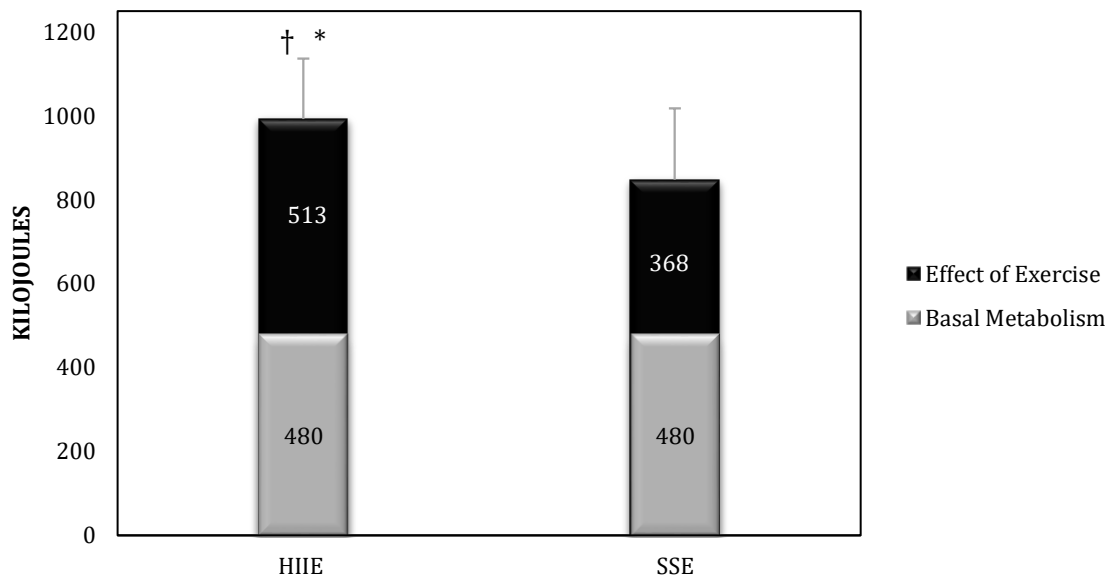


Figure 5: Post exercise EE above basal metabolism for HIIE and SSE during 90 minutes of EPOC.

† = Net exercise EE is significantly different from SSE ($P < 0.05$)

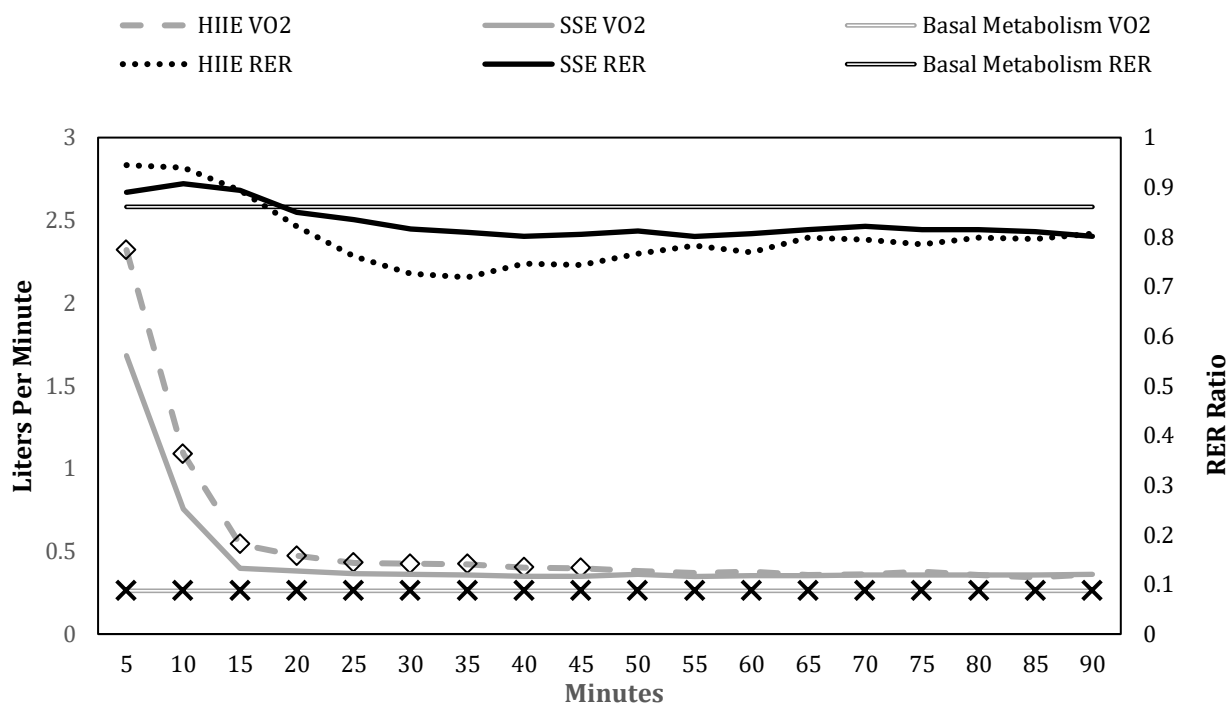
* = Gross EE is significantly different from SSE ($P < 0.05$).

A significant difference occurred between HIIE and SSE during 90 minutes of EPOC in regards to VO_2 (Figure 6A), HR and VE (Figure 6B). The exercise protocols elicited a significant difference between VO_2 { $F(2, 22) = 121.515, p < 0.01$ }, HR { $F(2, 22) = 77.410, p < 0.01$ } and VE { $F(2, 22) = 129.038, p < 0.01$ }. The RER between exercises was not significantly different during EPOC, { $F(1.195, 13.148) = 3.785, p > 0.05$ }. Both exercise protocols maintained a significantly greater VO_2 during the entire 90 minutes when compared to the Basal Metabolism (Figure 6A). The interval exercise was able to maintain a greater VO_2 for the first 45 minutes post exercise in comparison to SSE.

The high intensity interval protocol was associated with significantly greater HR than Basal Metabolism throughout the entire 90 minutes of EPOC. HIIE also differed from SSE as it was significantly higher throughout the entire post-exercise protocol with the exception of minute

30. SSE HR exceeded Basal Metabolism HR values for the full length of EPOC, aside from the 20th minute (Figure 6B). HIIE also generated a significantly greater VE during the first 45 minutes when compared to SSE, while the VE for both exercise protocols surpassed that of Basal Metabolism levels throughout the entire 90 minutes.

A



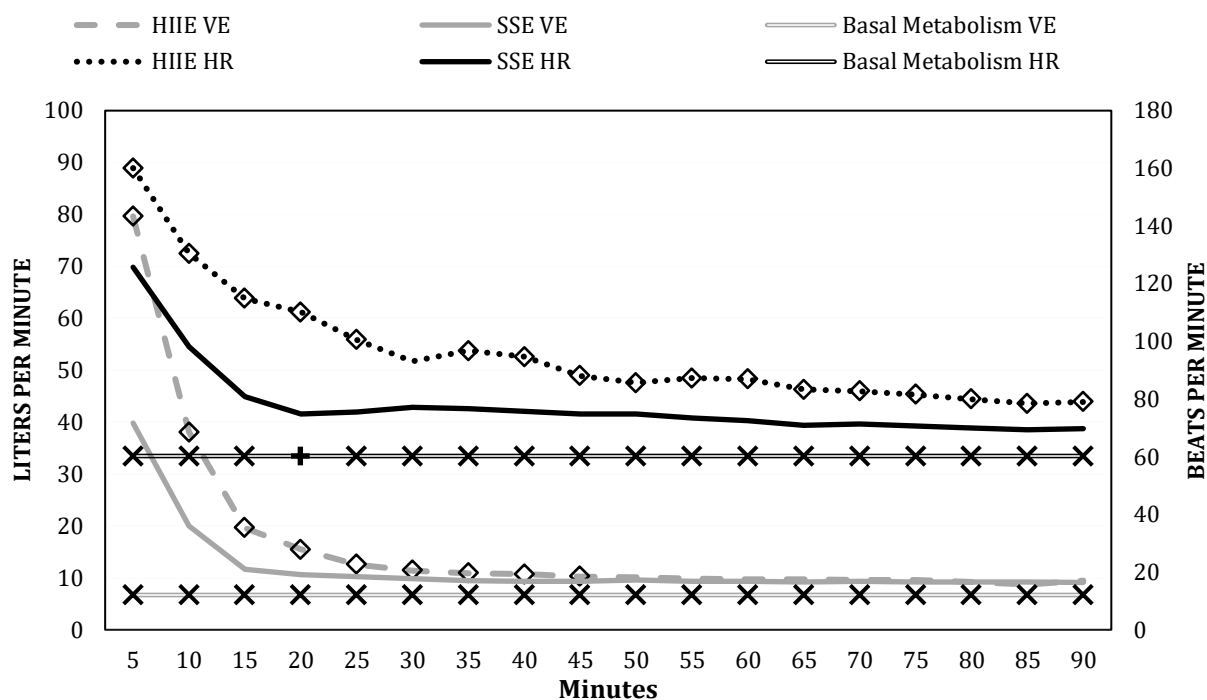
B

Figure 6: Post exercise VO_2 and RER (A) and VE and HR (B) over 90 minutes averaged in 5-minute intervals.

◇ = HIIE is significantly different from SSE ($P < 0.05$).

✕ = Basal Metabolism is significantly different from HIIE and SSE ($P < 0.05$).

✚ = Basal Metabolism is significantly different from HIIE ($P < 0.05$).

A significant difference was found between HIIE and SSE during EPOC, $\{F(1.000, 11.000) = 17.520, p < 0.01\}$. These differences occurred during the first 45 minutes of EPOC ($P < 0.05$), where HIIE expended more energy than SSE (Figure 7). No differences were found during the second half of EPOC.

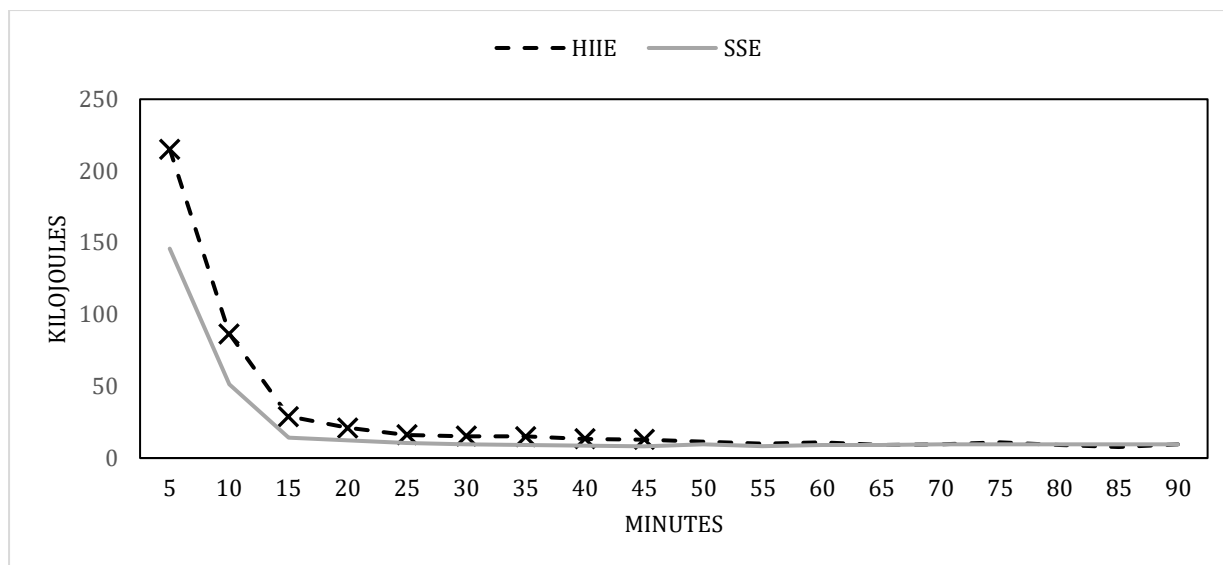


Figure 7: Post exercise EE over the course of 90 minutes averaged in 5-minute intervals.

X = HIIE is significantly different from SSE ($P < 0.05$).

A significant difference between HIIE (3033 ± 321 KJ) and SSE (3021 ± 399 KJ) ensued when combining the exercise and post exercise EE, $\{t(11) = 0.180, p > 0.05\}$ (Figure 8). HIIE utilized less energy during exercise but was able to expend more energy than SSE during EPOC, thus making their total EE equivalent.

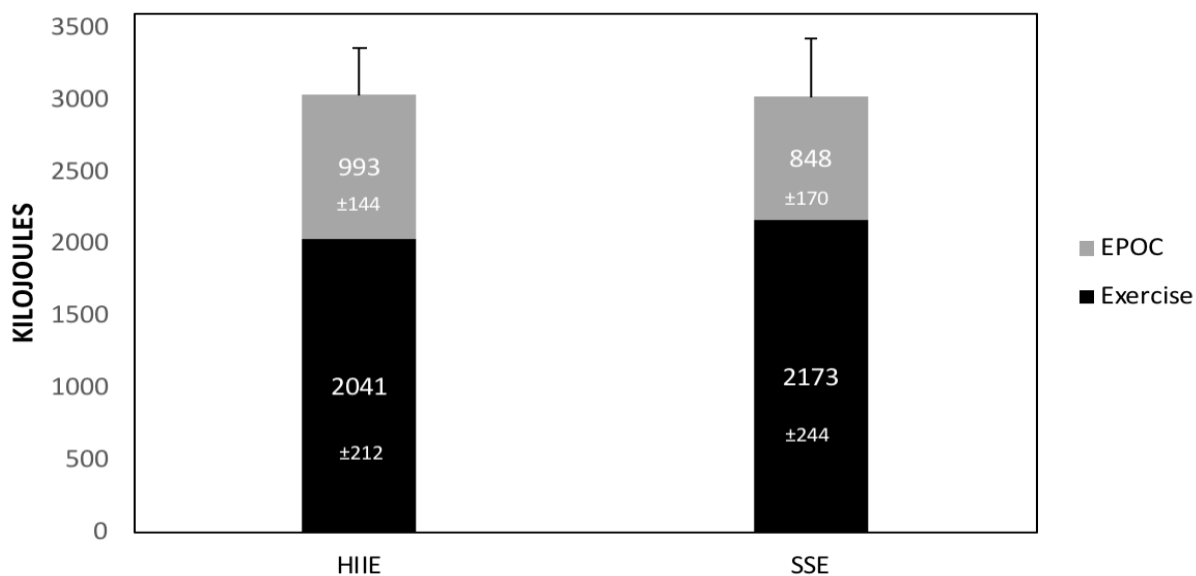


Figure 8: Total exercise EE and post exercise EE from HIIE and SSE.

A significant difference was identified in regards to post exercise fat utilization between exercise protocols, {F (2,22) = 69.397, $p < 0.01$ }. Differences ($P < 0.05$) were found between each protocol where HIIE expended 26% and 66% more fat above SSE and Basal Metabolism, respectively (Figure 9). No significant differences were found in post exercise carbohydrate utilization between exercise protocols, {F (2, 22) = 2.574, $p > 0.05$ }.

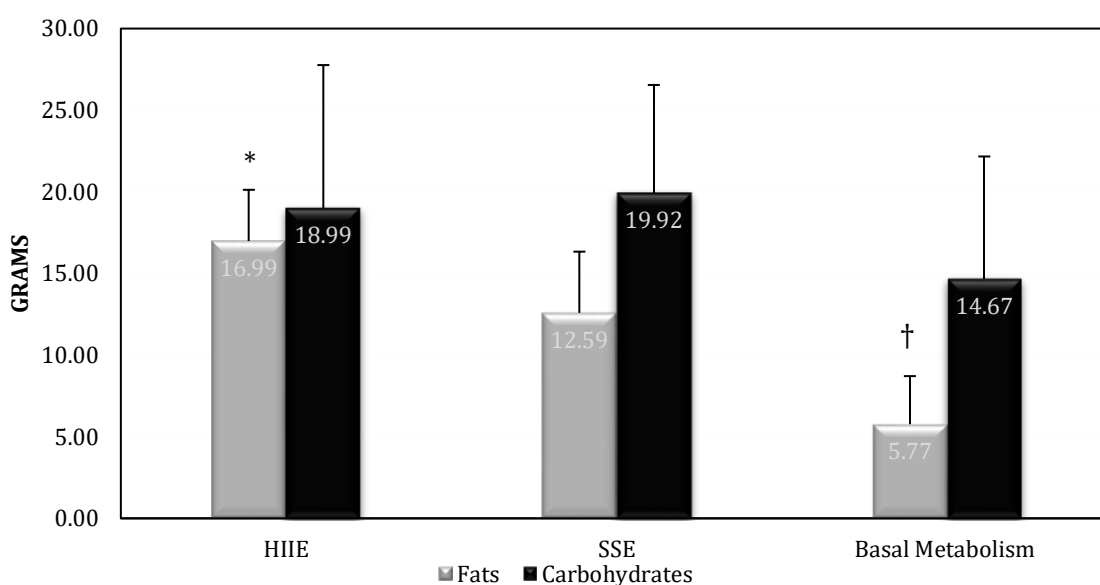


Figure 9: Fats and carbohydrates utilized during 90 minutes of EPOC.

* = HIIE is significantly different from SSE ($P < 0.05$).

† = HIIE & SSE are significantly different from Basal Metabolism ($P < 0.05$).

A significant difference was found between net fat utilization during post exercise measurements. HIIE net fat (11.2g) utilization during EPOC was significantly greater than SSE (6.8g), {t (11) = 4.249, $p < 0.01$ } (Figure 10). No significant differences occurred in terms of net

carbohydrate expenditure between both exercise protocols during EPOC, $\{t(11) = -0.415, p > 0.05\}$.

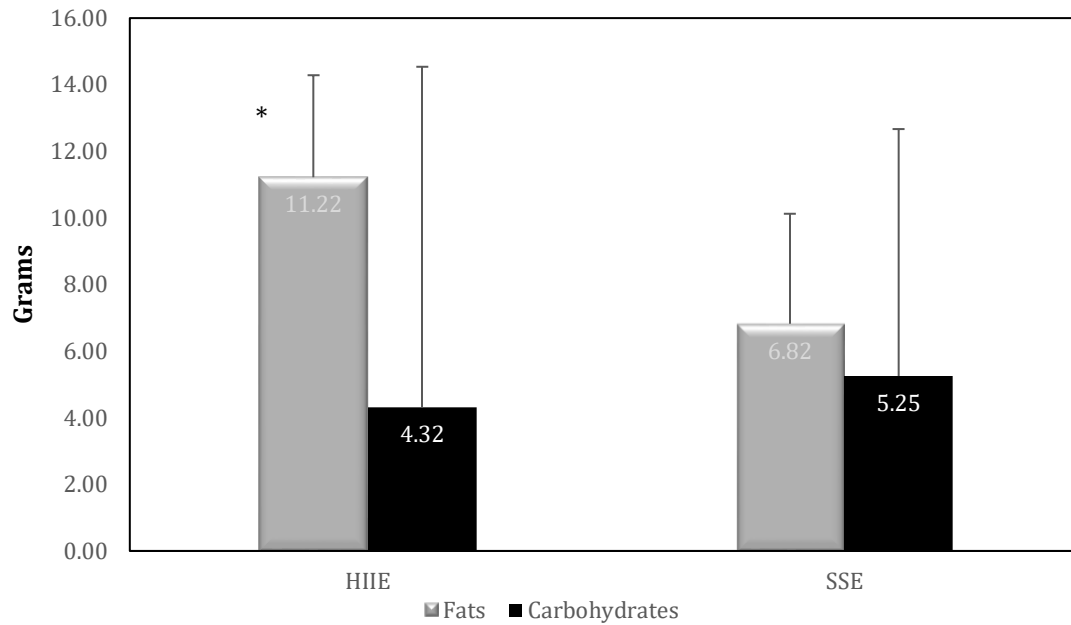


Figure 10: Net exercise fat and carbohydrate utilization during 90 minutes of EPOC (N=11).

** = HIIE is significantly different from SSE ($P < 0.05$).*

6. Discussion

The primary aim of this study was to establish whether SSE or HIIE would generate a greater EPOC while matching exercise EE between both protocols. We also wanted to analyze the effects of these exercise protocols on physiological parameters, such as VO_2 , RER, VE and HR over the course of 90 minutes of post exercise measurements. Our methodological approach differed from many prior studies considering we quantified both the aerobic and anaerobic attributable EE in order to attempt to have two equicaloric exercise protocols, which would enable us to parcel out the effect of exercise intensity. Many authors compared high intensity exercise/training with SSE/SST but did not match exercise EE (Burgomaster et al., 2008; Corte de Araujo et al., 2012; Keating et al., 2014; Matsuo et al., 2014; Nalcakan, 2014; Nybo et al., 2010; Tucker, Angadi, & Gaesser, 2016; Williams et al., 2013), whereas others matched both exercise protocols but neglected the anaerobic attributable EE by simply measuring the aerobic system via indirect calorimetry (Malatesta et al., 2009; Mitranun et al., 2014; O'Donovan et al., 2005; Trapp et al., 2008). It is important to mention that our subjects' characteristics greatly resembled those from previous studies that evaluated the effects of HIIE/HIIT (Greer, Sirithienthad, Moffatt, Marcello, & Panton, 2015; Malatesta et al., 2009; Matsuo et al., 2014; Nalcakan, 2014; Tucker et al., 2016; Williams et al., 2013). We attempted to match SSE EE with HIIE EE prior to the actual exercise protocols using data from the subjects' $\text{VO}_{2\text{max}}$, 30s sprint and the 60s-recovery phase. Our results indicated that we accurately predicted both HIIE (2040 ± 212 kJ) and SSE (2173 ± 244 kJ) to our pre exercise estimations (2145 ± 270 kJ) as no significant differences were identified. With that being said, HIIE and SSE EE did differ from one another (Figure 2).

Anaerobic contributions can be estimated with the use of the GE method as it measures the attributable aerobic mechanical power and subtracts it from total PO resulting in anaerobic attributable mechanical power (Noordhof et al., 2013a). The GE in constant loading during submaximal exercise is calculated by dividing the measured mechanical PO by the calculated metabolic rate (Nalcakan, 2014). Exercise above the anaerobic threshold leads to a negative relationship between GE and work rate, which indicates that a decreasing GE leads to a greater exercise EE (Luhtanen, Rahkila, Rusko, & Viitasalo, 1987). For example, anaerobic EE is found by subtracting the aerobic attributable energy from the PO. The PO is found by multiplying the resistance (Watts = joules/second) by the sprint duration, then multiplied by 100 and divided by the GE. If that GE value is lowered, it would imply that the PO will be greater than a PO which utilized a higher GE. Assuming that the aerobic EE remains constant, the anaerobic attributable EE will increase in order to respond to a greater energy demand from the body. GE has been shown to decrease during submaximal exercise set at 60% VO_{2max} (Passfield & Doust, 2000), during maximal time-trials where its reduction was negatively related to race distance (Noordhof, Mulder, Malterer, Foster, & de Koning, 2015), during 3-minutes of intense exercise at a constant intensity (Saltin, Bangsbo, Krustup, & Krustup, 2001) and after 4 minutes of 100% peak PO where GE diminished from 18.3% to 15.8% (de Koning et al., 2013). However, there is a possibility that the usage of a constant GE of 18% is actually an inferior percentage to what may have transpired during exercise. Considering 16-24% are reasonable GE values during a 30-s Wingate (Smith & Hill, 1991), it is plausible to assume that our GE value was simply too high, which would imply that our HIIIE EE values are inferior to their absolute values. de Koning et al.(2013) compared the aerobic and anaerobic PO for a constant and decreasing GE during cycling exercises. They found that energy generated from anaerobic pathways were more

elevated with decreasing GE compared to constant GE conditions. Considering that a decreasing GE increases total exercise EE, the anaerobic attributable EE would increase if $\text{VO}_{2\text{max}}$ was attained. Shorter races with greater PO have been shown to have a lower GE accompanied by a larger decline in terms of percentage when compared to longer distances. This can be rationalized by greater homeostatic disturbances due to incremental physiological demands, thus a higher PO and lower GE (Noordhof et al., 2015).

The approach in the current study consisted of 18% GE since many authors such as Serresse et al. (1988), Smith & Hill (1991), Davies & Sandstrom (1989), Kavanagh & Jacobs (1988) and Gaesser & Brooks (1975) used similar GE for the 30s Wingate protocol. Similar to Bogdanis, Nevill, Boobis & Lakomy (1996), our results reveal that energy contributions rely mainly on the anaerobic system during the first 30s of supramaximal cycling, however, we noticed an energy shift towards the aerobic system throughout the repetitions (Figure 3A). It is important to mention that our exercise protocol had a mean PO of 346W (140% W_{max}) with only 60s of active recovery. The anaerobic system represented 59.56% of the attributable energy during the first repetition and steadily dwindled to 38.12% during the fourteenth repetition (Figure 3B). Although the individuals were dependent of the aerobic system near the end of their respective workouts, BLC were similar at the midpoint and after the last repetition revealing that anaerobic pathways were still active throughout the entire workout. When comparing SSE and HIIE, it is imperative that BLC is measured in order to confirm the contributions of anaerobic pathways to EE.

The anaerobic system is comprised of two energetic pathways known as the alactic and lactic system. Together, they are able to provide sufficient energy during the first 6 seconds of

sprint exercise where as much as 50% of that energy is derived from ATP-PC (Lakomy, 2000). Nonaerobic breakdown of carbohydrates and glycogen succeeds the exhausted ATP-PC system resulting in lactic acid (Gastin, 2001), which subsequently increases throughout the body due to metabolic acidosis (Sahlin, 1978). For this reason, BLC was measured during pre-test, halfway through, immediately after, 3 and 5 minutes post exercise of the HIIE protocol. BLC has been shown to peak 6 minutes following the completion of repeated 30s sprint (13.6 ± 0.9 mmol.L⁻¹) (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995). Our results reveal that BLC₀ (13.00 ± 2.54 mmol.L⁻¹) was greater than BLC₅ (10.83 ± 2.69 mmol.L⁻¹) (Figure 4), which is in accordance with Williams et al. (2013) as they found a BLC₀ of 12.8 ± 1.9 mmol.L⁻¹ after four 30s Wingate tests. An analysis done by di Prampero & Ferretti (1999) focused on energy release deriving from the anaerobic pathways during supramaximal exercise. Their findings support the original notion developed by Margaria et al. (1933), confirming that the alactic and lactic systems provide the necessary energy for exercises that surpass the aerobic capacity.

Many studies have confirmed that exercise duration effects EPOC length, whereas exercise intensity plays a large role on the magnitude and duration of post exercise EE (Bahr & Sejersted, 1991; Gore & Withers, 1990; Laforgia, Withers, & Gore, 2006; Sedlock, Fissinger, & Melby, 1989). Low exercise intensities and/or low exercise duration has been typically related to small and short lasting EPOC, whereas more prolonged and abundant EPOC have been observed after strenuous exercise (Børsheim & Bahr, 2003). Furthermore, exercise surpassing 50-60% $\text{VO}_{2\text{max}}$ has resulted in an elevated EPOC (Børsheim & Bahr, 2003; Laforgia et al., 2006), which can last up to 14 hours (Knab et al., 2011; Ohkawara, Tanaka, Ishikawa-takata, & Tabata, 2008). Studies that have compared HIIE and SSE have shown that both protocols led to elevated EPOC from 1, 8, 21 and even 24h post exercise (Cunha, Midgley, Mcnaughton, & Farinatti, 2016; Greer

et al., 2015; Hazell et al., 2012; Laforgia, Withers, Shipp, & Gore, 1997; Skelly et al., 2014). Yet, there is still scepticism (Hazell et al., 2012; Matsuo et al., 2012; Williams et al., 2013). A study conducted by Greer et al. (2015) compared isocaloric bouts of HIIE, SSE and resistance training to establish which of these protocols would lead to a greater EPOC. They began with a 45-minute circuit training session while using indirect calorimetry to measure EE. The SSE and HIIE followed the circuit training and continued until the exercise protocols attained the EE measured from the resistance training. The exercise EE was determined by means of the Weir equation. They claim that resistance training and HIIE had a greater EPOC than SSE up to 21hr post exercise. The current study analyzed 90 minutes of EPOC following HIIE and SSE. The net interval exercise (513 kJ) expended 28% more energy than the net SSE (368 kJ) during post exercise measurements (Figure 5) despite utilizing less energy during exercise. These findings differ from Malatesta et al. (2009) as they found no significant difference in post exercise EE during the 3 hours of EPOC. They claim to have matched EE between SSE and HIIE, yet they did not consider the anaerobic EE generated from their 60s repeated sprints. Our methods differed from Malatesta et al. (2009) in regards to exercise intensity and sprint durations. During HIIE, our subjects were exposed to 150% W_{max} as opposed to their submaximal intensity of 80% W_{max} . This can explain why Malatesta et al. (2009) did not find a difference between post exercise EE for SSE compared to the HIIE protocol, which supports the notion that higher intensity exercise generates a greater EPOC.

Figure 6A illustrates 90 minutes of EPOC subdivided into 5 minute intervals. The VO_2 values for HIIE were found to be significantly different from SSE throughout the 5 minute intervals of EPOC. According to the non-protein respiratory quotient (Appendix 10.1), a higher VO_2 value results in a higher energy expenditure (Appendix 10.1). Although both protocols had

a higher VO_2 than baseline measures for the entire 90 minutes, HIIE VO_2 was significantly greater than SSE for the first 45 minutes of EPOC. This VO_2 increment can be explained as a result of ATP and PCr resynthesis, lactate to glycogen resynthesis, the oxidation of lactate and contribution to energy metabolism, the restoration of oxygen into myoglobin and hemoglobin, thermogenic effects of elevated core temperatures, thermogenic effects of hormones (catecholamines), effects of elevated heart rate, ventilation and other physiologic functions as well as the energy needs that are associated with the body's return to homeostasis (Bangsbo et al., 1990; Gaesser & Brooks, 1984; McArdle et al., 2010).

Heightened sympathetic activity occurs during HIIE, which in turn impairs the parasympathetic system due to the consistent elevation of adrenergic factors and local metabolites during recovery (e.g., epinephrine, norepinephrine, and venous blood lactate) (Buchheit & Laursen, 2007). The anaerobic contributions of HIIE has been found to be the main factor that leads to the delayed reactivation of the parasympathetic system (Buchheit & Laursen, 2007), which has been shown to affect the sinus node during the first hour of post exercise measurements when compared to SSE (Mourot, Bouhaddi, & Tordi, 2004). This reasoning can explain why HIIE was associated with a greater HR than SSE during 90 minutes of EPOC (Figure 6B). HIIE also had a significantly greater VE during the first 45 minutes of EPOC when compared to SSE (Figure 6B).

The VE correlates to the subjects' VO_2 , which is notably known as ventilatory equivalent (VE/VO_2). Farrell & Ivy (1987) proposed that increases in VE/VO_2 during incremental test to exhaustion is not caused by BLC, but rather, closely correlated with the metabolic rate of active muscles (Farrell & Ivy, 1987). Our findings also indicate that HIIE had a significantly greater EE compared to SSE for the first 45 minutes of EPOC (Figure 7). These results support statements

from Bahr & Sejersted (1991), Gore & Withers (1990), Laforgia et al. (2006) and Sedlock et al. (1989), which postulate that exercise intensity plays a larger role than duration on the magnitude and length of EPOC.

Similar to our study, Tucker et al. (2016) found that sprint interval exercise (six 30s-wingates) led to a greater EPOC and fat oxidation than SSE and high intensity aerobic exercise (HIAE) during 3h of post exercise measurements. However, when they added exercise and EPOC EE, they found that SIE was significantly lower than SSE and HIAE. When we added the sum of exercise and EPOC EE, we found that HIIE (3033 ± 321 kJ) and SSE (3021 ± 399 kJ) did not differ from one another (Figure 8). Williams et al. (2013) found no difference in EE during EPOC for both exercise protocols, but when they added the exercise EE and EPOC EE they found that the EE for SSE was greater than that of HIIE. Their SSE protocol expended ~560 kilocalories above baseline (exercise + EPOC), whereas HIIE only surpassed baseline measures by ~85 kilocalories. Although Greer et al. (2015), Williams et al. (2013) and Tucker et al. (2016) findings are interesting, they did not match exercise EE since they used indirect calorimetry to measure exercise EE and omitted the anaerobic contributions.

A key determinant of indirect calorimetry is the determination of carbohydrates and fat oxidation ratio with the use of a table of non-protein respiratory quotient (Appendix 10.1). This table illustrates, for a given ratio, the percentage and quantity (mg) of sugars and fats utilized per liter of O₂. RER begins at 0.7 where 100% of its energy is derived from fat stores and progressively translates to 100% sugar usage when RER is equal or greater than 1.0. During EPOC, we found a significant difference in total fat (grams) utilization between HIIE (16.99 ± 3.14), SSE (12.59 ± 3.75) and Baseline (5.77 ± 2.95) (Figure 9). This fat utilization can be

rationalized by an increase in catecholamine (Boutcher, 2010), especially epinephrine, which has been shown to drive lipolysis and is responsible for the release of fat from subcutaneous and intramuscular fat store (Issekutz & Recent, 1978). This is in accordance with Zouhal et al. (2008) as they found HIIE leads to a significantly greater catecholamine (epinephrine/norepinephrine) response compared to SSE.

Studies have assessed HIIT over the course of 6 to 15 weeks and found that this type of exercise leads to reductions in fat mass (Gillen et al., 2013; Heydari, Boutcher, & Boutcher, 2013; Tjonna et al., 2009; Trapp et al., 2008). Other authors such as Greer et al. (2015), Martins et al. (2016) and Mitranun et al. (2014) had methodical approaches that matched the EE between both protocols. Over a 12-week period, improvements were found in body composition, aerobic fitness (Martins et al., 2016; Mitranun et al., 2014), body fat % in comparison to pre-training phases (Mitranun et al., 2014) but no significant differences in fat loss between both exercise protocols (Martins et al., 2016). Our results differ as we found that one session of interval exercise led to greater fat oxidation during 90 min of EPOC. Intermittent exercise was able to utilize more grams of fat in order to replenish the energy consumed during exercise (Figure 9). Differences in fat utilization between HIIE and SSE remained once we subtracted the basal metabolism from 90 minutes of EPOC (Figure 10). Similar to our findings, Greer et al. (2015) found that high-volume resistant exercise and high intensity intermittent aerobic exercise led to greater post exercise EE compared to SSE as subjects burned an extra 12kcal/h between the 12h and 21h of EPOC.

7. Conclusion

Although Greer et al. (2015), Martins et al. (2016) and Mitranun et al. (2014) attempted to have two iso-caloric exercise protocols, they quantified the EE with the use of indirect calorimetry, which only measures the aerobic attributable EE. Our methodological approach is unique as we attempted to match EE between HIIE and SSE with the use of the GE method to determine whether intensity or duration would lead to a greater EPOC.

In summary, our study found that HIIE was a more efficient way to burn energy and fat stores during 90 minutes of EPOC. Although HIIE used less energy during the exercise, it was able to exceed SSE EE during 90-minutes of EPOC in addition to being 44% more time efficient. These findings are interesting and should be further explored in a training program where HIIE and SSE EE are properly controlled in order to determine the long-term effects of this type of training. With lack of time being the underlying restraint for attaining ideal levels of physical activity and associated health benefits (Kimm et al., 2006; Stutts, 2002), HIIE is a promising method to promote fat loss for those who want to avoid spending excessive hours in the gym. Although our study consisted of young and healthy individuals, clinicians should be aware that our HIIE protocol was very challenging and required considerable amounts of motivation.

8. Limitations and Future Research

Three subjects fell one repetition short of their pre-established interval feat, which would naturally increase the mean HIIE EE. We also believe that our HIIE EE was inferior to our estimations and to the SSE protocol as a result of using a constant GE throughout the repetitions as opposed to a decreasing GE. Even though 12 subjects participate in this study, the addition of more participants may have led to more significant differences. In regards to future research, it would be interesting if fat and carbohydrate utilization could be measured during SSE and HIIE exercise. In our study, interval exercise was able to utilize less energy during exercise succeeded by a greater EPOC EE all while utilizing more fat. Once we added exercise and EPOC EE, no significant differences were noted between the exercise protocols. We understand that it would be quite difficult to quantify the macronutrients used during HIIE due to the fluctuating anaerobic system. Nonetheless, it would be interesting to see how the body manipulates the use of its macronutrients for energy, and perhaps no differences would occur in terms of fat usage from the start of exercise to the end of EPOC.

9. Annex

Annex I: The estimated SSE EE

The energy expenditure from steady state exercise performed at 60% VO_{2max} for 45 minutes can be calculated with gas analyzers. If the individual's VO_{2max} was 3.5 L/min, we multiplied it by 60% giving us roughly 2.1 L/min. The resistance from his VO_{2max} at that particular time was the resistance used to begin the 45-minute exercise. Over time his VO_2 eventually increased therefore, we decreased the resistance to keep his VO_2 at 3.5 L/min. In order to estimate the EE during the training sessions, the RER and VO_2 (L/min) values found at 60% of their max was used to determine the targeted value. For example, a VO_2 of 2.1 L/min with an RER of 0.83 leads to an EE of 1,913 kJ. This was found by multiplying 2.1 L/min by the value of Kjoules found at a RER of 0.83 (Appendix III), which is equivalent to 20.25 Kjoules/ LO_2 and then multiplied that by 45 minutes. $2.1 \text{ L/min} * 20.25 \text{ Kjoules}/LO_2 * 45 \text{ minutes} = 1,913 \text{ Kjoules}$.

Annex II: The estimated HIIE EE

The intensities were set at 150% and 40% of their wattage established from their VO_{2max} . If their max resistance from their VO_{2max} was 300W, they cycled at 450W for 30s followed by an active recovery set at 120 watts ($300 * 1.5 = 450W$ & $300 * 0.4 = 120W$). Cycling at 450 watts for 30s leads to 13.5 KJ of mechanical work. Considering cycling at 450W is equivalent to 450J/s, we multiplied that by 30s, which gave us 13,500 Joules or 13.5KJ. Total metabolic energy (kJ) equals mechanical work divided by GE. Dividing 13.5 by 18% (GE) results in total metabolic energy (kJ). This means that during a single 30s sprint at 450watts, a total of 75KJ is used. The recovery rate (40% of their VO_{2max}) averages out to a VO_2 of 3.5 L/min with an RQ of 1.00 (Appendix III) for 60 seconds; this will account for 73.96 KJ ($3.5L/min * 21.13$ KJ {RQ at 1.00} = 73.96KJ). Every HIIT repetition will account for 61.5 KJ + 73.96 KJ giving us 135.46 KJ. Understanding that our goal is to reach 1913 KJ to match SSE, it will take ~14 repetitions to do so ($1913KJ / 135.46KJ = 14.12$ reps).

10. Appendices

Appendices I: Consent form

Comparing the physiological effects of high intensity interval training versus steady state exercise training with similar energy expenditures

Principal Investigators:

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INFORMED CONSENT

You are being asked to consider participating in a research study. A research study is a way of gathering information on a treatment, procedure or program or to answer a question about something that is not well understood. This form explains the purpose of this research study, provides information about the study procedures, possible risks and benefits, and the rights of participants.

Please read this form carefully and ask any questions you may have. The researcher will explain this form and all information concerning the study to you verbally. Please ask the researcher to clarify anything you do not understand or would like to know more about. Make sure all your

questions are answered to your satisfaction before deciding whether to participate in this research study.

Participating in this study is your choice (voluntary). You have the right to choose not to participate, and you have the right to withdraw from the study and stop your participation at any time. If you decide to stop participating, your data will be removed and there will be no consequences to you or the services you receive.

INTRODUCTION

Many adults who do not live an active lifestyle state that lack of time is the limiting factor to a healthier lifestyle. Steady state exercise (SS) is a common way to lose weight and improve your health, but over the last couple of years high intensity interval training (HIIT) has become very popular due to its short duration of exercise.

WHY IS THIS STUDY BEING DONE?

The purpose of this research is to determine if 45 minutes of steady state exercise or ~20 minutes of HIIT will lead to higher energy expenditure.

WHAT WILL HAPPEN DURING THIS STUDY?

You will first need to fill in a PAR-Q test prior to the testing in order to determine whether or not you are capable of performing the exercises. Your body mass index (BMI, mass in kg/height in m²) will be measured and if it surpasses 29 you will not be eligible to take part in this study. If you choose to accept, you will need to take part in various exercise protocols and data collection, which includes the collection of blood samples.

The exercise protocols & data collection

1st session (Total duration: 2 hours)

- Test 1: After an overnight fast you will visit the laboratory first thing in the morning between 8-10am. You will lie on your back for 30 minutes with a gas analyzer fixed to your mouth to analyze your resting metabolism.
- Test 2: Following the resting metabolism measurements I will measure your body fat distribution with the use of a hydrostatic tank and skinfold calliper.
- Test 3: After these first two test you'll have the opportunity to grab a bite to eat. You will then return to the lab (~2 hours later) to complete a VO_{2max} on a cycling ergometer.
- Test 4: Following the VO_{2max} you'll have 20 minutes to wander around the laboratory in order to recuperate. You will then get back on the bike to perform a 30s sprint at 150% of your maximal resistance.

Your oxygen consumption, respiratory ventilation and heart rate will all be measured during these tests. Micro blood samples will also be gathered in this study. The collection of blood will be done prior (t0) and immediately after (t1) the exercise protocols along with 3 (t2) and 5 minutes (t3) after the termination of the exercise. A blood lactate analyzer will measure the collected blood sample. Once measured, the blood will be disposed of in a in a biohazard disposal.

2nd & 3rd session: These sessions will be organized following your initial visit and set to fit your schedule. They will occur in a randomized order and interspersed by at least one day and will not require an overnight fast. You will have to take part in 45 minutes of SS at 60% of your

VO_{2max} and ~20 minutes of HIIT exercise protocol. You will cycle at 150% of your maximal resistance for 30s followed by 60s of active recovery set at 40% of your VO_{2max}. The HIIT session will have an estimated 10 to 15 repetitions. Your oxygen consumption will be measured for 90 minutes after the end of your exercise.

WHAT ARE THE RISKS OR HARMS OF PARTICIPATING IN THIS STUDY?

There are potential risks to this study considering you will be exercising for an extended period of time and at a relatively high intensity. Potential side effects include an increase in sweating, dehydration, dizziness or risk of exhaustion. If any of these complications occur and you start to feel uncomfortable, you have the right to end the exercise. Lab technicians with first aid and CPR training will be present during the exercises. After the exercise protocols are completed you will be obligated to stay in the laboratory for at least 30 minutes to make sure your heart rate decreases and that you aren't exhausted. You can also withdraw from this research at any given time. If you are dissatisfied after any of these tests you can contact Dr. Serresse or myself via e-mail or by telephone.

Potential Benefits

Your involvement in this study can possibly answer whether short lasting high intensity workouts burn more calories than long lasting steady state exercise. The results from this study can be quite relevant to the scientific community as they can be used in future studies. Your participation in this study will also come with physiological improvements, as it will lead to a healthier cardiovascular system.

HOW WILL MY INFORMATION BE KEPT CONFIDENTIAL?

The data collected, which will be kept secured on a computer, will help us conclude whether HIIT or SS is more effective for burning calories during post-exercise measurements. Only with your consent will we be able to use the collected findings for the production, oral presentation and publication of my thesis. Your identity will not be disclosed at any point and time.

INFORMATION ABOUT THE STUDY RESULTS

Once the data is collected and analyzed you will have the right to review the summary of the results. If you would like to receive the data you can contact me via e-mail or through my cell phone.

WHAT ARE THE RIGHTS OF PARTICIPANTS IN A RESEARCH STUDY?

If there is any problem or if you have any questions regarding this study you can contact Dr. Serresse at 705-675-1151 ext: 1085 or at oserresse@laurentian.ca. If you have any questions about the ethical conduct of this research feel free to contact Research Ethics Officer: Laurentian University Research Office. E-mail: ethics@laurentian.ca. Telephone: 705-675-1151 ext 3213.

DOCUMENTATION OF INFORMED CONSENT

You will be given a copy of this informed consent form after it has been signed and dated by you and the study staff.

Full Study Title:

**Comparing the physiological effects of high intensity interval training versus steady state
exercise training with similar energy expenditures**

Name of Participant: _____

Participant/Substitute decision-maker

By signing this form, I confirm that:

- This research study has been fully explained to me and all of my questions answered to my satisfaction
- I understand the requirements of participating in this research study
- I have been informed of the risks and benefits, if any, of participating in this research study
- I have been informed of the rights of research participants
- I have read each page of this form

Name of participant/
(print)

Signature

Date

Name of Person
administering

Signature

Date

Appendices II: Par-Q Test

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

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

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

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

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

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

**If
you
answered**

YES to one or more questions

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

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

NO to all questions

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

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

DELAY BECOMING MUCH MORE ACTIVE:

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

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

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

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

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

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

NAME _____

SIGNATURE _____

DATE _____

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

WITNESS _____

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



Appendices III: Respiratory quotient

Q.R. Non Protein	kJ per L O2 consumed	Kcal per L O2	% Sugar		mg (L/O2)	
			Sugar	Fat	Sugar	Fat
0.70	19.61	4.686	0.00	100.0	0	496
0.71	19.63	4.690	1.10	98.9	12	491
0.72	19.68	4.702	4.76	95.2	51	476
0.73	19.73	4.714	8.40	91.6	90	460
0.74	19.79	4.727	12.0	88.0	130	444
0.75	19.84	4.739	15.6	84.4	170	428
0.76	19.89	4.750	19.2	80.8	211	412
0.77	19.94	4.764	22.8	77.2	250	396
0.78	19.99	4.776	26.3	73.7	290	380
0.79	20.04	4.788	29.9	70.1	330	363
0.80	20.10	4.801	33.4	66.6	371	347
0.81	20.15	4.813	36.9	63.1	413	330
0.82	20.20	4.825	40.3	59.7	454	313
0.83	20.25	4.838	43.8	56.2	496	297
0.84	20.30	4.850	47.2	52.8	537	280
0.85	20.35	4.862	50.7	49.3	579	263
0.86	20.41	4.875	54.1	45.9	621	247
0.87	20.46	4.887	57.5	42.5	663	230
0.88	20.51	4.899	60.8	39.2	705	213
0.89	20.56	4.911	64.2	35.8	749	195
0.90	20.61	4.924	67.5	32.5	791	178
0.91	20.66	4.936	70.8	29.2	834	160
0.92	20.71	4.948	74.1	25.9	877	143
0.93	20.77	4.961	77.4	22.6	921	125
0.94	20.81	4.973	80.7	19.3	964	108
0.95	20.87	4.985	84.0	16.0	1008	90
0.96	20.92	4.998	87.2	12.8	1052	72
0.97	20.97	5.010	90.4	9.58	1097	54
0.98	21.02	5.022	93.6	6.37	1142	36
0.99	21.08	5.035	96.8	3.18	1186	18
1.00	21.13	5.047	100.0	0.00	1231	0

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