THE DETERMINATION OF TOTAL ENERGY EXPENDITURE DURING AND FOLLOWING REPEATED HIGH-INTENSITY INTERMITTENT SPRINT WORK

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ABSTRACT

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The literature addressing the glycolytic contribution to overall energy expenditure has primary been utilized during resistance training and cycling. There is a paucity of data examining the glycolytic contribution to overall energy expenditure (EE) in the form of high-intensityintermittent sprint work. Therefore, the purpose of this study was to examine the variation in oxidative and glycolytic contribution during two HIIT protocols using a 1:1 work-to-rest ratio and a 2:1 work-to-rest ratio. Seventeen physically active participants performed three exercise testing sessions. The first session involved an incremental maximal exertion treadmill test along with a sprint familiarization. Sessions two and three involved a 30:30 and 30:15 HIIT protocol performed in a counterbalanced order. The HIIT sessions involved four sets, of four sprints with three minutes of recovery between each set. During both HIIT sessions, oxygen consumption accumulation, carbon dioxide production, and respiratory exchange ratio were measured to calculate overall oxidative and EPOC contribution. Blood lactate concentration was measured to calculate overall glycolytic contribution. Total EE was determined by summating oxidative, glycolytic and EPOC measurements. Relative contribution of energy system involvement was analyzed using a 2 x 4 repeated measures ANOVA. Paired t-test determined significant differences in total EE between sessions. Comparing total session EE, independent t-test revealed no significant difference between HIIT sessions (p = .947). There was a significant difference between the two sessions with respect to overall kCal expenditure from the oxidative system (p = .037), glycolytic system (p < 0.01), and EPOC (p <0.01). Independent t-test revealed a significant difference in glycolytic (p < 0.01), oxidative (p < 0.01), and EPOC contribution (p < 0.01) between both HIIT sessions. Repeated-measures ANOVA revealed a significant difference between sets and the two

HIIT sessions in regards to the oxidative ($p = .047$) and glycolytic ($p = .022$) contribution, and a significant difference between relative contribution from oxidative ($p < 0.01$) and glycolytic system ($p < 0.01$) between sets. In conclusion, utilizing pulmonary gas exchange to represent the oxidative and EPOC in conjunction with blood lactate to represent the overall glycolytic contribution depicts an acceptable EE estimation during a bout of HIIT.

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CHAPTER 1: INTRODUCTION

High-Intensity Interval Training

High-intensity interval training (HIIT) is a well-established training modality used to improve performance in aerobic (e.g., cycling and long-distance running) and anaerobic (e.g., sprinting and team-sports) competitions alike (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Astorino, Allen, Roberson, & Jurancich, 2012; Bayati, Farzard, Gharakhanlon, & Agha-Alinejad, 2011). While there is no universal consensus on HIIT, it is generally described as brief, high-intensity bouts of effort, interspersed with planned periods of rest (Buchheit & Laursen, 2013). Thus, there are virtually endless permutations for structuring a program involving this form of training (e.g., work-to-rest ratios, intensity, duration, and modality), which is an attractive feature leading to its ubiquitous nature in sport and exercise training. Traditionally, HIIT has been shown to improve athletic performance within a strength and conditioning program (Dupont, Akakpo, & Berthoin, 2004; Farzad, Gharakhanlou, Agha-Alinejad, Curby, Bayati, Bahraminejad, & Mäestu, 2011; Sandbakk, Sandbakk, Ettema & Welde, 2013; Clark, 2010; Lindsay, Hawley, Myburgh, Schomer, Noakes, & Dennis, 1996). Within those programs, HIIT is commonly utilized to tax a specific metabolic system in order to stimulate desired physiological and biochemical responses such as, increase in VO_{2max}, peak power output, and post-exercise energy expenditure (EE) (Townsend, Stout, Morton, Jajtner, Gonzalez, Wells, Mangine, McCormack, Emerson, Robinson IV, Hoffman, Fragala, & Cosio-Lima, 2013; Astorino, Allen, Roberson, & Jurancich, 2012; Farzad, Gharakhanlou, Agha-Alinejad, Curby, Bayati, Bahraminejad, & Mäestu, 2011). However, what is becoming increasingly more popular is HIIT adoption within the general population in programs such as Crossfit™, UFC Fit™, P90X™, bootcamp classes and circuit training (Gibala,, Little,

MacDonald, & Hawley, 2012; Smith, Sommer, Starkoff, & Devor, 2013). It would appear that the growing attractiveness in this population is grounded in the ability to elicit optimal EE due to performing more work in less time compared to lower-intensity, steady-state work and increased EE post-exercise. (Gibala & McGee, 2008; Laforgia, Withers, Shipp, & Gore, 1997; Christensen, Hedman, & Saltin, 1960; Bahr & Sejersted, 1991; Townsend, Stout, Morton, Jajtner, Gonzalez, Wells, Mangine, McCormack, Emerson, Robinson IV, Hoffman, Fragala, & Cosio-Lima, 2013). With the increased demand for exercise programs incorporating HIIT, it seems prudent to accurately estimate the energy cost of HIIT. The determination of total EE during exercise has been widely researched in the field of exercise physiology (Laforgia, Withers, Shipp, & Gore, 1997; Scott, 2005a; Scott, 2002; Scott, Croteau, & Ravlo, 2009; Drenowatz, Eisemann, Carlson, Pfeiffer, & Pivarnik, 2011; Magosso, da Silva Junior, Neto, Neto, Baldissera, 2013; Scott & Fountaine, 2013). Traditionally, researchers have utilized indirect calorimetry to estimate metabolic heat production during exercise or at rest by measuring oxygen (O_2) and carbon dioxide (CO_2) gas exchange and, subsequently, utilizing the respiratory exchange ratio (RER) to determine macronutrient contribution to overall energy expenditure (EE) (Scott, 2005a). However, this method of estimating EE is not without controversy, especially when the mode of exercise presumably has a significant contribution from the glycolytic system (Scott, 2005a; Scott, 2006).

Estimating EE by means of an indirect calorimeter tends to underestimate the energy cost when the modality of exercise has considerable contribution from the glycolytic system (Scott, 2002; Scott, 2005a; Scott, 2006; Scott, Croteau, & Ravlo, 2009). The glycolytic system can produce ATP via substrate-level phosphorylation when oxygen is insufficient to sustain phosphorylation via oxidative pathways. Therein lies an inherent flaw when using only indirect

calorimetry to estimate EE, as this method tends to reflect only turnover from the oxidative system to estimate the total energy cost from a bout of exercise. Indeed, Scott (2005) has shown that by measuring only oxygen uptake to estimate EE during upright treadmill intermittent sprints (three 15-sec sprints at ~177% VO_{2max}), total EE was underestimated by 108 kJ (~26 kCals). Likewise, there exists another issue when using only pulmonary gas exchange to estimate EE during high-intensity work.

At the onset of a high-intensity bout of exercise, a phase occurs in which the O_2 demand is not met by the O_2 uptake. This phenomenon has been linked to the activation of the glycolytic system and is termed the oxygen deficit (Scott, 1999). When intensity exceeds oxygen uptake, substrate-level phosphorylation provides substantial contribution to the overall EE (Scott, 2002). Moreover, since O_2 deficit is considered an anaerobic contribution to EE, indirect calorimetry may not be sufficient in accurately estimating the energy cost. It was originally thought that O_2 deficit could be estimated during the recovery phase of an exercise bout known as excess post-exercise oxygen consumption (EPOC) (Hill, Long, & Lupton, 1924). To estimate O_2 deficit during EPOC the oxidative and glycolytic contribution would both be represented by complete glucose oxidation (21.1 kJ) (Scott, 2005). This representation would ignore the anaerobic contribution during EPOC because the restoration of ATP is fueled by oxidative phosphorylation. Along with glucose, oxidative phosphorylation also utilizes lipids to resynthesize ATP. If EPOC is solely represented as complete glucose oxidation, it would overestimate half of the ATP-turnover concomitant with ATP, PCr and the glycolytic system because these metabolic functions are re-synthesized by way of oxidative phosphorylation during recovery (Baechle & Earle, 2008). Thus, it appears that the use of indirect calorimetry to

estimate EE during high(er) intensity bouts of exercise will lead to incorrect measurement totals due to its inability to represent glycolytic contribution.

However, Scott (2013) has shown that glycolytic contribution can be estimated by measuring the change in blood lactate concentration after exercise (Margaria, Aghemo, & Sassi, 1971; Margaria, Cerretelli, di Prampero, Massari, & Torelli, 1963). This technique of estimating glycolytic contribution to EE has been used during resistance training due to its natural intermittent, high-intensity style of exercise. Despite its novel use during weight training, repeated upright sprint training differs in its physiological, biochemical and musculature demands. Surprisingly, though, there appears to be a paucity of data addressing the glycolytic contribution of EE to a session of HIIT using repeated upright sprints. This is problematic as running is well-established as a common form of exercise and training being employed by novices to elite athletes. Proper intensity and duration is crucial to prescribe an effective HIIT session into an exercise or training program. Therefore, it is important that exercise and sport conditioning professionals are able to estimate the metabolic systems stressed and the energy cost from a session of HIIT. Therefore, the purpose of this study was to examine the variation in oxidative and glycolytic contribution during two HIIT protocols using a 1:1 work-to-rest ratio and a 2:1 work-to-rest ratio. It is hypothesized that the 2:1 work-to-rest ratio protocol will elicit a larger glycolytic contribution to overall EE compared to the 1:1 work-to-rest ratio protocol.

CHAPTER 2: REVIEW OF LITERATURE

High-Intensity Interval Training

High-intensity interval training (HIIT) has proven to be a popular training method to enrich aerobic and anaerobic performance (Lindsay, Hawley, Myburgh, Schomer, Noakes, & Dennis, 1996; Dupont, Akakpo, & Berthoin, 2004; Clark, 2010; Enoksen, Shalfawi, & Tønnessen, 2011; Astorino, Allen, Roberson, & Jurancich, 2012). While HIIT has no universally accepted protocol it generally consists of repeated, short (< 45 sec) to long (2-4 min) bouts of high-intensity exercise interspersed with planned periods of rest. These series of work and rest (i.e., work-to-rest ratio) are then repeated for a specified duration (Buchheit, & Laursen, 2013). Specifically, HIIT has been shown to improve maximal oxygen consumption (VO_{2max}), increase time spent at lactate threshold during exercise, increase peak power output, decrease body fat percentage while maintaining lean body mass, and increase excess post-exercise oxygen consumption (EPOC) (Bayati, Farzard, Gharakhanlon, & Agha-Alinejad, 2011; Enoksen, Shalfawi, & Tønnessen, 2011; Shing, Webb, Driller, Williams, & Fell, 2013; Laforgia, Withers, Shipp, & Gore, 1997).

It has been established that different HIIT programs manifest diverse training adaptations stemming from the resultant physiological and biochemical responses (Laurent, Vervaecke, Kutz, & Green, 2014). Adaptations from HIIT include the ability to maintain VO_{2max} throughout a competitive season, a prominent increase on energy expenditure (EE) during EPOC, improved running economy, increase mitochondria capacity, improved lactate threshold and velocity at lactate threshold (Townsend, Stout, Morton, Jajtner, Gonzalez, Wells, Mangine, McCormack, Emerson, Robinson IV, Hoffman, Fragala, & Cosio-Lima, 2013; Astorino, Allen, Roberson, & Jurancich, 2012; Gibala, Little, MacDonald, & Hawley, 2012; Enoksen, Shalfawi, & Tønnessen,

2011). While HIIT is a common training method utilized in sport conditioning routines, various 'mainstream' exercise programs are beginning to embrace the HIIT methodology. Exercise programs such as Crossfit™ and P90X™ have become an effective and prevalent form of HIIT within the general population due the ability to perform more work in less time and increase energy expenditure post-exercise or the proposed "metabolic afterburn" effect (Laforgia, Withers, Shipp, & Gore, 1997; Bahr & Sejersted, 1991; Christensen, Hedman, & Saltin, 1960). The "metabolic afterburn" effect is a term that describes the ability to burn additional calories after the workout has concluded. The "afterburn" effect has been shown to increase EE for up to 24 hours post-exercise (Bahr & Sejersted, 1991). Moreover, HIIT has presented the capability to perform more work in less time and still achieve optimal EE when compared to low(er) intensity, continuous work (Laforgia, Withers, Shipp, & Gore, 1997;). The ability to perform more work and achieve optimal EE is dependent on the work-to-rest ratios that are implemented with a HIIT session.

Work-to-rest ratios during HIIT presents diverse variations (work: rest) (Little, & Williams, 2007; Gosselin, Kozlowski, DeVinney-Boymel, & Hambridge, 2012; Baechle, & Earle, 2008). Different work-to-rest ratios are commonly implemented within a program designed to tax a specific metabolic system in relation to the intensity and duration of the specific interval. Beyond simply targeting a desired work-to-rest ratio, individuals working with athletes may incorporate a needs analysis of a specific sport to determine appropriate ratios to administer. Athletes that compete in aerobic and anaerobic dominant sports (e.g., triathletes, cross-country skiers or football, wrestling, soccer) can benefit from HIIT when the effort is from 15 seconds to 10 minutes in duration (Sandbakk, Sandbakk, Ettema & Welde, 2013; Lindsay, Hawley, Myburgh, Schomer, Noakes, & Dennis, 1996; Astorino, Allen, Roberson, & Jurancich,

2012; Clark, 2010; Ziemann, Grzywacz, Luszczyk, Laskowski, Olek, & Gibson, 2011; Dupont, Akakpo, & Berthoin, 2004; Helgerud, Høydal, Wang, Karlsen, Berg, Bjerkaas, Simonsen, Helgesen, Hjorth, Bach, & Hoff, 2006). Sandbakk, Sandbakk, Ettema and Welde (2013) implemented an eight week intervention on highly trained cross-county skiers and compared two different high-intensity interval protocols. One group performed long duration high-intensity intervals (five-10 minutes) and the other group performed shorter duration high-intensity intervals (two-four minutes). Two weekly sessions of the high-intensity interval protocol were implemented within the participant's traditional training program. Pre- and post-test measures consisted of a 12-km roller ski, a 7-km hill run, and a VO_{2max} treadmill test to assess oxygen uptake at ventilatory threshold. After the eight week intervention, results showed both training protocols increased VO_{2max} by 3.7 ± 1.6% (long duration intervals) and 3.5 ± 3.2% (short duration intervals) from pre-test to post-test measurements. However, the longer duration high-intensity intervals were more effective at improving overall endurance by showing a significant improvement in the 12-km roller ski test (M = -139.6 seconds vs. M = -17.1 seconds) and in the 7-km hill run (M = -94.0 vs. M = -33.9 seconds) (Sandbakk, Sandbakk, Ettem, & Welde, 2013). Generally, when a training protocol consists of longer duration intervals, intensity will be lower. Consequently, an appropriate work-to-rest ratio may be a 1:1 or 2:1 when the duration is longer (Buchheit & Laursen, 2013). For endurance athletes, longer duration intervals produce metabolic stress on the oxidative system as well as the glycolytic system, which are the primary metabolic systems that fuel such events (Gastin, 2001; Joyner & Coyle, 2008). This, in turn, allows for training at or near lactate threshold, which can ultimately improve the maximal lactate steady state point and allow athletes to sustain a given power output for a longer duration (Pringle & Jones, 2002). To display the significance of training at or near lactate threshold, Enoksen,

Shalfawi, and Tønnessen (2011) compared a high-intensity low-volume (HILV) training program versus a high-volume low-intensity (HVLI) training program in middle-distance runners. This study aimed to examine the effects of two different training protocols on VO_{2max}, velocity at maximal oxygen consumption (vVO_{2max}), running economy, and velocity at lactate threshold. The intervention was conducted for a 10 week period. Pre- and post-test measures involved a treadmill test to measure lactate threshold and velocity at lactate threshold, VO_{2max} treadmill test, and a treadmill performance test. The results indicated that the HILV groups significantly improved their velocity at lactate threshold ($km \cdot h^{-1}$) from 14.6 ± 1.0 $km \cdot h^{-1}$ to 15.2 ± 0.8 $km \cdot h^{-1}$ ($p \leq 0.05$). The HVLI training group did not have a significant change in velocity at lactate threshold ($km \cdot h^{-1}$), 15.3 ± 0.8 $km \cdot h^{-1}$ to 15.7 ± 0.7 $km \cdot h^{-1}$ ($p \geq 0.05$). The HILV group trained near their lactate threshold during the training intervention. This indicates that training at lactate threshold is advantageous for athletes because it can increase vVO_{2max}. Increased vVO_{2max} acclimates an athlete to sustain an increase velocity for an extended duration without lactate production surpassing lactate clearance (Pringle & Jones, 2002). Additionally, short(er) duration work-to-rest (e.g., 30:30 seconds, 30:60 seconds, and 90:180 seconds) ratios have also shown to improve overall VO_{2max} which indicates an improvement in aerobic capacity, electing this method of training a respectable alternative to traditional endurance exercise (Astorino, Allen, Roberson, & Jurancich, 2012; Clark, 2010; Ziemann, Grzywacz, Luszczyk, Laskowski, Olek, & Gibson, 2011). Athletes that rely on high power output and the ability to recovery from these bouts may benefit from short(er) duration high-intensity intervals. Farzad, Gharakhanlou, Agha-Alinejad, Curby, Bayati, Bahraminejad, and Mäestu (2011) examined the effects of a short duration, high-intensity interval training protocol on elite wrestlers. The protocol consisted of 6, 35m sprints with 10 seconds of recovery between each sprint (~1:2 ratio). The interval sprint

protocol was implemented in addition to the traditional training program. The interval protocol was performed two times a week, for a four week duration. The control group performed the traditional training program without any additional training. Pre- and post-test measures included a VO_{2max} treadmill test to assess aerobic capacity, vVO_{2max}, maximal ventilation, and peak oxygen pulse. Four 30-second Wingate test assessed peak and mean power output and a treadmill time to exhaustion test at vVO_{2max} was also assessed. The results from the post-test measurements presented a significant improvement in VO_{2max} (+5.4%, $p = 0.01$), peak oxygen impulse (+7.7%, $p = 0.009$), and time to exhaustion at vVO_{2max} (+32.2%, $p = 0.002$) within the intervention group. The control group displayed no significant differences during post-test measures ($p > .05$). The protocol used by Farzad et al. (2011) elicited similar metabolic stress seen during a wrestling match and also presented an increase in physiological factors that can increase aerobic and anaerobic performance during a wrestling match (Farzad et al., 2011). What this tends to show is that, depending on the nature of the sport or training goal, various iterations of HIIT can produce optimal performance gains.

Metabolic System Involvement during HITT

HIIT incorporates three distinct processes that provide energy during exercise, which include the phosphagen system, glycolytic system, and oxidative system. Each system involves a sequence of adenosine triphosphate (ATP) hydrolysis and ATP re-synthesis called ATP turnover (Scott, 2005a). The primary function of the metabolic system is to repeatedly provide energy by way of ATP turnover. Given that there are virtually endless permutations of HIIT structure (e.g., work-to-rest ratios, intensity, etc.); each metabolic pathway has a unique involvement during different protocols. For the sake of brevity and specificity towards the specific research questions, this section will be associated with a HIIT protocol that incorporates a lower work to

rest ratio (e.g., 1:1, 30 seconds of work, paired with 30 seconds of recovery). This specific type of research design implementing 30 seconds of work serves to mimic the duration of the Wingate Anaerobic Test (WAnT). Beneke, Pollman, Bleif, Leithäuser, and Hütler (2002) measured the relative contribution from the anaerobic and aerobic systems during a 30-second WAnT. Results revealed that the anaerobic system contributed ~ 80% of the total energy expenditure during the WAnT. This design was aimed to develop a protocol that elicited a large glycolytic contribution from exercise, therefore 30-seconds of supramaximal (110% vVO_{2max}) intermittent sprinting was employed. Supramaximal sprinting has shown to have a significant anaerobic contribution to overall EE (Scott, 2005; Zagatto, Redkva, Loures, Filho, Franco, Kaminagakura, & Papoti, 2011). This form of exercise require high-energy output and is commonly short in duration. The initial metabolic system that re-synthesis ATP during high-intensity exercise is the phosphagen system, trailed by the glycolytic system.

The predominant source of energy during the first five to10 seconds of exertion is the phosphagen system. The primary phosphates include stored ATP and phosphocreatine (PCr) to release energy. Our muscles have a limited amount of stored ATP (~80 to 100 g) to be utilized during exercise, therefore ATP turnover does not rely heavily on stored ATP (McArdle, Katch, & Katch, 2007). When an electrical impulse is sent to muscles, ATP can quickly release energy (Kang, 2008). The degradation of ATP is known as hydrolysis and this reaction is demonstrated below (Kang, 2008):

$$ATP + H_2O \xrightarrow{ATPase} ADP + P_i + H^+ + Energy$$

The breakdown of ATP involves the enzyme ATPase and is catabolized in order to release energy, hydrogen (H^+), inorganic phosphate, and adenosine diphosphate (ADP) (Kang, 2008). Due to the limited amount of stored ATP, PCr must further participate in ATP re-synthesis.

There is a greater storage of PCr in the muscle compared to ATP (Brooks, Fahey, & Baldwin, 2005). The role of PCr is not to directly produce energy but to re-synthesize ATP and inhibit the depletion of ATP during exercise (Gabr, El-Sharkawy, Schär, Weiss, & Bottomley, 2011). PCr combines a phosphate molecule to ADP using the creatine kinase enzyme, to re-synthesize a molecule of ATP and further the muscle contraction, this process is termed the creatine kinase reaction (presented below) (Baechle, & Earle, 2008).

$$ADP + PCr \xrightarrow{\textit{Creatine kinase}} ATP + Creatine$$

ATP and PCr depletion occurs rapidly during intense, brief exercise (five-10 seconds) and evidence has shown PCr to depress 1.3 seconds after the initial contraction of muscle (Gastin, 2001). Stored PCr concentrations are greater in type II muscle fibers at rest and the degradation of PCr is greater during maximal intensity exercise when compared to type I muscle fibers (Karatzaferi, de Haan, Ferguson, van Mechele, & Sargeant, 2001). This suggests that during HIIT, PCr depletion will progress rapidly due to the contribution of type II muscles fiber in the lower extremity and, thus, a decrease in power output and energy contribution from PCr seems likely. These high energy phosphates cannot solely sustain sufficient energy for high-intensity effort lasting longer than 10 seconds. Moreover, many forms of HIIT exceed a 10 second work duration; therefore, the energy demand will shift towards the glycolytic system to contribute additional energy for two-to-four minutes of intense activity (Baechle, & Earle, 2008, Howard, von Glutz, & Billeter, 1978; Bouchard, Taylor, & Simoneau, 1991; Bangsbo, Gollnick, Graham, Juel, Kiens, Mizuno, & Saltin 1990).

The glycolytic system plays a vital role in producing energy during high-intensity exercises. High-intensity exercise such as HIIT, stimulates a significant amount of type II muscle fibers in the lower extremity (Esbjörnsson-Liljedahl, Sundberg, Norman, & Jansson, 1999). Type

II muscle fibers exhibit a large amount of stored glycolytic enzymes, therefore the contribution of the glycolytic system is of importance (Baechle and Earle, 2008). The glycolytic system converts carbohydrates into either glycogen, which is stored in the muscle and the liver to glucose that is transported into the blood plasma of active muscles to be used as energy (Baechle and Earle, 2008). Approximately 80% of the body's glycogen is stored in the muscle and the other 20% is stored in the liver (Sherman, & Wimer, 1991). The glycolytic system is considered to be an anaerobic process of ATP turnover since it is generated without the presence of oxygen (O_2). Additionally, this pathway re-synthesizes ATP using a process called substrate-level phosphorylation. In the course of this process, ATP is re-synthesized via the direct breakdown of carbohydrates into glycogen and glucose to phosphorylate ADP into ATP. If glycolysis begins with glycogen and undergoes glycogenolysis (the breakdown of glycogen), it produces three ATP, while glucose only produces two ATP (Ratamess, 2012). Glucose must be broken down to glucose-6-phosphate in order for glycolysis to proceed, this process requires one molecule of ATP (Ratamess, 2012). As glycolysis progresses and a deficiency of O_2 exists, the glycolytic pathway produces pyruvate. Following a series of reactions, pyruvate ultimately has two distinct end routes, the formation of lactate from pyruvate (anaerobic glycolysis) or pyruvate is converted to acetyl-CoA to be shuttled into the mitochondria (aerobic glycolysis). Through high-intensity exercise, substrate-level phosphorylation reduces the demand for pyruvate to shift towards aerobic metabolism (Scott, 2005a). Mitigating the shift towards aerobic metabolism can potentially ignite the symptoms of fatigue if high-intensity exercise continues without proper recovery. High-intensity exercise has shown to accumulate a significant quantity of lactate (Lacour, Bouvat, & Barthélémy, 1990; Scott, Croteau, & Ravlo, 2009). The accumulation of lactate has been associated with fatigue (Finsterer, 2012). However, lactate is not the metabolite

to cause fatigue during exercise, the conversion of pyruvate to lactate creates an accumulation of hydrogen ions (H^+) that inhibit muscle contraction (Fitts, 2008). The breakdown of ATP via hydrolysis also release H^+ into the blood (Kang, 2008). The accumulation of H^+ proceeds to decrease intracellular pH, which causes metabolic acidosis (Fitts, 2008). A decrease in pH can potentially inhibit calcium binding to the actin filaments preventing muscle contraction (Fitts, 2008). Calcium plays a vital role in the contraction of skeletal muscle. Calcium is released from the sarcoplasmic reticulum to bind to troponin in order for the myosin filament to interrelate with the actin filament, allowing the muscle to contract (Allen, Lamb, & Westerblad, 2008). If this process is interrupted by a decrease in pH, it creates a reduction in the calcium kinetics and reduces cross-bridge formation by the inhibition of calcium to bind to the contractile filaments (Allen, Lamb, & Westerblad, 2008; Parkhouse, & McKenzie, 1984). In order to regulate lactate accumulation, two primary methods of clearance involve the shuttling of lactate into type I muscle fibers to undergo oxidation to be used as fuel or lactate can be converted back to glucose within the liver, via the Cori Cycle and subsequent gluconeogenesis to reproduce a glucose molecule that can be used as a substrate (Finsterer, 2012; Brooks, 2007). Moreover, if the intensity of the exercise does not elicit lactate accumulation to a significant degree, pyruvate is not converted to lactate and undergoes a shift to meet the metabolic demands.

When the turnover rate of glycolytic-ATP re-synthesis matches the rate of aerobic metabolism it is called aerobic glycolysis (Scott, 2005a). Once pyruvate is formed, and the metabolic demand does not produce lactate, pyruvate is converted to acetyl-CoA to enter the mitochondria. Once acetyl-CoA enters the mitochondria, it proceeds to enter into the Krebs Cycle, also referred to as the citric acid cycle. The Krebs Cycle continues the oxidation of the substrate that began in glycolysis using guanine triphosphate (GTP) via substrate-level

phosphorylation (Baechle & Earle, 2008). The oxidation of the primary substrate only produces two ATP during aerobic glycolysis. The Krebs Cycle produces significantly more ATP (~40) when it undergoes oxidative phosphorylation in place of substrate-level phosphorylation (Baechle & Earle, 2008). The Krebs Cycle ultimately resynthesizes ATP at a slower rate when compared to anaerobic glycolysis, but this process can continue for an extended period as long as the intensity of the exercise is low(er) or fatigue does not manifest (Baechle, & Earle, 2008; Kang, 2008). If fatigue arises during HIIT, subsequently decreasing performance, the Krebs Cycle becomes a central supplier to re-synthesizing ATP. If the duration of the training session continues, the oxidation of substrates begins to shift towards aerobic metabolism.

When repetitions of HIIT increase and the demand for energy increases, the percent of fuel from the glycolytic system can potentially decrease (Buchheit, & Laursen, 2013). Moreover, if the involvement of ATP turnover from the glycolytic system begins to depress, the oxidative system initiates additional contribution to re-synthesize ATP. However, if the oxidative system begins to contribute significantly to the overall ATP re-synthesis, power output also begins to decline. Gastin (2001) has shown that aerobic contribution to HITT can range from 3% to 98% involvement depending on the frequency, intensity, modality and duration. More specifically, Spencer and Gastin (2000) estimated that at the 30 second mark during a high-intensity run (49.3 ± 0.2 seconds), peak aerobic involvement was 43% of total EE (Spencer, & Gastin, 2000). This finding illustrates the oxidative system as an essential provider to the overall energy demand for high-intensity exercise. Furthermore, the oxidative system utilizes fats and carbohydrates to re-synthesize ATP. During high-intensity aerobic exercise, the primary substrate used as fuel shifts from a mixture of carbohydrates and fats to almost exclusively carbohydrates (Coyle, 1995). During glucose oxidation, substrate-level phosphorylation is only capable of re-synthesizing a

minor amount of ATP; subsequently, pyruvate must shift towards aerobic metabolism to maintain ATP re-synthesis (Scott, 2005a). Pyruvate is further converted to acetyl-CoA to enter the mitochondria, where it enters into the Krebs Cycle. ATP is produced in the Krebs Cycle via substrate-level phosphorylation, albeit only an inconsequential amount. The Krebs Cycle removes hydrogen ions by using nicotinamide adenine dinucleotide (NAD) and flavin adenine dinucleotide (FAD). After removing the H^+ ions, NAD and FAD become $NADH^+$ and $FADH_2$. $FADH_2$ and $NADH^+$ then proceed into the electron transport chain (ETC), where the potential energy stored in the H^+ ions phosphorylates ADP to form ATP, this process is known as oxidative phosphorylation (Kang, 2008). Oxidative phosphorylation also plays a crucial role in re-synthesizing ATP during the recovery periods of HIIT. The phosphagen and glycolytic systems undergo oxidative phosphorylation to resynthesize ATP, PCr and glycogen levels. Moreover, the contribution of each metabolic system during HIIT plays a pivotal role when estimating EE during and after high-intensity exercise.

Energy Expenditure

As an individual utilizes energy to perform work, heat is produced (Kang, 2008). Heat is measured to estimate the total amount of energy expended during exercise. Originally, a direct calorimeter was utilized to estimate the amount of heat given off during exercise. This technique was originally employed by Zuntz and Hagemenn in 1898. Zuntz and Hagemenn developed an insulated, seal tight chamber, which contained water running through copper tubing (Wilmore, Costill, & Kenney, 2008). This chamber captured the heat produced by the body, the heat subsequently increases the temperature of the water traveling through the copper tubing (Scott, 2008; Wilmore, Costill, & Kenney, 2008). By measuring the water's change in temperature within the chamber, metabolic EE could be estimated (Scott, 2008; Wilmore, Costill, & Kenney,

2008). This measurement is consider the gold standard of estimating EE during at rest but not during exercise. Commonly, a treadmill is placed within the chamber and the treadmill radiates heat, thus effecting the temperature of the water (Wilmore, Costill, & Kenney, 2008). The heat from the treadmill, in additional to the individuals body heat will depict any inaccurate estimation of overall EE. Also, during exercise, EE can change rapidly, this technique cannot survey this rapid change (Wilmore, Costill, & Kenney, 2008). Lastly, not all heat is radiated from the body, some of the heat is stored within the human body, but still generating energy. Therefore direct calorimetry would not be a practical choice for estimating EE during exercise (Wilmore, Costill, & Kenney, 2008). Since direct calorimetry was not deemed a practical estimate of EE during exercise, a new technique was developed to estimate EE during exercise. An indirect calorimeter was developed to estimate heat production during exercise by measuring carbon dioxide production (VCO_2) and oxygen consumption (VO_2) (Scott, 2005). The estimation of heat production is shown in the following equation (Kang, 2008):

$$\text{Substrate} + O_2 \longrightarrow \text{Heat} + CO_2 + H_2O$$

The oxidation of substrates (e.g., carbohydrates and lipids) yields heat, CO_2, and water (H_2O). An indirect calorimeter relies on pulmonary gas exchange (e.g., RER and VO_2) to estimate the energy cost of exercise. The ratio of CO_2 production and O_2 consumption is the determination of the respiratory exchange ratio (RER). The RER is utilized to determine the percentage of energy produced from carbohydrates and fats during exercise. RER of ≥ 1.00 suggests that fuel is derived purely from carbohydrates and if RER is ≤ 0.70, the fuel source is derived purely from fats (Zuntz, 1901). It is important to determine the percentage of carbohydrates and fats oxidized due to the different metabolic heat production. The oxidation of glucose is converted to heat as 1 liter of $O_2 = 21.1$ kJ (~ 5 kcal) and fats are converted to heat as 1 liter of $O_2 = 19.6$ kJ (~ 4.5

kcals) (Scott, 2005a; Scott, 2005b). There is a 1.5 kJ (~ .39 kcal) or 7% difference between the oxidation of carbohydrates compared to fats at 1 liter of O_2 consumed. Utilizing RER and VO_2 is a valid estimation of EE for low-to-moderate, steady-state, aerobic exercise due to the availability of O_2 and absence of lactate accumulation. During aerobic exercise, there is adequate supply of O_2 available to allow the oxidative system to re-synthesize ATP. When the oxidative system provides a consistent rate of ATP re-synthesis, an indirect calorimetry is a valid and useful technique to determine EE. Pulmonary gas exchange has been utilized to estimate EE during high-intensity intermittent exercise, but this method is not without debate due to the significant contribution from substrate-level phosphorylation throughout glycolysis. A significant involvement from the glycolytic system occurs during a phase called oxygen deficit (Figure 1.). During the initial contraction of muscle during high-intensity exercise, a period exists in which the O_2 consumed by the body does not meet the O_2 demand.

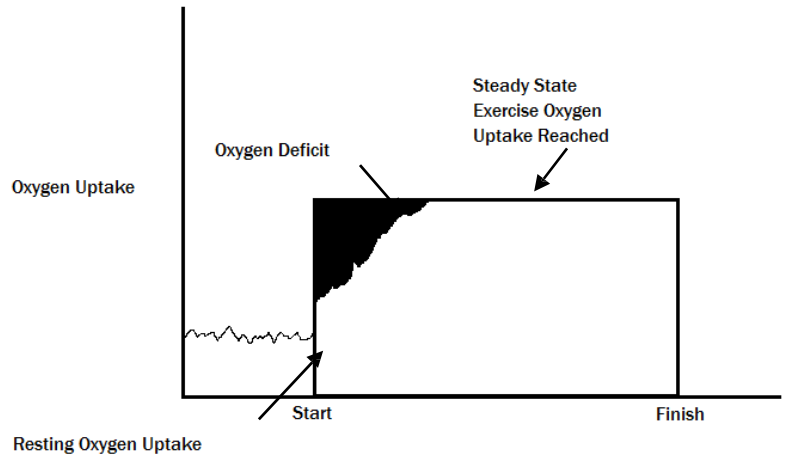


Figure 1.: Adapted from "*A Primer for the Exercise and Nutrition Sciences, Thermodynamics, Bioenergetics, Metabolism*" by Christopher B. Scott. Humana Press, Totowa, NJ, 2008.

The insufficient O_2 uptake by the muscles inhibits the oxidative system to generate ATP turnover (Scott, 1999). When demand for energy is elevated during the initial phases of high-intensity

exercise, substrate-level phosphorylation is a key component to provide energy. HIIT requires multiple transitions from resting or low-intensity activity to high-intensity activity. These transitions periods require a high energy demand during the initial phases of work (Figure 2.). This patterns develops several oxygen deficits during a bout of HITT, thus relying on substrate-level phosphorylation to re-synthesize ATP.

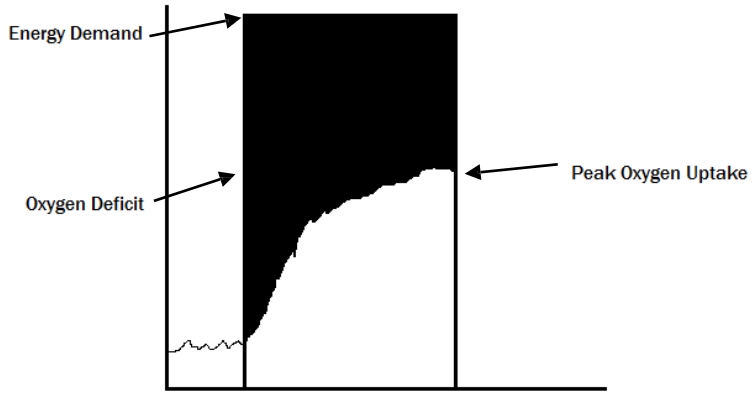


Figure 2. Adapted from "*A Primer for the Exercise and Nutrition Sciences, Thermodynamics, Bioenergetics, Metabolism*" by Christopher B. Scott. Humana Press, Totowa, NJ, 2008.

Due to the significant contribution from substrate-level phosphorylation during HIIT, an indirect calorimeter would not accurately estimate energy cost using pulmonary gas exchange alone due to the inadequacy of O_2 to supply energy via oxidative pathways. Therefore the glycolytic contribution must individually be accounted for during exercise.

Originally, the glycolytic contribution from the O_2 deficit was estimated during the recovery phase of an exercise known as excess post-exercise oxygen consumption (EPOC), formerly known as the oxygen debt (Figure 3.) (Hill, Long, & Lupton, 1924). EPOC is a phase that occurs post-exercise in which oxygen consumption (VO_2) is elevated above resting levels.

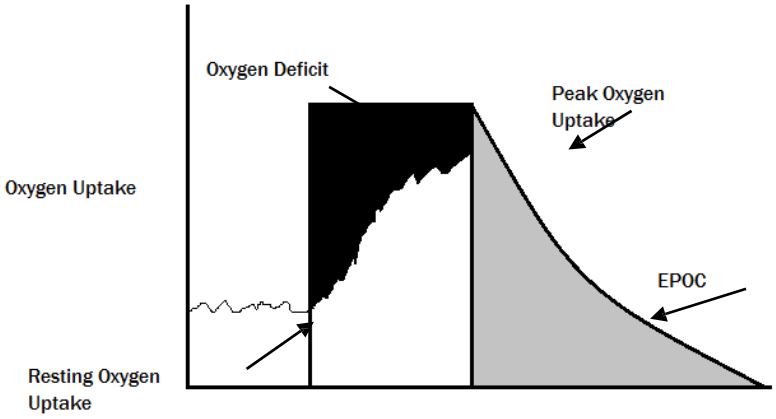


Figure 3: Adapted from "*A Primer for the Exercise and Nutrition Sciences, Thermodynamics, Bioenergetics, Metabolism*" by Christopher B. Scott. Humana Press, Totowa, NJ, 2008.

The O_2 consumption after exercise is elevated to restore the level of ATP, PCr, clear lactate from recovering muscles via the Cori Cycle, decrease core temperature, replenish O_2 content within the muscle and blood and regulate hormonal fluctuations (Bahr, & Sejersted, 1991; LaForgia, Withers, Shipp, & Gore, 1997; Townsend, Stout, Morton, Jajtner, Gonzalez, Wells, Mangine, McCormack, Emerson, Robinson IV, Hoffman, Fragala, & Cosio-Lima, 2013). These specific post-exercise processes have a significant metabolic cost after high-intensity training (Townsend, Stout, Morton, Jajtner, Gonzalez, Wells, Mangine, McCormack, Emerson, Robinson IV, Hoffman, Fragala, & Cosio-Lima, 2013). It was originally thought that in order to determine O_2 deficit during EPOC, the glycolytic and oxidative contribution to overall EE would be represented as complete glucose oxidation (21.1 kJ) (Scott, 2005). However, EPOC is a phase in which oxidative phosphorylation re-synthesize ATP, PCr and glycogen from lactate. Oxidative phosphorylation utilizes fats and carbohydrates to undergo these metabolic processes. If EPOC was represented as complete glucose oxidation, it would overlook the percentage of fats that contribute to this metabolic process. There are two techniques in which to accurately estimate the EE from EPOC. The first technique is to determine RER to accurately estimate the percentage of

carbohydrates and fats utilized during this phase. Secondly, EPOC can also be represented as complete fat oxidation if the glycolytic contribution from exercise is measured (Scott, 2005a). EPOC is represented as 19.6 kJ when the modality of exercise is of high-intensity, intermittent work and has a significant glycolytic contribution (Scott, 2005a). The contribution of EPOC to overall EE is important as ATP and PCr have shown to contribute up to 20% of EPOC during recovery, making it an integral component of EE and can be represented either during the O_2 deficit or during EPOC, but not both (Bangsbo, Gollnick, Graham, Juel, Kiens, Mizuno, & Saltin, 1990). Estimating EE from the phosphagen system (e.g., ATP and PCr) during EPOC has shown to be a valid estimation of phosphagen contribution during exercise (Gaesser & Brooks, 1984). Moreover, EPOC has been shown to increase as the intensity of the exercise increases, therefore EPOC is an integral component of overall EE (LaForgia, Withers, & Gore, 2005; Børsheim, & Bahr, 2003; Bahr, & Sejersted, 1991; Townsend, Stout, Morton, Jajtner, Gonzalez, Wells, Mangine, McCormack, Emerson, Robinson IV, Hoffman, Fragala, & Cosio-Lima, 2013). Laforgia et al. (1997) compared EPOC during a continuous bout of running at 70% VO_{2max} for 30 minutes to an interval run performed at 105% VO_{2max} that consisted of 20, 1-minute runs interspersed with 2-minute recovery periods. Total work for these two comparisons was equated to strictly determine which intensity and protocol (steady state compared to intervals) elicited the greatest effect on EPOC. Results from this study revealed the 70% VO_{2max} run resulted in an excess post-exercise energy expenditure (EPEE) of 133 ± 82 kJ (~32 kcals), 9 hours post exercise and the 105% VO_2 max interval run resulted in an EPEE of 268 ± 87 kJ (~64 kcals), 9 hours post exercise (LaForgia, Withers, Shipp, & Gore, 1997). Results from LaForgia et al. (1997) demonstrate that interval running conducted at a high-intensity has a comparatively greater effect on EPOC when compared to continuous, steady-state running. Townsend et al.

(2013) compared a 30-minute submaximal cycling protocol performed at 60% heart rate reserve to three repeated Wingate trials with four minute rest period between each bout. Results from this study revealed the Wingate protocol produced a significant effect on EPOC (p = .004) compared to the 30-minute, moderate cycling protocol. Post-exercise VO_2 displayed greater oxygen uptake during the Wingate trial when compared moderate cycling protocol by 5.7 ± .65 L O_2. Overall EE was also significantly higher (p= .04) in the Wingate trial (156.9 kJ) (~37.5 kcals) compared to the moderate cycling protocol (41.0 kJ) (~10 kcals) (Townsend, Stout, Morton, Jajtner, Gonzalez, Wells, Mangine, McCormack, Emerson, Robinson IV, Hoffman, Fragala, & Cosio-Lima, 2013). It seems, then, that HIIT can increase overall EE via post-exercise increases in energy consumption. Consequently, HIIT post-exercise EE can create a positive effect on overall EE because of the intensity at which HIIT is performed.

Estimating Energy Expenditure during HIIT

The glycolytic systems plays a significant role in overall EE during high-intensity exercise, labeling this metabolic pathway a vital component to produce energy (Scott, 2005a). Moreover, the glycolytic contribution to overall EE during exercise cannot be represented by the means of an indirect calorimetry. The O_2 demand can exceed the O_2 uptake during intense exercise, which results in the glycolytic system re-synthesizing ATP. An indirect calorimeter focuses on pulmonary gas exchange to estimate the energy cost from exercise, therefore this method cannot adequately represent glycolytic contribution due to substrate-level phosphorylation. In a review by Scott and Fountaine (2013) it was suggested that lactate must be obtained for an appropriate estimation of overall EE from an exercise session. The glycolytic contribution to overall EE is estimated by observing the change in blood lactate due to a bout of exercise. For every 1 millimole increase in blood lactate above resting levels is equivalent to the

consumption of 3.0 ml of O_2 per kilogram of body weight (Margaria, Aghemo, & Sassi, 1971). This technique has proven to be a reliable and valid estimation of the glycolytic contribution to overall EE during high-intensity intermittent modes of exercise (Vianna, Lima, Saavedra, & Reis, 2011; Scott, 2006; Scott, & Fountaine, 2013; Scott, Littlefield, Chason, Bunker, & Asselin, 2006; Lacour, Bouvat, & Barthélémy, 1990). This technique is an important contribution to estimating EE because oxygen uptake unaccompanied by lactate cannot account for the substrate-level phosphorylation that occurs during high-intense, intermittent exercise (Scott, 1997; Scott, 2006; Scott, 2005; Scott. 2002; Scott and Kemp, 2005). Indeed, Scott (1997) measured the EE for two different bouts of exercises while using three altered EE estimation techniques (O_2 consumption only, O_2 consumption and EPOC, and O_2 consumption, EPOC, and anaerobic contribution). The two different modalities of exercise incorporated a steady state walk and a bout of intermittent sprints. The steady state walk required 3.5 minutes of walking on a treadmill at 47% VO_{2max} and the intermittent sprints required the participants to perform three, 15 second sprints at 177% VO_{2max}. Total work was equated for both modalities. Measuring only exercising O_2 consumption, the steady state model expended 120 kJ (~29 kcals), while the sprints expended 16 kJ (~4 kcals). Measuring exercising O_2 consumption and EPOC, the steady state model expended 149 kJ (~36 kcals) and the sprints increased to 165 kJ (~39 kcals). Lastly, measuring exercise O_2 consumption, EPOC, and the anaerobic contribution to EE, the sprints produced 273 kJ (~65 kcals), while the steady state model remained the same. This study revealed the importance of factoring in the anaerobic contribution to high-intensity exercise because pulmonary gas exchange along will underestimate the total energy cost. Within Scott's (1997) study, if the anaerobic contribution was not accounted for, the sprints would have been underestimated by 108 kJ (~ 26 kcals). Scott (2006) also demonstrated an increase in overall EE

during a bout of resistance training when the addition of blood lactate measurements were added to oxidative measurements (VO_2, & EPOC) (Scott, 2006). It appears that the change in blood lactate due to exercise is a vital component to high-intensity, intermittent EE.

During a bout of high-intensity intermittent exercise, is it apparent that the glycolytic and oxidative systems play a crucial role in re-synthesizing ATP during and after exercise. The overall oxidative must be measured through pulmonary gas exchange during exercise and post-exercise (EPOC). The overall glycolytic contribution from exercise must be measured via the change in blood lactate concentration due to exercise. If lactate represents the overall glycolytic contribution to exercise, EPOC must be represented as complete fat oxidation (19.6 kJ) to eliminate the glycolytic contribution from this phase (Scott, 2005a). This technique has been utilized during high-intensity, intermittent cycling and resistance training. However, cycling and resistance training do not portray the same metabolic responses that occurring during upright, intermittent running. Given the popularity of running, there is a shortage of research investigating EE during HIIT in the form of running. This can be challenging for coaches and trainers to prescribe accurate and effective HIIT sessions if the metabolic demand and EE is absent. Hence, it is key that overall EE and metabolic demand be understood during HIIT.

Importance of Energy Expenditure during HIIT

Determining the energy cost and specific metabolic systems taxed during a bout of HITT can be beneficial to coaches and trainers when prescribing appropriate exercise programs, improving performance, regulating the athletes and clients lean body mass and fat mass, developing nutritional plans, and avoiding fatigue. Given the lack of research determining the energy cost of high-intensity programs, trained individuals may not be prescribing accurate exercise programs. If an exercise program is beyond the optimal load, the individual could be

prone to overtraining and musculoskeletal injuries (Smith, 2004). If the training load is depressed, the individual may plateau, decrease in performance or struggle with weight management (e.g., overall weight loss, decrease body-fat) due to an improper overload. It is also important for strength coaches in prescribing conditioning protocols for their athletes. For example; Rhea, Oliverson, Marshall, Peterson, Kenn, and Ayllón (2008), presented the importance of conditioning on power output for baseball players. This study examined the effects of two different conditioning protocols on power output. The first group performed continuous, moderate- to high-intense cardiovascular training and the other group performed sprints, intervals and speed endurance conditioning. These distinct protocols were implemented for an 18-week duration in addition to their traditional training program. Pre- and post-test power output was assessed using a counter-movement jump test. After the 18-week intervention there was a significant change in power output between the two groups ($p < .05$). The moderate- to high-intensity endurance group saw a decrease in power output of -39.50 ± 128.03 Watts and the sprint, interval and speed endurance group increased their power output by 210.63 ± 168.96 Watts (Rhea, Oliverson, Marshall, Peterson, Kenn, & Ayllón, 2008). These results display the importance of taxing a specific metabolic system while training anaerobic athletes. The moderate- to high-intensity group stressed the oxidative system during training, while the sprint, interval and speed endurance group stressed the glycolytic and phosphagen system. From this study, an inappropriate amount of oxidative work can decrease performance in anaerobic dominant athletes (i.e., power output). Although the moderate- to high-intensity endurance group were performing high-intensity training, it is plausible that this protocol taxed the oxidative system due to the lower intensity and continuous nature of the training. Significant aerobic training has been shown to be counterproductive in increasing peak power (Rhea, Oliverson,

Marshall, Peterson, Kenn, and Ayllón, 2008; Leveritt, Abernethy, Barry, & Logan, 2003). Increasing the stress on the oxidative system also has been shown to increase the percentage of type I muscle fibers (Wilson, Loenneke, Jo, Wilson, Zourdos, & Kim, 2012). If there is an increase in the percentage of type I muscle fibers utilized during exercise, this can also decrease power output because these muscle fibers are more associated with endurance and low-intensity energy output. Another important aspect of proper exercise prescription is recommending nutritional guidelines. For individuals that compete in endurance events (e.g., longer than two hours) or train multiple times a day (e.g., mixed martial artists, football, soccer, rugby, CrossFit) it is important that their glycogen levels are being replaced after training (Figueriedo & Cameron-Smith, 2013; Ørtenblad, Westerblad, & Nielsen, 2013; Aragon & Schoenfeld, 2013). If these athletes are taxing the glycolytic system to a substantial degree, there can be a significant decrease in glycogen stores depending on the intensity and the duration of the training. If glycogen stores are dramatically depleted during the first training session of the day and subsequent training was to occur, the athlete would need to replenish glycogen stores to perform optimally during the second training session. Glycogen stores are not only related to athlete performance but to the general population as well. HIIT is becoming a mainstream mode of exercise for the general population because an individual has the ability to perform equal or additional work within a short duration and benefit from the effect HIIT has on post-exercise EE (Gibala & McGee, 2008). It is important to note the degree to which HIIT taxes the glycolytic system. If the glycolytic system is contributing a significant amount of energy for a given exercise, an individual will have a decrease in their glycogen stores. If an individual is attempting to lose fat mass and is performing HIIT on a regularly basis or multiple times a week, replenishing glycogen stores is essential to continue training at a high-intensity. The

replacement of glycogen stores also relates to the supercompensation effect or the One Factor Theory described by Zatsiorski and Kraemer (2006). Zatsiorski and Kraemer (2006) state the effect of a training bout depletes specific substances within the body, these substance pertain to ATP, PCr, and glycogen or glucose depending on the intensity and the duration of the bout. If the glycolytic system contributes a significant sum to a training bout, muscle glycogen will have a certain degree of depletion. If glycogen levels are appropriately restored after exercise, there is the potential for glycogen levels to rise above the previous resting level (Zatsiorski & Kraemer, 2006). An increase in resting glycogen levels allows the muscles to utilize additional glycogen during training, therefore, prolonging fatigue for subsequent training bouts. Trainers, coaches and professionals in the field often overlook the metabolic contribution and overall EE during a bout of intense training. This current study, implementing a series of 1:1 work-to-rest ratios (i.e., 30 seconds of work paired with 30 seconds of recovery) and 2:1 work-to-rest ratios (i.e., 30 seconds of work paired with 15 seconds of recovery) will provide contextual data that will be beneficial to professional in the field implementing HIIT.

CHAPTER 3: METHODS

Experimental Approach to the Problem

This study examined the estimation of overall EE during two different HIIT protocols using a new, novel technique utilizing exercising VO_2, RER, blood lactate measurements and EPOC. Each participant was required to perform three testing sessions; an incremental maximal exertion VO_{2max} test along with a sprint familiarization and two HIIT sessions using different work-to-rest ratios that were performed in a counterbalanced order. EE was determined through the estimation of glycolytic, oxidative and EPOC contribution to the HIIT protocols. Moreover, this study examined the impact HIIT consisting of different recovery durations has on the relative contribution to overall EE. Employing this protocol of HIIT may provide a valid estimation of EE per session and could potentially exhibit the increase in EE associated with this form of training.

Participants

Seventeen healthy, physically active men (n = 8) and women (n = 9) were recruited to participate in this study. Out of the eight males that participated in the study, two were unable to complete the 30:15 HIIT session in its entirety, therefore they were excluded from that session. To be included in this study participants were currently performing HIIT or competing in an intermittent sport activity (e.g., soccer, rugby, basketball) at least two days per week and have been performing these sports or activities for the past three months. Exclusion criteria for this study included any musculoskeletal or orthopedic injury that may inhibit performance during the trials or if a participant was considered moderate risk or higher according to the ACSM guidelines (Thompson, Gordon, & Pescatello, 2010). Participants were asked to report to the Exercise Physiology Laboratory in a well-rested, hydrated state and be at least 4-h post-prandial as well as having abstained from caffeine for 4-h and alcohol for 24-h prior to testing. Each

participant was also asked eat a similar meal 4-h before each HIIT session. All testing procedures, risks, and benefits were explained to each participant before each session. Each participant was provided a written informed consent that was approved by the local Human Subject Research Board.

Experimental Procedures

Incremental maximal exertion treadmill test. The first lab session required participants to run an incremental maximal exertion treadmill test. Prior to testing, all participants were screened for inclusion using a medical history questionnaire, the PAR-Q and provided written informed consent before any testing was completed. Participant's height (cm) and body mass (kg) were measured using a stadiometer and beam scale (Detecto Scale Company, Webb City, Missouri, USA). Body fat measurement were estimated using a 3-site method (males: chest, abdomen, and thigh; females: triceps, iliac, and thigh) (Pollock, Schmidt, & Jackson, 1980) using skinfold calipers (Lange, Cambridge, Maryland, USA). All anthropometric measures for a participant were performed by the same technician.

Participants were allowed to perform a self-selected, five-10 minute warm up before the maximal exertion treadmill test. Participants then performed a maximal exertion treadmill test (Noakes, 1990) on a motorized treadmill (Truefitness, O'Fallon, MO). Participants were connected via a mouthpiece to a calibrated metabolic cart (ParvoMedics TrueOne 2400, Sandy, UT) that recorded metabolic measures throughout the test. The metabolic cart was calibrated before each participant performed a maximal exertion treadmill test. Participants began running on the treadmill at a speed of 6.2 mph with a 0% incline, and every minute the speed was manually increased by 0.6 mph until volitional fatigue. After completion of the test, the participant were monitored during a low-intensity cool down. The final speed reached during the last full stage of

the test determined the velocity eliciting maximal oxygen uptake (vVO_2). The vVO_2 was used to control the speed at which the participants ran at during session two (30:30) and three (30:15). Specifically, 110% vVO_{2max} was the target speed during the HIIT sessions. 20 minutes following the incremental maximal exertion treadmill test, the participant was required to run a sprint familiarization trial. The treadmill was set to 110% of the participant's vVO_{2max}. The participant then performed one set of four sprints, at a 30:15 (work-to-rest) ratio.

Session 2: HIIT 30:30. This session took place two to seven days after the maximal exertion treadmill test. Upon arrival to the laboratory, participants were fit to the metabolic cart to record resting EE. Participants were seated in a chair for five-10 minutes while metabolic measurements (e.g., VO_2 and RER) were recorded. Immediately following resting EE measurement, a resting blood lactate measurement was obtained and recorded. Following the determination of resting measures, participants performed a standardized warm-up in agreement with the procedures developed by Vetter (2007). The protocol consisted of a four minute walk at 3.7 mph, a two minute run at 7.5 mph, and three rounds of a dynamic warm up (see Table 1). Following the warm up, participants performed a series of high-intensity interval sprints at 110% vVO_{2max}, established from session one. The intervals consisted of a 30 second sprint paired with 30 seconds of passive recovery (straddling the treadmill belt). The participants were asked to complete four sets of four 30 second sprints that were interspersed with 30 seconds of recovery (i.e., 30:30 protocol). Prior to each sprint, participants were provided a five second countdown to indicate the beginning of a new sprint. Between each set of four 30 second sprints, participants were afforded three minutes of passive recovery (sitting in a chair). During the three minute passive recovery period, a blood lactate sample was taken at the two minute mark of the recovery phase. Scott, Croteau, and Ravlo (2009) reported that blood lactate concentration generally

peaked two minutes after intense exercise. This process was repeated for each of the four sets of sprints. At the end of the session (completion of the four sets of sprints), participants were required to sit in a chair with metabolic measures still recording to determine EPOC, these measurements continued for a seven minute time period.

Table 1.
Standardized warm-up protocol

Dynamic warm-up exercise	Repetitions	Cadence (reps per min)
Toe Raises	10	30
High knee lift	20	30
Buttock kick	20	30

Note. Adapted from "Effects of six warm-up protocols on sprint and jump performance" by R. E. Vetter, 2007. *The Journal of Strength and Conditioning Research, 21,* 819-823.

Session 3: HIIT 30:15. This session was the same as session two but the work-to-rest ratio was set at 30 seconds of sprinting and 15 seconds of passive recovery. All other measures and procedures were the same.

Measures

Heart rate (HR). Each participant's HR was measured via a heart rate monitor (Polar Inc., Port Washington, New York, USA) that consists of a small elastic band that was strapped around each participant's chest before any activity began. The heart rate monitor signals are transmitted to a digital wrist watch that was monitored by the researchers. Heart rate was recorded after each individual sprint.

Metabolic measures. Each participant was wearing a mouthpiece that was connected to a metabolic cart analyzing exhaled air to provide pulmonary gas responses to exercise and other metabolic and respiratory measures of interest. Specifically, the metabolic cart assessed oxygen consumption accumulation ($ACVO_2$, L/min), carbon dioxide production (VCO_2, L/min), and respiratory exchange ratio (RER) every minute.

Blood lactate concentration. Capillary blood samples were taken throughout the high-intensity exercise sessions to analyze blood lactate levels (mmoL) using a calibrated, portable blood lactate analyzer (Lactate Plus, Nova Biomedical Corp., Waltham, Washington USA). The blood lactate analyzer was calibrated before each exercise testing session. Samples were taken immediately after resting energy expenditure measurement, at the two minute mark during each three minute rest period between sets of sprints and once during the last recovery period at the two minute mark for a total of five samples.

Rating of perceived exertion (RPE): Participants provided a rating of perceived exertion after the second and fourth sprint of each set, using the OMNI Scale of Perceived Exertion (Utter et al., 2004). The scale ranges from 0-10, with zero representing no exertion and 10 representing maximal exertion during exercise. Average RPE was reported for each set of sprints. The scale was explained to participants before each session began.

Session rating of perceived exertion (S-RPE). Approximately 20 minutes after the participants were finished with the final set of sprints, they provided a session rating of perceived exertion for the entire session using the Session Rating of Perceived Exertion (S-RPE) (Foster et al., 2001). Similar to the OMNI RPE scale, the S-RPE scale is a 0-10 scale in which zero signifies no exertion and a 10 indicates maximal exertion for the entire session. The scale was described to the participants as the global difficultly of the entire session.

Perceived recovery status (PRS). Perceived recovery between each series of intervals was determined using a modified Perceived Recovery Status Scale developed by Laurent et al. (2011). The PRS is a scale ranging from 0-10. A value of 0 indicates the participant is very poorly recovered / extremely tired and a value of 10 indicates the participant is very well recovered / highly energetic. An explanation of the scale was given to the participant before the

session begins. PRS was recorded at minute one, two, and three during each three minute passive recovery stage.

Oxidative contribution. A metabolic cart was utilized to estimate the oxidative contribution to overall energy expenditure during the sessions. To calculate this estimation, each participant was properly fit to a metabolic cart to measure accumulated VO_2 (L/min), gross VCO_2 (L/min), and RER. Net VO_2, which is the oxygen consumed due to exercise (i.e., oxygen consumed above rest), was calculated for every minute of the protocol. RER measurements were also recorded every minute during the sessions and corresponded with the table, "Thermal Equivalents of Oxygen for the Nonprotein RQ" (Zuntz, 1901), to determine the kCals expended per liter of oxygen consumed (kCals per LO_2). Net VO_2 was then be multiplied by kCals per L of O_2 to determine the kCals expended during that minute of exercise.

Formula for Oxidative Contribution

$VO_{2gross} - VO_{2rest} = VO_{2net}$

RER corresponds to kCals per L of O_2

kCals per L of O_2 x VO_{2net} = kCals per minute

Example:

1.93 = L O_2/min - .32 L O_2/min = 1.61 L O_2/min

RER (0.86) corresponds with Nonprotein RQ = 4.875 kCals/L O_2

1.61 L O_2/min * 4.875 kCals/L O_2 = **7.85 kcals/min**

Glycolytic contribution. Glycolytic contribution to overall energy expenditure was estimated utilizing blood lactate concentration observed during testing. For every 1 mmol increase in blood lactate above resting is equivalent to the consumption of 3.0 ml O_2 per kilogram of body weight. Lactate samples were obtained every two minutes during the three minute passive recovery

phase of each HIIT session. The O_2 equivalent was converted as 1 L of O_2 = 21.1 kJ, to represent complete glucose oxidation. Kilojoules were further converted to kcals by dividing kJ by 4.184. An example is provided below:

Mass = 70kg

Resting lactate = 1.0 mmol / After 4 sprints = 8.0 mmol = 7 mmol change in lactate

7 mmol * 3.0 ml O2 = 21 ml O2

21 ml O2 * 70kg = 1470 ml O2

1470 ml O2 = 1.470 L O2

1.470 L O2 * 21.1 kJ = 31.02 kJ / 4.184 = **7.41 kCals**

Excess Post-Exercise Oxygen Consumption (EPOC) measurement. During the three minute passive recovery period and the final recovery period EPOC was measured. This is done in accordance with Scott (2009) by recording the gross VO_2 and converting that measurement to represent energy expenditure as 1 L of O_2 = 19.6 kJ. An example is provided below:

EPOC = 5.0 L O_2 min

Resting = .32 L O_2 min

Gross EPOC – Resting VO_2: 5.0 L O_2 min - .32 L O_2 min = 4.68 L O_2 min

4.68 L O_2 min * 19.6 kJ = 91.7 kJ / 4.184 = **21.92 kCals**

Total Energy Expenditure. During session two and three total energy expenditure was calculated. Total energy expenditure was determined by summating oxidative, glycolytic and EPOC EE measurements.

Statistical Analysis

Relative contribution of energy system involvement reported during the HIIT sessions was analyzed using a series of 2 (HIIT bout) x 4 (set of sprints) repeated measures ANOVA to identify any significant main effect. When appropriate, univariate post-hoc follow-ups including a dependent paired t-tests to identify significant differences and 95% confidence interval for real change. In order to determine significant differences in total energy expenditure between sessions, a paired t-test was employed. All data was presented as mean ± SD unless stated otherwise. Power was reported as 1-β and effect size for main effects are reported as partial eta squared (η_p^2) whereas post-hoc effect sizes are presented as Cohen's *d*. Post-hoc effect sizes were classified, in accordance with Cohen (10) with a small effect size $d = 0.20$, a medium effect size $d = 0.50$ and a large effect size $d = 0.80$. Statistical significance is set at the 0.05 level and all data was analyzed using the statistical package for social sciences (SPSS, v 22, IBM Corporation, Armonk, NY).

CHAPTER 4: RESULTS

Descriptive characteristics of the participants including body fat (BF) percentage, height, weight, age, VO_{2peak}, VO_2 percentile rank, peak velocity and 110% of peak velocity are shown in Table 2.

Table 2. Participant characteristics (30:30; n = 17) (30:15; n=15)

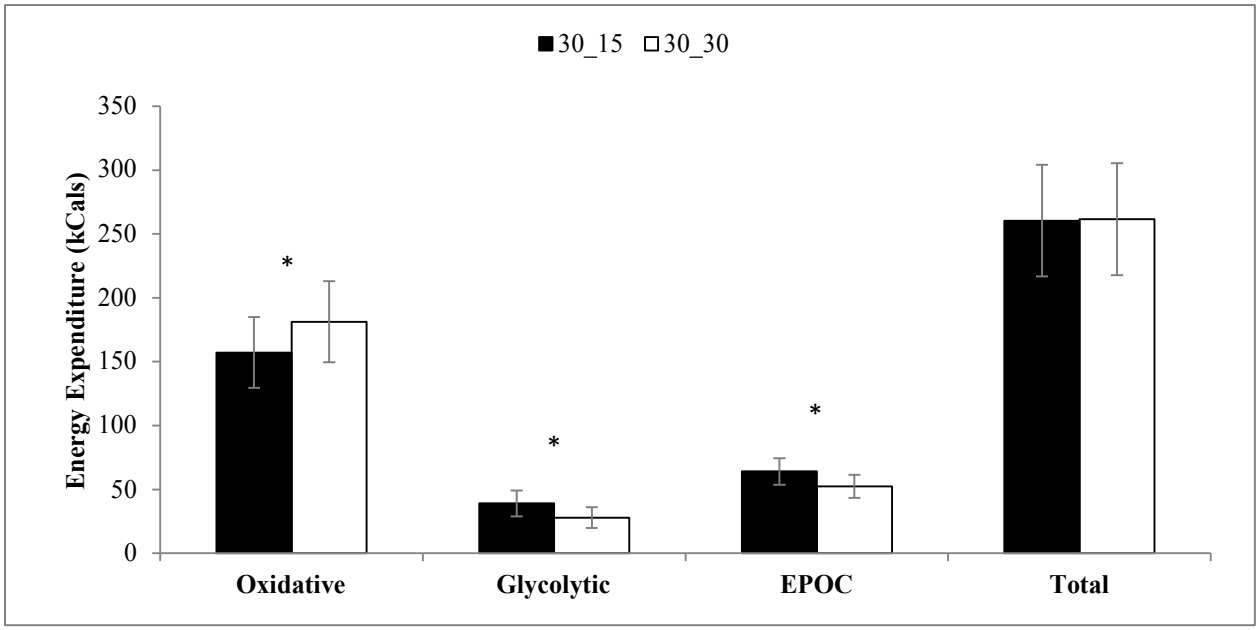
	Mean	SD
Body fat (%)	16.6	6.5
Height (cm)	174	7.5
Weight (kg)	72.9	11.6
Age (years)	23.4	3.7
VO_{2peak} (ml/kg/min)	50.9	5.8
VO_2 % Rank	86.2	12
pVEL (mph)	10.3	0.7
VEL110% (mph)	11.3	0.9

Note: Descriptive data is displayed as mean and standard deviation (SD) for 8 males and 9 females. BF, body fat; VO_{2peak}, peak oxygen consumption; VO_2, oxygen consumption; pVEL, peak velocity; VEL110%; 110% of peak velocity.

Absolute kCal Contribution between Sessions

Figure 1 displays the differences in absolute kCal expenditure between sessions from the oxidative system, glycolytic system, and EPOC. When comparing total session energy expenditure, independent t-test revealed no significant difference between 30:15 or 30:30 HIIT sessions ($p = .947$). However, there was a significant difference ($p = .037$) between the two sessions with respect to overall kCal expenditure from the oxidative system during 30:30 compared to 30:15. There was also a significant difference ($p < 0.01$) in kCal expenditure from the glycolytic system during 30:15 compared to 30:30. Similarly, a significant difference ($p <0.01$) in EPOC contribution to overall kcal expenditure when comparing 30:15 and 30:30 was found.

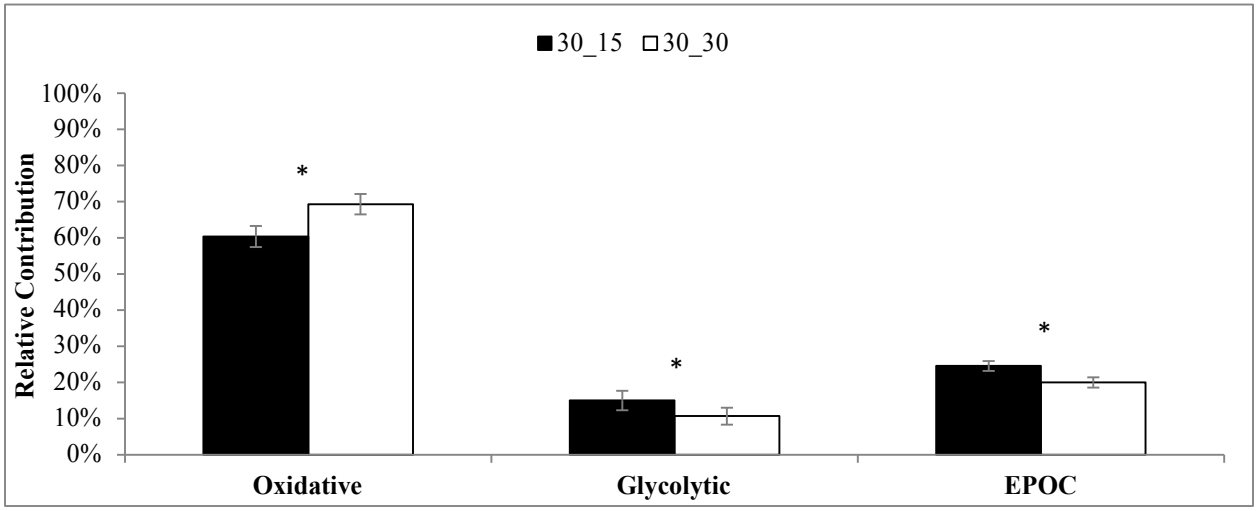
Fig. 4. Absolute kCal contribution from the oxidative system, glycolytic system, EPOC, and total kCal expenditure. Energy expenditure is reported as kCals per session; *, $p \leq 0.05$ when comparing 30:15 and 30:30.



Relative Energy System Contribution between Sessions

Figure 2 displays the relative contribution to total EE between sessions from the oxidative system, glycolytic system, and EPOC. When comparing relative glycolytic contribution, an independent t-test revealed a significant difference between 30:15 and 30:30 ($p < 0.01$).There was also a significant difference ($p < 0.01$) between the two sessions with respect to the oxidative contribution during 30:30 when compared to 30:15. There was also a significant difference ($p < 0.01$) in EPOC contribution during 30:15 compared to 30:30.

Fig. 5. Relative contribution from the oxidative system, glycolytic system, and EPOC. Relative contribution is represented as a percentage of total energy expenditure; *, $p \leq 0.05$ when comparing 30:15 and 30:30.



Relative Energy System Contribution per Set

Results from the repeated-measures ANOVA revealed a significant difference between sets and the two HIIT sessions in regards to the oxidative ($p = .047$, $\eta_p^2 = .105$, $1\text{-}\beta = .587$) and glycolytic ($p = .022$, $\eta_p^2 = .121$, $1\text{-}\beta = .713$) contribution. Repeated-measures ANOVA revealed there was no significant interaction on EPOC contribution between sets and the two HIIT sessions ($p = .841$, $\eta_p^2 = .006$, $1\text{-}\beta = .076$). Post-hoc measures revealed a significant difference comparing 30:15 and 30:30 between all four sets, in regards to the overall oxidation contribution and glycolytic contribution ($p < 0.01$ at all comparative points) (See Figure 3).

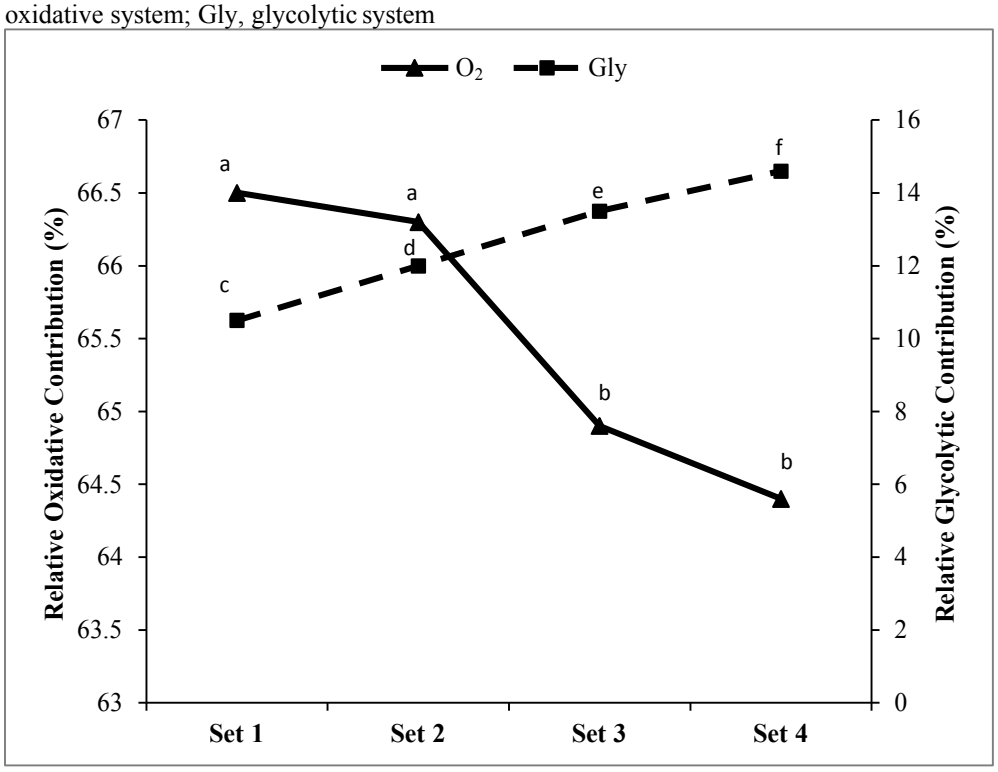
Energy System Contribution between Sets of Sprints

Figure 3 displays the relative contribution of energy from the oxidative system and glycolytic system between both HIIT sessions. Results from a repeated-measures ANOVA revealed a significant difference between relative contribution from oxidative system ($p < 0.01$) and glycolytic system ($p < 0.01$) between sets.

As can be seen, the oxidative contribution to overall EE during set 1 was not significantly different from set 2 ($p = .59$). There was, however, a significant difference between set 1 and set 3 ($p = .001$) and set 4 ($p < 0.01$). There was also a significant difference between set 2 and set 3 ($p < 0.01$) and set 4 ($p < 0.01$). There was no significant difference between set 3 and set 4 ($p = .071$). There was a significant interaction on the oxidative contribution between sets and the two HIIT sessions ($p = .047$, $\eta_p^2 = .105$, $1\text{-}\beta = .587$).

Repeated-measures ANOVA also revealed that glycolytic contribution to set 1 was significantly different than set 2 ($p < 0.01$), set 3 ($p < 0.01$), and set 4 ($p < 0.01$). Set 2 was also significantly different from set 3 ($p < 0.01$) and set 4 ($p < 0.01$). Lastly, set 3 was significantly different from set 4 ($p = .002$). There was a significant interaction on the glycolytic contribution between sets and the two HIIT sessions ($p = .022$, $\eta_p^2 = .121$, $1\text{-}\beta = .713$).

Fig. 6. Relative contribution from the oxidative system and glycolytic system across the four sets. Relative contribution is represented as a percentage of total caloric expenditure; *, $p \leq 0.05$ when comparing sets. O_2, oxidative system; Gly, glycolytic system



Note: Means with different letters indicate significant difference between sets.

CHAPTER 5: DISCUSSION

High-intensity interval training has undoubtedly become an effective training modality for athletes and among the general population (Gibala & McGee, 2008; Gibala, Little, Macdonald, & Hawley, 2012; Dupont, Akakpo, & Berthoin, 2004; Sandbakk, Sandbakk, Ettema, & Welde, 2013; Greeley, Martinez, & Campbell, 2013). The growth in popularity is, in part, due to the notion that an individual can expend the same or more kCals in less time when contrasted to more traditional steady-state exercise (Gillen & Gibala, 2013). Due to the increase in popularity stemming from the notion of similar EE in less time, providing an accurate estimation of EE during and following a bout of HIIT should be considered a valuable component for improving exercise regimens (Scott, 2002; Scott & Fountaine, 2013). Previously, Scott (2005a) affirmed that singularly relying upon pulmonary gas exchange may not be the most valid estimation of overall EE when the modality consists of high-intensity, intermittent work due to the purported glycolytic contribution. What has been shown, though, to be a valid method of estimating EE is the integration of pulmonary gas exchange in conjunction with change in blood lactate during high-intensity, intermittent exercise (Margaria, Aghemo, & Sassi, 1971; di Prampero & Ferretti, 1999). While novel, this technique has primarily been utilized within resistance training and cycling research (Scott, 2006; Scott, Croteau, & Ravlo, 2009; Capelli & Prampero, 1995). Therefore, the primary purpose of this study was to examine the variation in oxidative and glycolytic contribution during two HIIT protocols using a 1:1 work-to-rest ratio and a 2:1 work-to-rest ratio. The most salient finding from this study revealed 30:15 expended nearly identical kCals as 30:30. In addition, results indicated 30:15 elicited a greater glycolytic contribution on overall energy expenditure when compared to 30:30.

An interesting finding from the current study revealed 30:15 resulted in a nearly identical amount of kCals expended compared to 30:30 (260 kCals vs. 261 kCals, respectively; see Figure 1). It is important to note that total amount as well as rate of work performed was held constant during the two HIIT sessions. The only difference between protocols was recovery after each sprint (30 seconds vs. 15 seconds). Despite the similarity in total EE between the two protocols there were significantly different contributions of kCal production among the energy systems. During 30:15 (vs. 30:30), results revealed ~4% increase in glycolytic contribution and ~4% increase in EPOC contribution to overall EE. In contrast, during 30:30 (vs. 30:15), whereas results revealed ~9% increase in the oxidative contribution to overall EE.

During 30:15 and 30:30, results revealed a steady increase in the overall contribution from the glycolytic system. However, 30:15 exhibited greater demand from the glycolytic system during exercise when compared to 30:30. The amplified demand from the glycolytic system during 30:15 was due to increased accumulation of blood lactate across all four sets when compared to 30:30 (as reflected in greater glycolytic contribution). This finding is in agreement with those of Gosselin, Kozlowski, DeVinney-Boymel, and Hambridge (2012) that report significant increases in blood lactate concentration when increasing work-to-rest ratios from 30:30 to 60:30 ($p < .05$). Lactate has long been an 'of interest' metabolite to study and it is now becoming apparent that lactate is associated (but not causal) with fatigue and, subsequently, an individual's recovery (Brooks, 2001; Bishop 2012). Indeed, it has been noted by Bishop (2012) that the accumulation of H^+ may be one of the most influential underlying metabolites that affect high-intensity, intermittent activity. Inadequate recovery between intermittent sprints produces an accumulation of H^+ which disrupts the muscle pH balance (i.e., metabolic acidosis) and impairs the calcium kinetics, ultimately effecting the excitation-contraction coupling phase for a

cross-bridge (Bishop, 2012). These processes tend to coincide with greater accumulation of lactate across sets of sprints.

Moreover, lactate is an appropriate metabolite to analyze within this paradigm as it is the end product of anaerobic glycolysis, therefore, increased blood lactate should indicate increased contribution from the glycolytic system (Brooks, 2007). It seems plausible, that if the "work" duration is consistent, and the "rest" ratio decreases (e.g., 1:1 versus a 2:1), the shorter recovery duration would inhibit the ability of oxidative pathways to clear significant lactate. Indeed, Tomlin and Wenger (2001) note that during recovery after a bout of high-intensity intermittent exercise, ~65% of blood lactate accumulation is converted back to pyruvate whereupon it will be transitioned into the Krebs cycle, and subsequently the electron transport chain, which is an oxidative process. Lactate is also cleared from muscle via gluconeogenesis utilizing the Cori Cycle and can undergo shuttling via oxidation into type I muscle fibers (Brooks, 2007). Thus, it seems that the 15 seconds of recovery during 30:15 utilized in this study was an insufficient duration, disallowing the oxidative system to clear a significant amount of lactate when compared to 30:30. In contrast, 30:30 allowed participants 15 additional seconds to recovery after each sprint and results seem to indicate increased recovery duration after each sprint allowed improved lactate clearance, inducing a greater oxidative contribution to overall energy expenditure.

During a recovery period after a high-intensity effort, the oxidative system is the primary pathway to resynthesize ATP (Gastin, 2001; Brooks, 2007; Tomlin & Wenger, 2001). It is clear that during the repeated sprints used in this study, the oxidative system contributed significantly, especially during recovery. Indeed, results show that 30:30 displayed a greater contribution from the oxidative system when compared to the 30:15 (~9% difference). The difference in oxidative

contribution could likely be attributed to the additional 15 seconds of recovery during 30:30 when compared to 30:15. The additional recovery time allowed participants to consume a greater quantity of O_2 and, subsequently, the increase in O_2 consumption allowed for adequate clearance of blood lactate via oxidative pathways (Brooks, 1999). This, in turn, ultimately decreased the overall glycolytic contribution during the 30:30 to overall EE. The additional time during recovery could have also potentially augmented PCr replenishment. If PCr is re-synthesized to a greater extent in the 30:30, this could have allowed for greater PCr contribution to the subsequent sprint, which could have increased high energy phosphates to produce energy and therefore, also decreasing the utilization of the glycolytic system. However, direct measurement of PCr contribution was not included in this study so this is a speculative notion, but plausible nonetheless.

The available research has shown high-intensity, intermittent exercise stimulates an increase in EPOC. (Bahr & Sejersted, 1991; Laforgia, Withers, Shipp, & Gore, 1997; Børsheim & Bahr, 2003; Townsend *et al*., 2013). Not only does EPOC indirectly represent EE from the phosphagen system, it is also known to replenish O_2 saturation within the muscle, blood, and water, aid in the removal of lactate post-exercise, repair damaged tissue, and decrease body temperature by utilizing oxidative pathways (Bahr & Sejersted, 1991; Baechle & Earle, 2008). The findings within the current study are consistent with the previous literature stating high-intensity, intermittent exercise elicits a significant effect on EPOC (Bahr & Sejersted, 1991; Laforgia, Withers, Shipp, & Gore, 1997; Børsheim & Bahr, 2003; Townsend *et al*., 2013). Although, not the primary aim of the study, results showed significantly greater effect on EPOC during 30:15 (64 kCals), compared to 30:30 (52 kCals). While the difference in kCal expenditure between the two sessions stemming from EPOC was modest at 12 kCals, the larger focus should

be directed towards the notion that both HIIT protocols elicited a noticeable contribution on EPOC.

In this investigation, EPOC was represented by the three-minute passive recovery between each set of sprints, as well as the seven-minute post-exercise session recovery period (i.e., 19 minutes per session of EPOC). While traditional measures of EPOC range from 1 hour post-exercise to 24 hours post-exercise, our focus centered around within session EPOC as well as immediate post-exercise EPOC (Bahr & Sejersted, 1991; Laforgia, Withers, Shipp, & Gore; 1997; Abboud, Greer, Campbell, & Panton, 2013). EPOC duration is an important aspect of this study because it is meant to serve as a representation of the phosphagen system (ATP and phosphocreatine) contribution to overall EE (Bangsbo *et al*., 1990; Haseler, Hogan, & Richardson, 1999; Scott, Littlefield, Chason, Bunker, & Asselin, 2006). Even within this relatively narrow frame of EPOC, results indicate that EPOC is valuable component to overall EE as it represented 61% (30:15) and 54% (30:30) of the total session duration.

This study is among the first to examine the glycolytic, oxidative, and EPOC contribution during bouts of repeated sprints utilizing two different work-to-rest ratios. Results from this study showed a linear increase in the glycolytic contribution as sets progressed (e.g., set 1 to set 2, set 2 to set 3) (See Figure 3.). This study revealed an overall glycolytic contribution of 15% during 30:15 and 10.7% during 30:30 to total EE. The quantification of the contribution of kCals resulting from glycolytic activity serves to underscore the importance of utilizing change in blood lactate during high-intensity, intermittent activities. If pulmonary gas exchange exclusively estimated EE during these two sessions, overall kCal expenditure would have underestimated 30:15 by 39 kCals (15%) and 30:30 by 28 kCals (10.7%). Also shown within Figure 3 is a logarithmic decay in oxidative contribution across all four sets. This decline can potentially be

attributed to reduced reliance of oxidative pathways to restore ATP within a sufficient time span as the sets progressed. As the sets of sprints progressed, metabolite accumulation systematically increased and may have facilitated a greater reliance upon type II muscle fibers. This, in turn, may have precipitated a shift in reliance on the glycolytic system and decreasing the reliance on the oxidative system due to its ATP turnover rate. Interestingly, as the sets progressed, there was no significant difference in the relative contribution to EE from EPOC. The recovery duration (EPOC) between sets did not allow full recovery before the subsequent set of sprints due the noticeable accumulation of blood lactate and elevated O_2 consumption. Lactate accumulation has been shown to clear 1 hour post-exercise, O_2 resaturation of blood and tissue occurs ~1 hour post-exercise and PCr recovery may take ~3-5 minutes (Bahr & Sejersted, 1991; Tomlin & Wenger, 2001). It may be probable the three-minute recovery after each set of sprint merely allowed for a specific amount of lactate clearance, O_2 saturation and PCr recovery. EPOC could have potentially been greater across the sets if the recovery duration was extended (i.e., longer EPOC measurement), but since each recovery phase was three minutes, it may have elicited a similar metabolic recovery after each set, therefore producing a similar EPOC contribution.

While the findings within this study are novel and serve to further the body of knowledge around EE in HIIT, it is not without limitations. Specifically, the measurement of blood lactate to represent glycolytic contribution, while validated, has not been utilized during HIIT, but has been shown to portray an accurate representation during running (Margaria, Aghemo, & Sassi, 1971; di Prampero & Ferretti, 1999). The limitation might center on with the timing of peak blood lactate accumulation after a set of sprints. The current study measured blood lactate at two minutes into the three minute passive recovery phase. Scott (2006) has shown blood lactate to peak two minutes after exercise, however, this was examined during resistance training and not

during intermittent sprint work. Our findings are further restricted by the small sample size, particularly within gender (9 females; 8 males). However, our intent was to duplicate the sample size between the two sessions (e.g., 9 females, two HIIT sessions, 18 females total; 8 males, two HIIT sessions, 16 males total; $n = 34$).

In conclusion, the primary findings from this study show that total energy expenditure between 30:15 and 30:30 elicited similar amounts of total kCals expended. Despite near identical kCal expenditure, the relative contribution between the glycolytic and oxidative system were different. There was a steady linear increase in the overall glycolytic contribution and a logarithmic decrease in the overall oxidative contribution between both sessions as the sets progressed. Interestingly, though, there were no differences in EPOC when examining the sessions globally. Our results display the relative contribution between the primary energy systems during a bout of HITT and the relative contribution during EPOC. Utilizing pulmonary gas exchange to represent the oxidative system and EPOC and measuring the change in blood lactates to represent the overall glycolytic contribution depicts an acceptable EE estimation during a bout of HIIT.

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APPENDIX A: INFORMED CONSENT

BGSU
BOWLING GREEN STATE UNIVERSITY

Exercise ScienceProgram
School of Human Movement, Sport, and Leisure Studies

Informed Consent

Investigators: Chris Irvine **Phone:** (419) 509-8522
Kaitlyn Kielsmeier
Matt Laurent, Ph.D., CSCS

Project Title: Determination of Total Energy Expenditure During and Following Repeated High-Intensity Intermittent Sprint Work

Purpose: We are Chris Irvine and Kaitlyn Kielsmeier and we are graduate students in Kinesiology at Bowling Green State University. You are being asked to participate in a study to look at total-body energy expenditure (caloric burn) during high-intensity interval sprints and during the recovery following. The purpose of the study is to estimate the "metabolic burn" or energy cost involved with high-intensity activities, which are largely unknown, therefore, many people may be misinformed. We are interested in testing men and women that are fairly well-trained and are used to performing high-intensity interval-type work. (Examples of high intensity interval-type work would be interval training as a runner or cyclist, recreational but competitive team sport participation such as soccer, volleyball, basketball would also be an example). With the rising popularity of interval-type fitness programs such as Crossfit, P90X, Insanity, and circuit training, it is important to know how much energy is being expended with these activities in order to properly train without over-working or under-working thebody.

If you participate, you will be asked to complete three (3) exercise sessions on a treadmill in our laboratory. The first lab session will last approximately 45-60 minutes, and the second and third lab sessions will last approximately 60-75 minutes each for a total of approximately 2.45-3.15 hours to complete this particular study. You are welcome and encouraged to ask any questions about the study at any time if you wish. If at any time during the study you would like to stop participating, you may do so. You are not required to complete the study.

To be included in thisstudy:
1. You must be **at least** 18 years of age
2. You must be **younger** than 35 years of age
3. You must regularly exercise a MINIMUM of 3 times perweek
4. Your exercise sessions must be AT LEAST 30 minutes induration
5. You must participate in interval training or interval-type training
   (Examples are competitive basketball, volleyball, soccer, etc.)

Procedures:
If you are able and willing to participate, you will be asked to complete two (2) sessions of exercise tests on a treadmill. Each session is described in detail below. All testing will be conducted in the Exercise Physiology Lab in Eppler South Room 124 at Bowling Green State University.

Session 1: *Health Screening, Descriptive Data, Maximal Exertion Treadmill Testing and Sprint Familiarization. Before performing any exercise, you will be asked to come to the lab and fill out some forms about yourcurrent health status and current workout schedule. These forms will be used to make sure you can safely participate. The information you provide will be kept confidential. It is important that you answer these questions accurately and completely. Any questions you may have about your participation or the forms you complete are welcomed and will be answered to your satisfaction. If these forms indicate it may not be safe for you to participate, you will not be allowed to continue.

After you have filled out the forms and it is determined if it is safe for you to participate you will be measured for descriptive data (age, height, weight, and percent body fat). Percent body fat will be estimated by measuring skinfold thickness; (men: chest, abdomen, and thigh; women: tricep, iliac, thigh). This process requires me to pinch your skin and use a device to measure the thickness of the pinched skin. You will then be asked to perform a maximal exertion treadmill test. During this test, you will run on treadmill for 6-12 minutes depending on your fitness. Once a standard warm-up has been completed, the maximal test will begin. The test will start at a moderate jogging speed. During exercise, we will make it more difficult by increasing the speed of the treadmill every minute. The first part of the test will be easy, but the test will get slightly harder each minute, getting very hard after several minutes. You will be encouraged to provide your best effort and continue until you feel you cannot maintain the required effort. When you let us know that you can no longer continue, the test will be stopped, and you will be monitored during a low intensity cool-down. The test may also be stopped if we feel it is not safe for you to continue. During this test you will be required to breathe through a mouthpiece and wear a nose clip, but you will be able to freely breathe room air through your mouth.

Approximately 20 minutes after the maximal exertion treadmill test, you will be asked to perform one set of four high-intensity interval sprints. The speed during the final full minute of the maximal exertion treadmill test will elicit velocity at maximal oxygen consumption (vVO_2 max). The high-intensity interval sprints will be performed at 110% of that final speed. For example, if 10 mph was the speed reached and maintained for the last full minute at maximal oxygen uptake, then running at 110% of vVO_2 max would be a speed of 11mph.

**Maximal* refers to exercise intensity. The final portion of the treadmill test will require you to exercise at your "maximal" effort (i.e., as hard as youcan).

Session 2: Repeated High-Intensity Running Trial. This session will begin at least two (2) days from the maximal exertion treadmill test. You will be given at least 2 days but no more than 7 days rest between sessions. Prior to the start of the test, you will sit and have your resting energy expenditure recorded for 5-10 minutes. This requires you to sit quietly in a chair breathing through the same mouthpiece that you were wearing in session 1. You will then be asked to perform a standardized warm-up (same one as session 1). After you warm-up, the sprint intervals will be performed on a motorized treadmill where you will be asked to perform four (4), 30-second sprints with a 30-second rest period. The speed of the sprints depend on the speed that you ended with on your maximal exertion test in session 1. Simply, you will run at 110% of the velocity that you reached before termination of the maximal test. (For example, if you reached a velocity of 10 mph as the last stage of your maximal test, your sprints would be run at 110% of 10 mph, which is 11 mph). Three (3) minutes of seated rest will be given once your first set of sprints is complete. One more set of four (4), 30-second sprints will be performed after the three (3) minute rest. At the end of the fourth set of sprints, you will sit in a chair while energy expenditure is again recorded while you continue breathing through the mouthpiece until your metabolic measurements have come down to resting values (no longer than 20 minutes of seated recovery). This lab session will last about 45-60 minutestotal.

Session 3: Repeated High-Intensity Running Trial. This session will begin at least two (2) days from session 2. You will be given at least 2 days but no more than 7 days rest between sessions. This session will be the same as session 2 except you will perform four sets of 4, 30 seconds sprints with a 15-second rest period. All other testing measures and procedures are the same.

Prior to beginning each sprint, you will use a *Perceived Recovery Status Scale* that is presented to you that lets us know how recovered you feel when you are to perform the next set of sprints. At the completion of each 30-second sprint, you will be asked to rate how you feel using the *Rating of Perceived Exertion Scale* provided. There is no "right" or "wrong" answer to how you feel. Approximately 20 minutes after you complete your session, you will be asked to rate the entire session using the same scale.

During both sessions you will be required to wear a heart-rate monitor around your chest near the breastbone. The monitor resembles a small belt, and it does not hurt nor stick to your skin. Also, you will be asked to wear the same mouthpiece and nose clip throughout all of session 2 that you wore during the maximal exertion treadmill test.

Risks: Potential risks to your health and well-being because of participation include: 1) cardiovascular injury (heart attack, stroke, and death – risk is estimated at <0.01%), 2) severe acute fatigue (100% likely) at the end of the maximal exercise test, 3) lightheadedness, dizziness, nausea - commonly experienced 4) all other possible risks associated with intense exercise. You should know that the chance of having a heart attack, stroke or other complication is possible but highly unlikely. You should understand that you will almost definitely experience fatigue during at least one of the sessions. This fatigue is similar to what you would experience during a normal, high-intensity exercise session that you would perform as a part of your training or sport participation. Some of the common side-effects associated with this type of exercise would be short periods of dizziness and in some cases lead to an upset stomach similar to when you feel sick.

If you happen to experience any of these side effects, they will most likely go away, except for a cardiovascular injury. If fatigue or sickness does happen, it will happen quickly after you stop exercising and will most likely go away within 10 – 15 minutes. If you experience any of these conditions, you will be asked to stay in the laboratory with the investigator so that you can be monitored until all symptoms have gone away and you feel better. If injury occurs, such as a heart attack or stroke, you will be provided immediate care by the investigators and emergency medical assistance will be sought when necessary. You will be responsible for paying for any emergency measures that may be required if you incur an injury due to the stated risks of participation. You are not releasing the researchers from liability. If you incur an injury due to failure of equipment or negligence of the researchers, you will not pay for your own injury.

Benefits: Benefits to you for participating in this research are - you will receive information regarding your fitness: VO_{2max}, anaerobic threshold, and percent body fat, and your rate of energy expenditure. Your VO_{2max} will show you your aerobic capacity and is the gold standard for determining aerobic fitness level. Your anaerobic threshold is a useful tool in subsequent training as it is an indicator of the highest physical intensity you can maintain before experiencing premature fatigue. You can use this data to help plan your day-to-day training program if you so desire. This information will be shared with you following the second session. You are encouraged to ask questions about this data to maximize your benefits.

Confidentiality: After initial data collection, your name will not be associated with this data. Only the investigators and other personnel associated with this study will have access to this information, which will be kept in a locked room. No publication or other public material will carry your name as a participant.

Voluntary Participation: Your participation in this study is completely voluntary, and you can refrain from participating or answering any or all questions without penalty or explanation. You are free to withdraw consent and to discontinue participation in any exercises at any time. Deciding to not participate or to withdraw will not affect your relationship with BGSU.

Contact Information: If you have any questions or comments about this study, you can contact Chris Irvine at 419-509-8522 or cirvine@bgsu.edu, Dr. Matt Laurent at (419) 372-6904 or cmlaure@bgsu.edu, or Kaitlyn Kielsmeier at (815) 543-0943 or kkielsm@bgsu.edu. If you have questions about the conduct of this study or

your rights as a research participant, you may contact the Chair, Human Subjects Review Board, Bowling Green State University, (419) 372-7716 (hsrb@bgsu.edu).

Authorization: I have read this document, and the study has been explained to me. I have had all of my questions answered. I volunteer to participate in this study.

I know that I will receive a copy of this letter.

_____ _____

Participant's Signature Date

APPENDIX B: MEDICAL HISTORY QUESTIONNAIRES

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

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

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

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

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

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

If you answered

YES to one or more questions

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

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

NO to all questions

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

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

DELAY BECOMING MUCH MORE ACTIVE:

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

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

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

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

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

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

NAME _____

SIGNATURE _____ DATE _____

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

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

continued on other side...

EXERCISE PHYSIOLOGY LABORATORY
124 EPPLER SOUTH, SCHOOL OF HMSLS
BOWLING GREEN STATE UNIVERSITY

MEDICAL HISTORY QUESTIONNAIRE

All information given is personal and confidential. It will enable us to better understand you and your health and fitness habits. In addition, we will use this information to classify your health status according to the American College of Sport Medicine (ACSM) recommendations for risk stratification (ACSM, 2009). Please let us know if and when you have changed your medication (dose & type), diet, exercise or sleeping habits within the past 24 or 48 hours. It is very important for you to provide us with this information.

NAME_____ AGE_____ DATE_____

OCCUPATION_____

1. *FAMILY HISTORY

Check each as it applies to a blood relative:

Heart Attack yes_____ no_____ unsure_____
If yes, age at onset____ yrs; relation to you _____

Sudden Death yes_____ no_____ unsure_____
If yes, age at onset____ yrs; relation to you _____

Coronary Revascularization
If yes, age at onset____ yrs; relation to you _____

Father's Age_____ Deceased_____ Age at death_____
(*Before 55 yr. in father or first-degree male relative)

Tuberculosis yes_____ no_____ unsure_____
Stroke yes_____ no_____ unsure_____
Asthma yes_____ no_____ unsure_____
High Blood Pressure yes_____ no_____ unsure_____
Circulatory Disorder yes_____ no_____ unsure_____
Heart Disease yes_____ no_____ unsure_____

Mother's Age_____ Deceased_____ Age at death_____
(*Before 65 yr. in mother or first-degree female relative)

2. PERSONAL HISTORY

Check each as it applies to you:

* **Age** (men ≥ 45 yr; women≥ 55 yr) yes_____no_____

* **Current Cigarette Smoking** yes_____no_____unsure_____

* **Sedentary Lifestyle** yes_____no_____unsure_____
Persons not participating in at least 30 min of moderate intensity physical activity on at least 3 days/wk for at least 3 months.

* **Obesity – BMI >30 kg·m^{-2}** yes_____no_____unsure_____
If yes, give value:_____kg·m^{-2}
Waist circum. > 40" men; 35" women: yes_____no_____

* **High Blood Pressure** yes_____no_____unsure_____
Systolic Blood Pressure >140mmHg or diastolic >90mmHg
If yes, give value:_____/_____mmHg.

* **Dyslipidemia** yes_____no_____unsure_____
Total Serum Cholesterol >200 mg·dl^{-1} ; value:_____mg·dl^{-1}
LDL-C ≥ 130 mg·dl^{-1} ; value:___mg·dl^{-1}
HDL-C ≤ 40 mg·dl^{-1} ; value:____mg·dl^{-1}
On lipid lowering medication: yes_____no_____unsure_____

* **PreDiabetes** yes_____no_____unsure_____
If yes, age of onset:_____years
Impaired fasting glucose ≥ 100 mg·dl^{-1} ; value:_____mg·dl^{-1}
Impaired glucose tolerance test: yes_____no_____
(Note: values confirmed by measures on two separate occasions)

***Negative Risk Factor:** yes_____no_____unsure_____
HDL ≥ 60 mg·dl^{-1} ; value:_____mg·dl^{-1}

Have you ever had:

Diabetes yes_____ no_____ unsure_____
Tuberculosis yes_____ no_____ unsure_____
Heart Attack yes_____ no_____ unsure_____
Angina yes_____ no_____ unsure_____
EKG Abnormalities yes_____ no_____ unsure_____
Asthma yes_____ no_____ unsure_____
Emphysema yes_____ no_____ unsure_____
Surgery yes_____ no_____ unsure_____
Stroke yes_____ no_____ unsure_____
Severe Illness yes_____ no_____ unsure_____
Hospitalized yes_____ no_____ unsure_____
Black Outs yes_____ no_____ unsure_____
Gout yes_____ no_____ unsure_____
Nervousness yes_____ no_____ unsure_____
Joint Problems yes_____ no_____ unsure_____
Allergy yes_____ no_____ unsure_____
Convulsions yes_____ no_____ unsure_____
Paralysis yes_____ no_____ unsure_____
Headaches yes_____ no_____ unsure_____
Depression yes_____ no_____ unsure_____
Chest Pain yes_____ no_____ unsure_____
Arm Pain yes_____ no_____ unsure_____
Shortness of Breath yes_____ no_____ unsure_____
Indigestion yes_____ no_____ unsure_____
Ulcers yes_____ no_____ unsure_____
Overweight yes_____ no_____ unsure_____
Hernia yes_____ no_____ unsure_____
Back Pain yes_____ no_____ unsure_____
Leg Cramps yes_____ no_____ unsure_____
Low Blood Pressure yes_____ no_____ unsure_____
Insomnia yes_____ no_____ unsure_____

For Office Use Only:
_____**Sum of positive and negative *CVD risk factors* (according to Table 2-3 ACSM (2009)**
NOTE: All risk factors are explained verbally to each person completing the questionnaire.

Classification according to ACSM (2009) (check one): _____ Low risk; _____Moderate risk; _____High risk

3. MEDICAL HISTORY

Are you presently taking any medications? Yes_____ No_____ _____
(Including over-the-counter medications and/or herbs) List name and dosage

Have you ever taken:

Digitalis	yes_____ no_____ unsure_____	Insulin	yes_____ no_____ unsure_____
Nitroglycerin	yes_____ no_____ unsure_____	Pronestyl	yes_____ no_____ unsure_____
High Blood Pressure Medication	yes_____ no_____ unsure_____	Vasodilators	yes_____ no_____ unsure_____
		Other	yes_____ no_____ unsure_____
Sedatives	yes_____ no_____ unsure_____		
Inderal	yes_____ no_____ unsure_____	If yes, list medications: _____	

4. EXERCISE HISTORY

Do you exercise? Yes_____ No_____ What activity_____

How long have you been exercising?_____

How many days do you exercise?_____ How many minutes per day?_____

What kinds of shoes do you work out in?_____

Where do you usually exercise?_____

Do you monitor your pulse during your workout?_____

Additional information from client interview to further assess health/coronary risk status:

_____

_____

_____

_____

_____ _____
Signature of Tester Date

APPENDIX C: RECUITMENT FLYER

INTERESTED IN EXERCISE PERFORMANCE TESTING?

A study being conducted at Bowling Green State University is in need of exercise enthusiasts to serve as volunteers to be evaluated in a research study. The study will be conducted over approximately the next 4 months and will take three (3), 30 minute to 1 hour exercise sessions on separate days for you to complete.

To qualify for the study:
1. You must be **at least** 18 years of age
2. You must be **younger** than 35 years of age
3. You must regularly exercise a MINIMUM of 3 times per week
4. Your exercise sessions must be AT LEAST 30 minutes in duration
5. You must participate in interval training or interval-type training
   (Examples are competitive basketball, volleyball, soccer, etc.)

As a result of participating you will receive a fitness evaluation (aerobic capacity, anaerobic threshold measurement and body fat percentage determination) and a training consult as a result of your participation free of charge!

LIMITED AVAILABILITY!!

If you are interested in learning more about this opportunity please contact:

Chris Irvine
Phone: 419-509-8522
Email: cirvine@bgsu.edu

BGSU
Bowling Green State University

APPENDIX D: CLASS ANNOUNCEMENT

Exercise Science Class Announcement

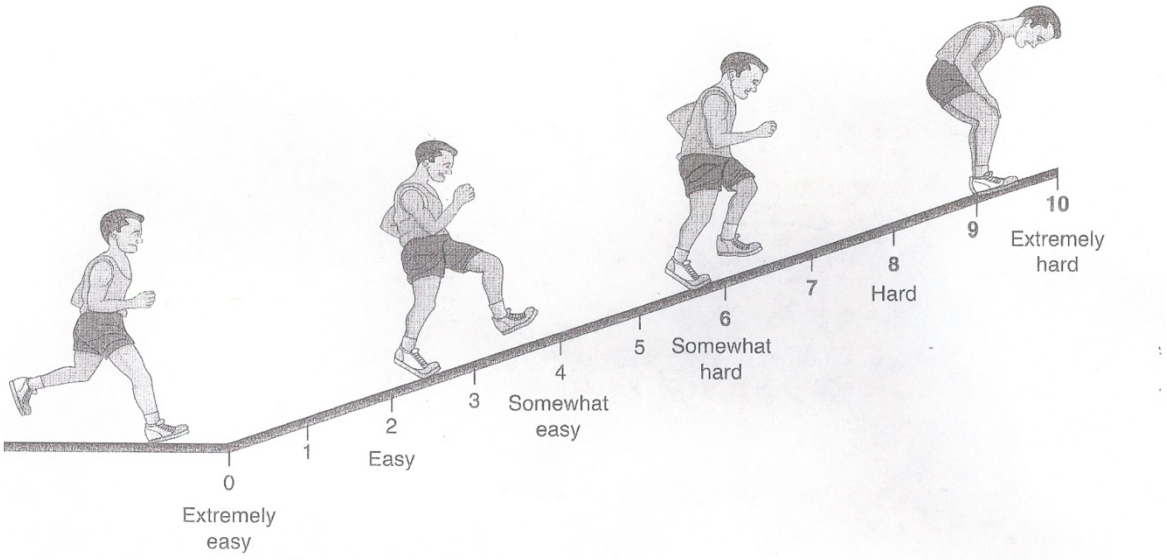
Dr. Matt Laurent and his graduate research assistant are in the process of conducting a study that is looking at energy expenditure (caloric burn) during and after repeated high-intensity sprint intervals. They are currently seeking healthy, trained individuals to volunteer for participation in their study. In order to volunteer, you must be at least 18 years old but not older than 35 years of age, exercise at least 3 days per week with at least one of those sessions being interval training or interval-type activity (i.e. basketball, volleyball, soccer, etc.). If you were to be selected as a participant, they would ask you to perform three (3) exercise testing sessions that would last about an hour to an hour and a half each. All trials will be conducted in the Exercise Physiology Lab in Eppler South. If you are interested in possible participation and want to set up a meeting with the investigators, please feel free to contact Dr. Matt Laurent at cmlaure@bgsu.edu Chris Irvine at cirvine@bgsu.edu for further information. By emailing Dr. Laurent or Chris, you are not agreeing to participate but are simply indicating that you are interested in hearing more about the study and the specific details concerning participation.

APPENDIX E: RECRUITMENT EMAIL

Email/Phone Correspondence

You have indicated you would potentially be interested in participating in a research project being conducted in the Department of Kinesiology. If you were to participate, you would be asked to complete three exercise sessions on a treadmill. The total time involved will be close to an hour to one hour and a half per session (3.25-3.75 hours total) with a one session lasting only 6-10 minutes of exercise depending on how fit you are while the other session will last about 12 minutes of exercise with about 30 minutes of passive rest/recovery. Various measures will be taken such as your heart rate and oxygen consumption which requires that you breathe through a mouthpiece and tube while you exercise. Certain risks are involved with participation including very high heart rates and all pains and discomforts associated with intense physical exertion (these also include cardiovascular problems and death). If you are still interested, please indicate so we can set up a time to meet in the lab and discuss in more detail the requirements and risks associated with participating. By agreeing to an initial meeting you are NOT necessarily agreeing to participate – only to meet for the chance to ask additional questions to determine if you would like to participate. All participation is voluntary, and you are free to decide not to participate or to stop participating at any point. If you would like to set an initial meeting to discuss potential participation, please list/provide some dates and times in the near future that would be convenient for you.

APPENDIX F: ADULT OMNI SCALE OF PERCEIVED EXERTION FOR RUNNING (UTTER ET AL, 2004



OMNI Scale of Perceived Exertion: Adult, Walking to Running Format

From *Perceived Exertion for Practitioners: Rating Effort With the OMNI Picture System* by R.J. Robertson. Champaign, IL: Human Kinetics, 2004.

APPENDIX G: PERCEIVED RECOVERY STATUS SCALE (LAURENT ET AL., 2011)

Perceived Recovery Status Scale

10	Very well recovered / Highly energetic	Expect Optimal Performance
9		
8	Well recovered / Somewhat energetic	
7		
6	Adequately recovered	Expect Average Performance
5		
4	Somewhat recovered	
3		
2	Not well recovered / Somewhat tired	Expect Weak Performance
1		
0	Very poorly recovered / Extremely tired	

APPENDIX H: SESSION RPE (FOSTER ET AL., 2001)

Rating	Descriptor
1	Rest
2	Very, very easy
3	Moderate
4	Somewhat hard
5	Hard
6	-
7	Very hard
8	-
9	-
10	Maximum

APPENDIX I: INCREMENTAL MAXIMAL EXERTION TREADMILL TEST RECORDING SHEET

Participant #:
Date:

Name: _____ Gender:____
Age: _____
Body mass (kg): _____
Height (cm):_____
Body Fat: _____

Room temperature (°C): _____
Barometric Pressure (mm/Hg): _____
Relative humidity (%): _____

Time	**Speed**	**HR**	**VO_2**	**RPE**
0-1	6.2 mph	_____	_____	_____
1-2	6.8 mph	_____	_____	_____
2-3	7.4 mph	_____	_____	_____
3-4	8.0 mph	_____	_____	_____
4-5	8.6 mph	_____	_____	_____
5-6	9.2 mph	_____	_____	_____
6-7	9.8 mph	_____	_____	_____
7-8	10.4 mph	_____	_____	_____
8-9	11.0 mph	_____	_____	_____
9-10	11.6 mph	_____	_____	_____
10-11	12.2 mph	_____	_____	_____
11-12	12.8 mph	_____	_____	_____
12-13	13.4 mph	_____	_____	_____

Time to exhaustion _____ VO_2 peak _____ Peak HR _____

APPENDIX J: SESSION 2 AND 3 RECORDING SHEET

Date:

Participant #:
Protocol:

Set speed (110% vVO_2 max): _____mph

Resting [La]: _____mmol

Set 1:

Sprint #	HR	RPE
1		
2		
3		
4		

PRS:

Minute	
1	
2	
3	

3-min recovery [La]: min 2: _____mmol

Set 2:

Sprint #	HR	RPE
1		
2		
3		
4		

PRS:

Minute	
1	
2	
3	

3-min [La]: min 2: _____mmol

Set 3:

Sprint #	**HR**	**RPE**
1		
2		
3		
4		

PRS:

Minute	
1	
2	
3	

3-min [La]: min 2: _____mmol

Set 4:

Sprint #	HR	RPE
1		
2		
3		
4		

PRS:

Minute	
1	
2	
3	

3-min [La]: min 2: _____mmol

S-RPE: _____