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Examining Work-To-Rest Ratios To Optimize Upper Body Sprint Interval Training

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EXAMINING WORK-TO-REST RATIOS TO OPTIMIZE UPPER BODY SPRINT INTERVAL TRAINING

by

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ABSTRACT

The primary purpose of this study was to compare the metabolic influence of varying work-to-rest ratios during upper body sprint interval training (SIT). Forty-two recreationally trained men were randomized into one of three training groups [10s work bouts with two minutes of rest (10:2, n = 11) or four minutes of rest (10:4, n = 11), or 30s work bouts with four minutes of rest (30:4, n = 10)] or a control group (CON, n = 10). Participants underwent six training sessions over two weeks with four to six 'all-out' sprints. During pre- and post-intervention visits, participants underwent a graded exercise test to determine maximal oxygen consumption ($\dot{V}O_2$peak) and peak power output (PPO), four constant-work rate trials to determine critical power (CP), anaerobic working capacity (W'), and electromyographic fatigue threshold (EMG_{FT}), and an upper body Wingate test to determine peak power (PP), mean power (MP), and total work (TW). Oxygen consumption and blood lactate during the Wingate test generated estimates of oxidative, glycolytic, and ATP-PCr energy system provisions. An analysis of covariance was performed on all testing measurements collected at post with the associated pre-values used as covariates. $\dot{V}O_2$peak was greater in 30:4 ($p = .007$) and 10:2 ($p = .036$) compared to CON and PPO was greater in 30:4 than CON ($p = .007$). No differences were observed between groups in CP ($p = .530$), W' ($p = .900$), EMG_{FT} ($p = .692$), PP ($p = .692$), MP ($p = .290$), or TW ($p = .291$). Relative energy contribution ($p = .026$) and energy expenditure ($p = .019$) of the ATP-PCr energy system was greater in 10:4 compared to CON. SIT protocols with larger work-to-rest ratios induce enhanced aerobic adaptions, whereas smaller work-to-rest ratios may enhance ATP-PCr utilization in the upper body over a short-term two-week intervention.

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CHAPTER ONE: INTRODUCTION

Sprint interval training (SIT) is a popular method of training that incorporates four to six brief, but intense, maximal effort bouts of exercise such as repeated 30 s Wingate tests (Buchheit, Abbiss, Peiffer, & Laursen, 2012; Burgomaster, Heigenhauser, & Gibala, 2006; Franchini, Takito, & Kiss, 2016). As a form of high intensity interval training (HIIT), SIT has led to improvements in aerobic and anaerobic capacity. This type of high intensity training has led to alterations in skeletal muscle oxygenation and deoxygenated hemoglobin/myoglobin values along with increased muscle oxidative capacity and $\dot{V}O_2$peak (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Gillen et al., 2014; Jacobs et al., 2013; Sloth, Sloth, Overgaard, & Dalgas, 2013). In addition, SIT has shown enhanced glycolytic enzyme activity and maximum anaerobic power (Burgomaster et al., 2006; MacDougall et al., 1998). Hazell, MacPherson, Gravelle, and Lemon (2010) highlighted the importance of rapid peak power attainment as the most likely cause for SIT adaptations during lower body cycling. In comparison to the commonly used traditional SIT protocol incorporating 30 s work bouts, the authors observed that 10 s work bouts produced similar increases in $\dot{V}O_2$max and 5-km time trial performance (Hazell et al., 2010). The ability to maintain peak power, rather than maximizing total work done, over a series of 10 s work bouts in the modified SIT protocol was likely responsible for the similar improvements in a more time efficient manner (Hazell et al., 2010). Therefore, the authors concluded that, during lower body cycling, similar aerobic and anaerobic performance enhancements might occur with shorter work bouts (i.e. 10 s vs. 30 s) and overall less time commitment (Hazell et al., 2010).

When training the lower body is not feasible, training the upper limbs can also improve aerobic capacity considerably within a short or moderate period of time (Schoenmakers, Reed, Van Der Woude, & Hettinga, 2016; Zinner et al., 2016). Various sports such as judo, wrestling, kayaking, and cross-country skiing rely on the upper body musculature during training and competition (Garrett & Kirkendall, 2000). For instance, upper body anaerobic performance (mean and peak power) has shown to distinguish between elite and amateur wrestlers (García-Pallarés, López-Gullón, Muriel, Díaz, & Izquierdo, 2011) and has been associated with 50-m freestyle swim performance in males and females ($r = .68 - .89$) (Hawley & Williams, 1991). In addition to athletes, upper body training may be relevant to individuals undergoing rehabilitation and to a variety of special populations (those with limited mobility, spinal cord injuries, overweight/obesity, and aging). Unfortunately, minimal training studies on the upper body musculature have been conducted while specifically utilizing SIT (Vandbakk et al., 2017; Zinner et al., 2016). Since the upper body musculature has a smaller diffusion area and larger diffusion distance, along with greater type two fiber distribution (Calbet, De Paz, Garatachea, Cabeza de Vaca, & Chavarren, 2003; Sanchis-Moysi et al., 2010; Zinner et al., 2016) than the lower body musculature, it has a predisposition to utilize anaerobic resources and therefore, may be more susceptible to aerobic training adaptations. Research is equivocal due to the specificity of energy systems (i.e. two versus three) on whether the upper body relies on a greater relative aerobic contribution than the lower body during Wingate tests (Harvey, Bousson, McLellan, & Lovell, 2015; M. Price et al., 2014). Nonetheless, improvements in aerobic capacity as a result of high-training may be greater for the upper body than the lower body (Price et al., 2014). Zinner and colleagues (2016) noted that the anaerobic predominance in the arms does not appear to limit their ability to increase aerobic capacity in response to SIT. The authors found that two weeks of

SIT increased aerobic energy production more so in the arms then legs via higher $\dot{V}O_2$peak, greater oxygen consumption during a Wingate test along with a lower O_2 deficit, and improved mechanical efficiency (Zinner et al., 2016). Thus, the efficacy of a modified (<30 s work bouts) short duration SIT intervention in the upper body, as employed by Hazell and colleagues (2010) in the lower body, is warranted.

Since the upper body has the potential for aerobic and anaerobic adaptations, parameters from the work-time relationship may provide insight into the unique adaptations involved with altering the work-to-rest ratio during upper body SIT. These parameters may be determined using multiple high-intensity, exhaustive exercise bouts yielding estimates of critical power (CP), defined as the highest attainable intensity that can be maintained without fatigue, and anaerobic working capacity (W'), defined as the finite work capacity that can be performed above CP (Poole, Ward, Gardner, & Whipp, 1988). In particular, CP is associated with aerobic function (A. M. Jones, Vanhatalo, Burnley, Morton, & Poole, 2010) while W' is associated with anaerobic metabolism and the $\dot{V}O_2$ slow component (Monod & Scherrer, 1965; Vanhatalo, Poole, DiMenna, Bailey, & Jones, 2011). Indeed, endurance training interventions have been shown to improve CP (Jenkins & Quigley, 1992; Poole, Ward, & Whipp, 1990) and reduce the amplitude of the $\dot{V}O_2$ slow component without any effect on W' (Jones & Carter, 2000; Poole et al., 1990), while high-intensity exercise has shown to improve W' without any change in CP (Jenkins & Quigley, 1993). While the assessment of work-time relationship in the upper body has been evaluated (Belasco Junior, Oliveira, Serafini, & Silva, 2010; Capodaglio & Bazzini, 1996; Fukuda et al., 2013; Taylor & Batterham, 2002; Yang, Lee, Hsu, & Chan, 2017) and CP has been determined to be a valid assessment tool for upper body endurance (Belasco Junior et al., 2010), the potential impact of a SIT intervention has yet to be explored.

Similar to CP derived from the work-time relationship, the electromyographic fatigue threshold (EMG_{FT}) is considered the highest power output that can be achieved without an increase in EMG amplitude over time (Moritani, Takaishi, & Matsumoto, 1993). Electromyographic fatigue threshold can be been estimated using a similar method of assessment as CP (Devries, Moritani, Nagata, & Magnussen, 1982) and may also be used to demarcate between exercise intensity domains (Camic et al., 2010; Poole et al., 1988). Electromyographic fatigue threshold occurs when there is a progressive recruitment of additional motor units or an increase in firing frequency of previously fatigued motor units in order to compensate for the deficit in fatigued motor units (Moritani et al., 1993). In response to cycling exercise, HIIT can cause a delay in neuromuscular fatigue within a short period (3-6 weeks) (Smith et al., 2009). In addition, increases in muscle fiber recruitment [i.e. elevated root mean square (RMS) values] and a decrease in mean frequency have been found after four weeks of cycling SIT (Creer, Ricard, Conlee, Hoyt, & Parcell, 2004). Both of these findings would imply greater synchronization and force potentiation that can improve efficiency and coordination, thereby delaying fatigue. While a considerable amount of research has focused on EMG_{FT} during lower body cycling, its application in the upper body is lacking and may provide insight into the unique adaptations resulting from altered work-to-rest ratios during SIT.

Short-term training and interval sprint training results in rapid blood flow kinetics (Laughlin & Roseguini, 2008; Shoemaker, Phillips, Green, & Hughson, 1996) due to the demand of oxygenated hemoglobin from working muscle and the recovery of deoxygenated hemoglobin. Oxygen uptake kinetics has been shown to be accelerated and associated with greater muscle oxygen extraction capacity in response to short-term SIT (Jones, Hamilton, & Cooper, 2015). At the onset of exercise, there is an increase in blood volume to the working muscle carrying

oxygenated hemoglobin (Hb) to meet the energy demands of the activity. As exercise continues to intensify, such as a ramp incremental graded exercise protocol, oxygenated hemoglobin tends to drop off and deoxygenated Hb tends to rise (Boone, Vandekerckhove, Coomans, Prieur, & Bourgois, 2016). This rise is a natural consequence of oxygen being extracted from the red blood cells into the muscle's mitochondria as per the oxygen-dissociation curve, which continues until there is a plateau due to limits imposed by oxygen extraction and utilization (Boone et al., 2016). This plateau is synonymous with deoxygenation threshold or break point $[HHb]_{BP}$ which delineates the point at which maximal oxygen extraction, by the working muscle, has been achieved (Boone et al., 2016). Continued exercise following $[HHb]_{BP}$ in a ramp protocol elicits exhaustion within minutes as the supply cannot keep up with the demand. Therefore, the deoxygenation curve is often represented by a sigmoidal curve with the top portion signifying $[HHb]_{BP}$ at the beginning of the plateau (Boone et al., 2016). This threshold potentially represents type I muscle fiber fatigue and, during continued physical exertion, yields subsequent reliance on type II muscle fibers, which are much more limited in their energy storage capabilities (Boone et al., 2016). Along with the recruitment of type II muscle fibers, increases in lactate and other metabolic by-products (e.g. hydrogen ions, inorganic phosphate) can be observed similar to exercise above CP (Burnley & Jones, 2018). Since $[HHb]_{BP}$ may differentiate between oxidative (type I) and glycolytic (type II) fibers, and is strongly related to $\dot{V}O_2max$ (Boone et al., 2016), a training program used to increase aerobic capacity should allow for a rightward shift in the deoxygenated Hb signal allowing for greater deoxygenation at the same relative intensity. Given that SIT has beneficial adaptations to both aerobic and anaerobic capacities, there may be a rightward shift in the deoxygenation Hb curve allowing more work to be done before fatigue. In fact, SIT has shown to increase deoxygenated hemoglobin and

myoglobin amplitudes, which increases muscle extraction capacity (B. Jones et al., 2015). Furthermore, a two-week SIT program stimulated a 15% improvement in capillary density within the triceps brachii, showing that oxygen extraction may be the mechanism behind aerobic improvements (Zinner et al., 2016). However, McKay and colleagues (2009) showed that a faster rate in muscle O_2 utilization was not accompanied by an increase in muscle O_2 extraction in the vastus lateralis (reported as no change in deoxygenated hemoglobin) in response to high intensity training and endurance training despite faster VO_2 kinetics. Therefore, systemic and peripheral adaptations underlying the improvements in aerobic capacity need further examination.

Previous research has shown CP and deoxygenated hemoglobin $[HHb]_{BP}$ thresholds are interrelated due to their potential physiological equivalence (Keir et al., 2015). Meanwhile, the progressive recruitment of motor units, along with reduced contractile efficiency and the oxygen cost of recovering fatigued fibers, is associated with the development of the $\dot{V}O_2$ slow component which corresponds to W' (Vanhatalo et al., 2011). Despite the limited direct comparisons between thresholds, CP, $[HHb]_{BP}$, and EMG_{FT} tend to theoretically define the same boundaries and have been strongly correlated with one another (Boone et al., 2016). Although some investigations have concluded significant differences between them, all breakpoints and boundaries appear to occur within a narrow range (~76-88% $\dot{V}O_2$max) of intensities (Boone, Barstow, Celie, Prieur, & Bourgois, 2015). As a result, the breakpoints in oxygenation and fatigue thresholds may be interrelated and mechanistically linked (Boone et al., 2015). Consequently, the precise order of appearance among boundaries is equivocal within the literature. During a continuous ramp protocol at intensities above CP there will be an increase in additional motor units (most likely type II fiber recruitment), stimulated by loss of contractile

function, and a need to maintain or increase power output. In turn, this results in an increased RMS amplitude, decreased pH levels and increased pCO_2, followed by increased minute ventilation leading to a higher oxygen cost of breathing which adds to the amplitude of the $\dot{V}O_2$ slow component. Concomitantly respiratory fatigue may ensue compromising blood flow to the working muscles and limiting oxygen delivery causing maximal oxygen extraction and utilization (Burnley & Jones, 2016).

Sprint interval training with work bouts of less than 30 s may alter the oxygen kinetics and in turn, relative energy contributions. Total energy supply derived from the aerobic energy system is greater during longer durations of maximal exercise (Gastin, 2001). For example, the estimated relative anaerobic and aerobic contribution for a 10 s maximal sprint is 94% and 6%, respectively, versus 73% and 27%, respectively, for a 30 s maximal sprint (Gastin, 2001). For a single Wingate bout, Lovell and colleagues (2013) estimated the relative energy contributions for the upper body to be 11.4% oxidative, 60.3% glycolytic, and 28.3% ATP-PCr. Meanwhile, only two studies have compared these contributions within the same group of participants during a Wingate bout for both upper and lower body (Harvey et al., 2015; M. Price et al., 2014). Both Harvey and colleagues (2015) and Price and colleagues (2014) show conflicting reports on the differences between upper and lower body relative energy provisions which may be explained by the differences in Wingate loads utilized for the upper body and the differences in the authors breakdown of energy systems (three systems versus two systems). More recently, Franchini and colleagues (2016) reported that the glycolytic system had the greatest drop in energy provision over the course of four repeated upper body Wingate bouts in well-trained Judo athletes. In fact, the fourth and last Wingate bout had a greater reliance on the oxidative and ATP-PCr systems rather than glycolytic contributions (Franchini et al., 2016). Regardless of the work bout

duration, sprint trained individuals appear to utilize their energy systems differently than endurance trained individuals (Gastin, 2001), thus varying work-to-rest ratios may alter the specific proportion of energy system contributions following SIT.

The primary objective of this study was to evaluate the effectiveness of upper body SIT protocols with varying work-to-rest ratios on both aerobic and anaerobic performance. The secondary objective was to investigate the changes in metabolic and neuromuscular fatigue thresholds from two weeks of SIT in recreationally active men. The third objective was to examine the influence of SIT on energy system utilization during a maximal anaerobic task.

Purpose

1. To evaluate the effectiveness of sprint interval training protocols with varying work to rest ratios on the upper body in both aerobic and anaerobic performance.
2. To investigate the changes in metabolic and neuromuscular fatigue thresholds from two weeks of sprint interval training in recreationally active men.
3. To examine the influence of sprint interval training protocols on relative energy system utilization.

Research Questions

1. Will sprint interval training protocols with varying work to rest ratios increase aerobic and anaerobic performance to the same extent?
2. Will sprint interval training protocols with varying work to rest ratios increase metabolic and neuromuscular fatigue thresholds after two weeks of sprint interval training in recreationally active men?

3. Will sprint interval training protocols with varying work to rest ratios alter the relative energy contributions during a 30-second anaerobic task?

Hypotheses

1. Both aerobic and anaerobic performance will increase from sprint interval training with longer rest periods.
2. Anaerobic performance will increase to a greater extent than aerobic performance after two weeks of sprint interval training.
3. Both metabolic and neuromuscular thresholds will be delayed after two weeks of sprint interval training.
4. Sprint interval training will lead to an increase in muscle oxygen extraction capacity.
5. Sprint interval training will lead to a greater efficiency in glycolytic energy provision.

CHAPTER TWO: REVIEW OF LITERATURE

Sprint Interval Training

Burgomaster, Hughes, Heigenhauser, Bradwell, Gibala, 2005

Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans

The aim of this study was to examine the aerobic effects of six sessions of SIT. Sixteen recreationally active individuals were randomly assigned to a sprint interval training intervention or a control group that only participated in testing. Testing was completed before and after the two-week, six-session training protocol. All participants underwent a $\dot{V}O_2$peak test, to determine aerobic capacity, and a cycle endurance capacity test (at ~80%VO_2peak), to determine time-to-exhaustion, on a cycle ergometer. In addition, muscle biopsies from the vastus lateralis were taken in the training group only to evaluate muscle oxidative potential [citrate synthase, ATP, phosphocreatine (PCr), creatine, and glycogen]. The training protocol consisted of repeated 30 s all-out sprints, with four minutes of rest, against a load equivalent to 7.5% of their body weight (in kg) three times per week for six sessions across two weeks. The number of repetitions increased from four sprints on day one, to five on day two, to six on days three and four, and to seven on day five. On the last day, participants only performed four sprints. Peak power, mean power, and fatigue index were determined during training.

The training group increased 100% during the cycle endurance capacity test, while the control group showed no change, however VO_2peak did not change in either group. Peak power increased, but so did fatigue index from the first training session to the last training session with no change in mean power over the four sprints. Maximal citrate synthase activity increased by

38% and glycogen concentrations increased by 26% in the training group, but no changes were observed in ATP, PCr, or creatine. In summary, this investigation showed that a small training volume of intense exercise (~15min) over two weeks positively influenced the metabolic profile of skeletal muscle and endurance capacity in recreationally active individuals.

Burgomaster, Heigenhauser, Gibala, 2006

Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance

The purpose of this research was to examine the effects of a two-week sprint interval training (SIT) intervention on muscle carbohydrate metabolism during submaximal, time-trial exercise. Sixteen recreationally active men were equally divided into two groups (a training group or a control group) for two weeks. First, participants completed a $\dot{V}O_2$peak test to determine aerobic capacity and to prescribe subsequent workloads used during the main experimental trials. Second, participants completed a time trial (10km) exercise test to establish time-to-completion and average peak power on a cycle ergometer. Third, participants underwent a Wingate test to determine peak power, mean power, and fatigue index. Lastly, participants in the training group only underwent an invasive metabolism test which included submaximal cycling test at 60% and 90% $\dot{V}O_2$peak interspersed with muscle biopsies from the vastus lateralis to determine the amount of pyruvate dehydrogenase (PDH), 3-hydroxyacyl-CoA (HAD), and citrate synthase (CS) activity along with lactate, ATP, PCr, creatine, and glycogen concentrations. The training intervention consisted of six sessions of SIT over two weeks consisting of 30 s all-out efforts with four minutes of recovery. The number of sprints were

increased from four to seven over the first five training sessions until the last day were only four sprints were performed.

Time-trial performance was significantly shorter in the training group as shown by a decrease in time-to-completion (9.6%) and an increase in average peak power, whereas the control group remained the same. Likewise, peak (5.4%) and mean power (8.7%) in the Wingate test were also significantly increased while the fatigue index was reduced (17.9%) by the training intervention. In contrast, the control group had no change. The training intervention also induced increases in maximal activity of CS and PDH, but no change in HAD. In conjunction, increases in muscle glycogen content were noted and net muscle glycogenolysis was reduced post training. Blood lactate was lower and net lactate accumulation was attenuated post training. There were lower contents of creatine and ATP, but unchanged PCr following training. Aerobic capacity did not change from pre to post. In conclusion, six sessions of SIT reduced muscle glycogenolysis and lactate accumulation during submaximal exercise while increasing PDH activity. As a result, time trial performance improved despite no change in $\dot{V}O_2$peak.

Hazell, MacPherson, Gravelle, Lemon 2010

<u>10 or 30-s sprint interval training bouts enhance both aerobic and anaerobic performance</u>

The purpose of this investigation was to examine a traditional sprint interval protocol consisting of 30 s of work and 4 min of recovery (30:4) with modified versions consisting of 10 s of work and either 4 (10:4) or 2 min (10:2) of recovery. The investigation recruited 48 recreationally active young adults that were not involved in a specific training program currently or four months prior. Participants underwent pre and post testing surrounding two weeks of

sprint interval training on an electronically braked cycle ergometer. Testing consisted of body composition via BodPod to analyze percent body fat, anaerobic capacity via Wingate test to determine peak and mean power, aerobic capacity to determine $\dot{V}O_2$max, and aerobic performance via 5-km time trial performance. The training consisted of six sessions spread over two weeks. Participants were split into one of three training groups of all-out efforts with either (30:4), (10:4), (10:2), or a control group. Their recovery consisted of active unloaded cycling rather than the work intensity of 10% of the participant's body weight (in kg). Each training session was separated by 48-72 hours while training was progressed one repetition every two sessions so that the first two sessions included four repetitions, the middle two sessions included five repetitions, and the last two sessions included six repetitions. The authors examined the reproducibility of power (peak, mean, and minimum) within each training session relative to their highest respective power measure from the training intervention. Additionally total work was calculated during each training session.

All groups, except for the control group, significantly improved in 5-km time trial performance; however, no differences were noted between groups. There were no significant differences between groups in $\dot{V}O_2$max values. The 30:4 group significantly increased 9.3%, the 10:4 min groups significantly increased 9.2%, while the control group showed no change. Although, the 10:2 group increased 3.8% it was not significantly different from pre ($p = 0.06$). There was a significant interaction in relative power output via 30 s Wingate test. The 30:4 group significantly increased 9.5%, the 10:4 group increased 8.5%, and the 10:2 improved 4.2% while the control group showed no change. A similar interaction occurred in mean power output. The 30:4 group improved 12.1%, the 10:4 group increased 6.5%, while no change was noted in the 10:2 (2.9% improvement) or control group. In regards to maintaining power during

the training sessions, the 10 s:4 min group was able to maintain 96% while the 10 s:2 min group was able to maintain 95% of their peak power output, both resulting in significant increases over the 30:4 group (89% of their peak power output). Likewise, there were significant differences in the ability to maintain average power output during training sessions. The 10:4 group and 10:2 group were able to maintain 84% and 82%, respectively, of their highest mean power output, which were both significantly greater than the 30:4 group (58%). The 10:4 group and 10:2 group were able to attain a greater minimum power output (73% and 69% of their highest minimum power output, respectively), than the 30:4 group (40%). Lastly, no body composition changes were observed and there was significantly greater work performed in the 30 s group compared to both 10 s groups, while there was no difference between the 10 s groups.

The results demonstrated that both modified sprint interval training protocols of 10 s work bouts produced similar aerobic improvements compared to the established 30 s work bout. The 10 s groups performed ~50% of the work completed in the 30 s group and in 33% of the training time, therefore it is a very time efficient protocol. The peak power output and the mean power output obtained were significantly higher in the 10 s group suggesting that the generation of peak power is likely responsible for the sprint interval training adaptations.

Zinner, Morales-Alamo, Ortenblad, Larsen, Schiffer, Willis, Gelaber-Robato, Perez-Valera, Bouchel, Calbet, Holmberg, 2016

<u>The physiological mechanisms of performance enhancement with sprint interval training differ between the upper and lower extremities in humans</u>

The aim of this study was to determine whether the arms and legs adapt differently to the same short-term sprint interval training program. This investigation recruited 16 healthy

recreationally trained men. Participants underwent pre- and post-testing surrounding six training sessions of the upper and lower body. Testing consisted of limb specific aerobic capacity, via $\dot{V}O_2$peak, and 30 s Wingate tests with four minutes of recovery to determine mean power output, peak power output, and fatigue index, a four by four-minute submaximal incremental exercise test to determine work efficiency, and a five-minute all-out time trial. Additionally, muscle biopsies were taken from the vastus lateralis and the triceps brachii six days before training and six days after training. Participants underwent sprint interval training for six training days over two weeks. Each training day consisted of one arm and one leg cycling sessions separated by one hour. Half the participants trained upper body first while the other half trained the lower body first. Both sessions consisted of four to six 30 s all-out sprints (Wingate test with 7.5 bodyweight in kg) with four minutes of unloaded pedaling recovery. Progression was implemented by increasing one repetition on both sessions every two training days.

The maximal ventilatory response to leg exercise was 24% greater than the arms, but the relative VO_2 and VCO_2 to V_E was similar. Blood lactate was significantly greater in the legs than the arms after the training sessions (14.8 ± 2.4 mmol/L vs. 12.6 ± 2.3 mmol/L, respectfully). Training did not have an effect on body composition as determined by DEXA scan. Aerobic capacity was increased in the upper (9.8%) and lower (6.1%) body. The gross efficiency for the arms was significantly lower than the legs as determined by the submaximal incremental exercise test, however the arms had a training efficiency improvement of ~9% where the legs did not. A more pronounced peak power, relative $\dot{V}O_2$peak to peak power during the graded exercise test, and relative $\dot{V}O_2$peak to limb lean mass were noted for the arms. Greater improvements in mean power output (14.5 vs. 13.9%) and mean VO_2 (11.4 vs. 7.9%) were noted in the arm as compared to the legs, respectfully. Likewise, peak (10 vs. 5%) and mean power (7

vs. 5%) and output in the Wingate test improved higher for the arms then the legs, respectfully. There were no differences in muscle fiber composition between the upper and lower extremities and no adaptations occurred post training. The upper body had significantly greater myosin heavy chain type II fibers compared to type I, but no changes in the upper body were observed. The upper body was able to significantly improve capillary density per fiber, but not alter aerobic enzymes or glycogen content. Although the upper body had greater proportion of type II fibers, it did not limit its capacity to increase $\dot{V}O_2$peak in this sprint protocol.

This was the first investigation examining the impact of sprint interval training on the arms. The arms improved VO_2peak to a greater extent than the legs which could be explained by an enhancement of oxygen extraction given increased capillarization and no change in blood lactate or glycogen content before and after training. Unfortunately, blood flow and oxygen delivery were not measured.

Nalçakan, Songsorn, Fitzpatrick, Yüzbasioglu, Brick, Metcalfe, Vollaard, 2017

<u>Decreasing sprint duration from 20 to 10 s during reduced-exertion high-intensity interval training (REHIT) attenuates the increase in maximal aerobic capacity but has no effect on affective and perceptual responses</u>

This study offered a way to achieve the same aerobic capacity benefits of traditional sprint interval training (4-6 reps of 10 s or 30 s) with reduced-exertion high-intensity interval training. The authors offer an alternative hypothesis to the unclear investigations that activate a series of signaling pathways, but rather increases in $\dot{V}O_2$max stem from relations with rapid glycogenolysis. The aim of the study was to determine how two reduced exertion high intensity

training protocols with either 20 s or 10 s sprints affect changes in $\dot{V}O_2$max, ratings of perceived exertion, and changes in mood state.

Thirty-six sedentary and recreationally active men and women were randomized into two groups undergoing either 10 s or 20s sprints for six weeks (three days per week) on a bike with a resistance of 7.5% their bodyweight. Subjects performed graded exercise tests to establish $\dot{V}O_2$max before and after their six-week training session of only two sprints per session. Both training groups worked up to 20 s and 10 s intervals over the 18 training sessions with three to four minutes of rest in-between sprints. Ratings of perceived exertion were measured at baseline and immediately post-exercise every third session while the mood scale was measured before and five minutes after the second sprint for each training session.

The 20 s group experienced a greater increase in $\dot{V}O_2$max (+10%) versus the 10 s group (+4%) from pre- to post-testing. Meanwhile there were no significant differences between groups or training sessions in ratings of perceived exertion. In addition, there were no increases in negative outlook or mood disturbances directly after exercise between groups. Therefore, the authors suggest the use of 20 s sprints as a time-efficient alternative to the traditional 30 s sprints for improving aerobic capacity without influencing perceived exertion, acute negative responses or mood states.

Critical Power and Upper Body Arm Cranking

Capodaglio and Bazzini, 1996

Predicting endurance limits in arm cranking exercise with a subjectively based method

The purpose of this study was to validate and predict endurance capacity using subjective perception of effort during arm cranking at different workloads. Three trained paraplegic subjects performed a maximal incremental test to determine the power output at ratings of perceived effort (using the 10-point Borg scale) corresponding to moderate, somewhat heavy, and heavy effort. Those ratings of perceived effort were then utilized to prescribe three different workloads for the subjects to perform nine, 15-min endurance tests, which ultimately constructed 'iso-perception' curves. In addition, subjects performed three time-to-exhaustion trials at constant loads (e.g. 50 W, 37.5 W, and 25 W) to determine CP using methods previously established. The subjective measures were validated against the previously established work-time model. The CP value in each subject corresponded to the inferior asymptote of the hyperbolic power output versus time curve as illustrated by the 'iso-perception' curves. Therefore, CP could be calculated using the work-time model or by subjective measures using the hyperbolic power-duration curve.

Miura, Sato, Sato, Whipps, Fukuba, 2000

The effect of glycogen depletion on the curvature constant parameter of the power-duration curve for cycle ergometry

The aim of this investigation was to examine the physiological basis of W' with the manipulation of muscle glycogen states. Seven healthy men performed an incremental exercise

test to determine $\dot{V}O_2$max and four different high-intensity constant load trials to exhaustion under both normal (NG) and glycogen depleted (GD) states to determine CP and W' on a cycle ergometer. Trials were designed to elicit fatigue within two to 10 min. In order to ensure a glycogen depleted state subjects performed cycling the evening prior at 60% $\dot{V}O_2$max for 75 min, followed by one-minute cycling bouts at 115% $\dot{V}O_2$max until they could no longer maintain a pedaling rate above 50 RPM, and then fasted overnight in order to perform a single high intensity exercise test the following morning. On the other hand, the NG group were instructed not to perform heavy exercise the day prior.

There was no significant differences in CP between GD and NG, however W' was significantly reduced in the GD condition. In response, the mean RER values during the 15-min warm-up prior to high-intensity exercise was significantly lower in the GD state than the NG state (0.84 ± 0.02 vs. 0.94 ± 0.04, respectfully) showing that glycogen stores were likely reduced due to the glycogen depletion protocol. During high-intensity exercise peak V_E and VCO_2 were significantly reduced in the GD state; however, VO_2 and HR were not significantly altered between conditions. This suggests that all participants achieved maximal effort. As a result, muscle glycogen appears to be a significant determinant for W' in the hyperbolic relationship between power and time.

Taylor and Batterham, 2002

<u>The reproducibility of estimates of critical power and anaerobic work capacity in upper-body exercise</u>

The purpose of this investigation was to examine the parameters of CP and AWC and their reliability during upper body exercise, using the linear relationship between power and the

inverse of time. Sixteen active men performed two staged incremental exercise test to determine VO_2peak using a cycle ergometer adapted for upper body use. Subsequently, participants underwent two sets of four constant-power exercise bouts order at 85%, 100%, 105%, and 115% of the power output associated with VO_2peak in random order on separate days. A fifth bout was administered to evenly spread the times to exhaustion between one and 10 min. There was no significant systematic bias between either set of constant-power trials and time-to-exhaustion, predicted AWC, or predicted CP. Critical power equated to 96 ± 16 W (73 ± 7% PPO) and 94 ± 17 W (72 ± 8% PPO), in the first and second set of constant-power trials. Critical power had a higher test-retest correlation (r^2 = .881) compared with AWC (r^2 = .358). Based on the 95% limits of agreement, repeated measures of CP may range from 0.64 to 1.59 times the baseline measurement, while AWC may range from 0.57 to 1.67 times the baseline measurement. Therefore, despite the absence of systematic bias and high test-retest correlation of CP, the potential variation in the parameter estimates of CP and AWC suggest poor reliability under the power and inverse of time model.

Belasco Junior, Oliveira, Serafini, Silva 2010

<u>Determination of the power-duration relationship in upper-limb exercises</u>

The purpose of this investigation was to explore the hyperbolic relationship of critical power (CP) in the upper limbs and investigate its relationship with aerobic parameters. Ten physically active men performed a graded exercise test to attain $\dot{V}O_2$peak, ventilatory threshold (VT1) and respiratory compensation point (VT2). Participants then underwent five constant load tests, at 70, 80, 90, 95, and 100% of the difference between the load at VT1 and $\dot{V}O_2$peak, to determine CP and anaerobic working capacity (W'). One constant load test was conducted per

day and designed to elicit exhaustion between one and 20 minutes. Lastly, each participant performed a constant load trial at CP and at 5% above CP.

The work-time relationship was found to be hyperbolic and linearized by taking the inverse of time in all participants (r = 0.94 – 1.00). The $\dot{V}O_2$ at CP (2.66 ± 0.62 L/min) was significantly greater than the $\dot{V}O_2$ at VT1 (1.62 ± 0.38 L/min) and VT2 (2.36 ± 0.59 L/min), however no differences in load between CP (103.0 ± 26.0 W) and VT2 (103.5 ± 30.8 W) were noted. The $\dot{V}O_2$ and load at CP were associated with the corresponding $\dot{V}O_2$ and loads at $\dot{V}O_2$max, VT1, and VT2. All participants fatigued, on average, at 42.9 min during the CP load trial, but reached fatigue within ~12 – 16 min in the 5% above CP trial. The above CP trial elicited exhaustion in substantially less than the CP trial illustrating the validity of estimating CP in the upper limbs. Additionally, $\dot{V}O_2$ during the CP trial stabilized and corresponded to a respiratory exchange ratio value of 0.98 ± 0.02. Critical Power indicated that it is predominantly aerobic in nature by correlating with VT1, VT2, and $\dot{V}O_2$max, whereas W' did not correlate to any of the aerobic parameters suggesting separate metabolic divisions.

Energy Systems

Lovell, Kerr, Wiegand, Solomon, Harvey, McLellan, 2013

The contribution of energy systems during the upper body Wingate anaerobic test

The purpose of this study was to measure the oxidative, ATP-PCr, and glycolytic energy systems during an upper body Wingate test. Secondarily, it was to assess correlations between active musculature and energy contribution. Fourteen physically active men reported to the lab on three separate occasions which included, familiarization, body composition (via DEXA) to

determine upper body lean muscle mass, and an upper body Wingate test to determine peak and mean power. Additionally, oxygen consumption was measured five minutes prior through 20 min post Wingate test and blood lactate was measured at rest, immediately post, and every two minutes until 20 min post. Energy system contribution was measured via VO_2 consumed above rest (aerobic), an energy equivalent of 3 mL $O_2 \cdot kg^{-1}$ for every 1 $mmol \cdot L^{-1}$ of blood lactate accumulation (anaerobic lactic), and the fast component of excess post oxygen consumption (EPOC) using a bi-exponential four parameter model (anaerobic lactic). The anaerobic alactic energy system was significantly correlated with peak (r = 0.71) and mean (r = .83) Wingate power. Likewise, whereas the anaerobic lactic energy system was significantly correlated to peak (r = 0.56) and mean (r = .61) Wingate power. No correlations existed with the aerobic energy system. Arm lean muscle mass significantly correlated with peak (r = 0.93) and mean (r = .88) Wingate power. In regards to energy contribution, the anaerobic lactic system contributed 60.3%, the anaerobic alactic system contributed approximately 28.3%, and the aerobic system contributed 11.4%. Arm lean body mass was a significant predictor of peak power accounting for 84% of the variability, whereas body mass and arm lean body mass were significant predictors of mean power accounting for 85% of the variability. The upper body has a greater reliance on the anaerobic lactic system and a lower reliance on the aerobic system than lower body Wingate tests reported in previous literature. The anaerobic lactic system also provided strong correlations to peak and mean power.

Freese, Gist, Cureton, 2013

Physiological responses to an acute bout of sprint interval cycling

The objective of this investigation was to examine the VO_2 and cardiorespiratory responses during four consecutive 30 s all-out sprints. Twelve recreationally trained men women performed two acute bouts of sprint interval cycling approximately one week from each other. Each cycling session consisted of four 30 s all-out sprints with four minutes of active recovery. Oxygen uptake, heart rate, ventilation, fatigue, and work performed were used to analyze the time-efficiency of the training modality.

Average work significantly decreased and fatigue rate significantly increased across the four sprints. In particular, fatigue rate was significantly increased from sprint two to sprint three. Aerobic metabolism significantly increased from sprint one to two due to an increase in VO_2, however no further increase was observed in the following third or fourth sprint. The peak oxygen consumption and heart rate throughout the session was observed at the end of sprints two through four, whereas RER progressively decreased across each sprint and within each sprint. The results imply that after the second sprint subsequent sprints are predominantly aerobic. Sprint interval cycling proved to be a time-efficient modality to stimulate aerobic adaptations to exercise.

Price, Beckford, Dorricott, Hill, Kershaw, Singh, Thornton, 2014

Oxygen uptake during upper body and lower body Wingate anaerobic tests

The objective of this investigation was to determine the aerobic contribution to an upper body and lower body Wingate anaerobic test within the same participants. Eight recreationally

trained men performed an upper body and lower body graded exercise test to determine $\dot{V}O_2$ and peak power output. Subsequently, participants performed two upper body and two lower body Wingate anaerobic tests with each test separated by a week. The first Wingate test for each body section served as a familiarization trial. The resistance used for the lower body Wingate test was 7.5% of body weight, in kg, whereas the upper body resistance was 4.0% of body weight, in kg. During each Wingate test, fatigue index, peak, mean, and minimum power were calculated. Oxygen uptake was recorded from five minutes of rest, during the 10 min warm up, during the 30 s test, and for the first five minutes of recovery. Blood samples were taken prior to exercise, immediately after exercise, and five minutes post exercise to determine pH, blood lactate, and bicarbonate (HCO_3-) concentration.

Except for fatigue index, all performance variables were greater in the lower body. No differences were observed in blood lactate, pH, or HCO_3- between upper and lower body Wingate tests. Relative oxygen consumption was greater for upper body (71.1 ± 25.0% $\dot{V}O_2$peak) than lower body (63.9 ± 18.8% $\dot{V}O_2$peak) throughout the 30 s Wingate test, but the greater consumption was from five seconds into the test to five seconds post exercise (i.e. 5 – 35 s) for both upper and lower body. No differences in HR or RPE were noted between upper and lower body Wingates. The oxidative contribution in the upper body (43.5 ± 29.3% and 44.2 ± 22.4%) was greater than the lower body (29.4 ± 15.8% and 32.1 ± 15.2%) during the Wingate test (0 – 30 s) and during the 5 – 35 s segment of collection, respectively. Alternatively, the glycolytic contribution for the lower body (68.3 ± 11.8%) was greater than the upper body (39.3 ± 7.3%). Thus, the upper body had a greater oxidative contribution, but lower glycolytic contribution than the lower body during a single Wingate test when using the same participants.

Franchini, Takito, Kiss, 2016

Performance and energy systems contributions during upper-body sprint interval exercise

The aim of this study was to investigate the performance and energy systems contribution during four upper-body Wingate tests interspersed by 3-min intervals. Fourteen well-trained male Judo athletes performed four Wingate bouts on a cycle ergometer adjusted for the upper body using 5% of the athlete's weight (in kg) as the load. Oxygen uptake and heart rate were measured continuously during exercise and rest intervals including three minutes after the last Wingate test, whereas lactate was measured before and one minute after each Wingate test. Participants remained seated during each recovery period. Peak and mean power were recorded for each Wingate test. Energy system contributions were carried out using oxygen uptake, blood lactate concentrations, and the fast phase of excess post oxygen consumption. Energy system estimates were calculated using a bi-exponential function and the sum of all energy systems was calculated as total metabolic work.

Peak and mean power progressively decreased during each Wingate, while blood lactate progressively increased after each Wingate test. $\dot{V}O_2$peak and peak heart rate did not change throughout the Wingate tests, but progressively increased during each successive test. Over the course of four Wingate tests, the ATP-PCr and oxidative systems maintained their energy output. An interaction for absolute energy expenditure was observed. There was higher glycolytic compared to oxidative and ATP-PCr during the first bout, but a lower glycolytic contribution compared to ATP-PCr during the third bout and a lower glycolytic contribution compared to oxidative and ATP-PCr during the last bout. Over the course of four bouts, glycolytic contribution was the only energy system to progressively decline with higher contribution in the

first two bouts compared to the last two bouts. An interaction for relative energy expenditure was observed. There was higher glycolytic contribution compared to oxidative contribution during the first bout, but lower glycolytic contribution compared to ATP-PCr contribution during the third bout. Also, lower glycolytic contribution compared to oxidative and ATP-PCr contributions for the last bout and lower oxidative contribution compared to ATP-PCr contribution during the last bout. Across the bouts, ATP-PCr contribution was greater in the last bout than the first bout. Likewise, the glycolytic contribution progressively decreased with the first bout being significantly greater than the third and fourth bouts and higher in the second bout than the fourth bout. The relative percent energy contributions are show below.

Table 1. Relative energy system contribution across repeated upper body Wingates

Bout	Oxidative	Glycolytic	ATP-PCr
1	21 ± 10%	46 ± 12%	34 ± 9%
2	28 ± 9%	35 ± 8%	37 ± 8%
3	35 ± 12%	21 ± 6%	44 ± 13%
4	35 ± 15%	12 ± 6%	53 ± 15%

Lastly, mean power was significantly correlated with glycolytic contribution ($r = 0.64$) and total energy expenditure ($r = 0.71$), while peak power was significantly correlated to absolute glycolytic contribution ($r = 0.51$) and total energy expenditure ($r = 0.62$). Absolute oxidative and ATP-PCr contributions remain constant across all bouts; however, there was an increase of relative ATP-PCr contribution in the last bout compared to the first bout most likely due to the partial PCr resynthesis during recovery periods as opposed to decreased glycolytic activity.

Fatigue Thresholds

Vanhatalo, Poole, DiMenna, Bailey, Jones, 2011

Muscle fiber recruitment and the slow component of O2 uptake: constant work rate vs. all-out sprint exercise

The purpose of this study was to investigate the mechanistic response of the $\dot{V}O_2$ slow component during a three-minute all-out cycle exercise test. Eight habitually active men were required to conduct cycling tests over four visits. The first test was a ramp incremental test to assess $\dot{V}O_2$max and gas exchange threshold (GET), two three-minute all-out cycle efforts (one being a familiarization trial) to assess critical power (CP) and anaerobic working capacity (W'), and one constant work rate (CWR) test with a workload estimated to ensure fatigue in three minutes. Gas exchange, lactate, and neuromuscular activity, via integrated electromyography (iEMG), of the vastus lateralis were measured. Time to attain $\dot{V}O_2$max and the $\dot{V}O_2$ slow component were calculated during the all-out CP and CWR tests. The $\dot{V}O_2$max did not differ in the CP and CWR tests, but the slow component development was significantly greater in the CP than in the CWR test. Significant positive correlations were observed in the $\dot{V}O_2$ slow component and W' from the all-out CP test (r = .87) and from the CWR test (r = .83). The peak iEMG during the CP test was observed initially and then steadily declined, whereas the iEMG increased throughout the CWR test with the peak occurring at the point of fatigue. Alternatively, the mean iEMG over the initial 30 s of the CP test was greater than in the CWR test. However, the iEMG relative to workload steadily increased in the CWR test whereas the iEMG steadily decreased in the CP test. The authors concluded that a substantial $\dot{V}O_2$ slow component was produced during the CP test even with a steady decline in power output and muscle activation. Given the same exercise duration, the magnitude of the slow component in the CP test was

greater than the CWR test. The authors concluded that a progressive muscle fiber recruitment is not a necessity for the development of the $\dot{V}O_2$ slow component during maximal exercise. Instead, the $\dot{V}O_2$ slow component can be generated by the high oxygen cost of fatigued fibers, which either do not significantly contribute to power output or do so at a substantially greater oxygen cost relative to the work done, thereby reducing the oxygen utilization efficiency of muscular work. In summary, the slow $\dot{V}O_2$ kinetics of initially recruited fibers, reduced contractile efficiency due to the accumulation of metabolites, and the recovery processes of the fatigued fibers may play a significant role in the $\dot{V}O_2$ slow component during all-out exercise.

Keir, Fontana, Robertson, Murias, Paterson, Kowalchuk, Pogliaghi, 2015

Exercise Intensity Thresholds: Identifying the Boundaries of Sustainable Performance

The aim of this investigation was to first evaluate whether the oxygen consumption at CP, respiratory compensation point (RCP), maximum lactate steady state (MLSS), and deoxygenated hemoglobin $[HHb]_{BP}$ were equivalent, and, secondly, to represent the demarcation between heavy and very heavy exercise domains. Twelve healthy young men completed a series of tests on a cycle ergometer which included a ramp incremental (RI) test to determine $\dot{V}O_2$peak and PPO, four to five time-to-exhaustion trials between 60 – 115% PPO to determine CP, and two to three 30-minute constant power trials at a fixed cadence to determine the VO_2 and power output associated with MLSS. A second RI test to evaluate potential training effects from which the $\dot{V}O_2$peak and power output associated with RCP and $[HHb]_{BP}$ were determined. Oxygen consumption and muscle oxygenation/deoxygenation were measured during all tests, and lactate was measured at specific intervals during the constant load tests.

No training effect was observed as there was no difference between the initial and second RI test. A plateau of VO_2 was noted at the PO corresponding to CP and MLSS by the 13^{th} minute of exercise during the constant power trials. No differences were noted in oxygen consumption and heart rate between either intensity threshold, however PO differed with RCP and $[HHb]_{BP}$ both being greater than the PO at MLSS and CP. The results indicate that CP, MLSS, RCP, and $[HHb]_{BP}$ are not different from each other with regard to VO_2, suggesting that each may delineate between heavy and very heavy exercise domains. This also suggests that each threshold may be interrelated and may occur because of a common or similar mechanism that has yet to be determined.

Bailey, Wilkerson, DiMenna, Jones, 2009

<u>Influence of repeated sprint training on pulmonary O_2 uptake and muscle deoxygenation kinetics in humans</u>

The purpose of this investigation was to assess the effect of two different work-matched training protocol [repeated sprint training (RST) and endurance training (ET)] on the kinetics of VO_2, HR, and muscle deoxygenation. Twenty-four recreationally trained individuals (men and women) performed pre and post testing incremental ramp tests to determine VO_2peak, and GET on a cycle ergometer and step tests, split into two moderate intensity bouts and one severe intensity bout, to determine VO_2 kinetics. The individuals were randomly placed in RST, ET or a control (CON) group. Both training groups performed six training sessions over a two-week period, while CON maintained their normal levels of physical activity. The RST group progressively performed 30 s all-out sprints against a load equivalent to 7.5% of their body weight (in kg) with four minutes of recovery in between each sprint. The progression of

repetitions included four in the first session, five in the second session, six in the third and fourth sessions, and seven in the fifth and sixth sessions. On the other hand, the ET group cycled continuously for approximately 15 – 25 min, which equated the duration needed to match the total work done in the corresponding session of the RST group at 90%GET.

Each group had similar VO_2peak, VO_2 at GET, and VO_2 kinetics at baseline. The RST significantly improved VO_2peak and peak work rate from pre to post, whereas no changes were noted in ET and CON. However, no changes were noted in the VO_2 associated with GET in either group. There were significant reductions in the amplitude of VO_2 response, mean response time for VO_2, and oxygen deficit in the RST group only at moderate intensity showing greater efficiency of metabolic work. In addition, only the RST group had a significant reduction in blood lactate. In regards to severe exercise, only the RST group showed significant speeding of the $\dot{V}O_2$ response amplitude and mean response time along with a reduction in the rate of development for the $\dot{V}O_2$ slow component showing an attenuation of fuel storage depletion within the muscle. In terms of muscle deoxygenation, the RST group showed a significant reduction in the deoxygenation time delay, but no differences were noted in the development of the $\dot{V}O_2$ slow component between groups or post training at moderate intensity. Additionally, the deoxygenated amplitude response and the change in deoxygenated hemoglobin relative to the change in VO_2 were significantly increased in the RST group only during moderate and severe intensity exercise. However, the time delay in deoxygenation was unchanged within the RST group. The net result for RST deoxygenated hemoglobin kinetics was generally faster and the magnitude of oxygen extraction was greater. No differences were noted in HR kinetics other than a significant decrease in end exercise HR within the RST group only. Lastly, individuals in

the RST group were able to significantly 53% increase their time-to-exhaustion during severe intensity exercise, whereas ET resulted in a non-significant increase of 13%.

Pringle and Jones, 2002

Maximal lactate steady state, critical power and EMG during cycling

The aim of this investigation was to compare whether three different fatigue thresholds (MLSS, CP, and EMG_{FT}) occur at the same intensity (i.e. power output) during lower body cycling. Secondarily, this investigation examined if exercise above MLSS would result in a continued increase in $\dot{V}O_2$, blood lactate, and iEMG over time. Eight recreationally active individuals completed an incremental exercise test to determine $\dot{V}O_2max$ and lactate threshold. Participants then performed four time-to-exhaustion trials to determine CP, via the linear power versus the inverse of time model, at power outputs between 50%Δ (50% of the difference between the $\dot{V}O_2$ at lactate threshold and $\dot{V}O_2max$) and 110% $\dot{V}O_2max$. Participants also performed four 30-min constant-load transitions with power outputs between 100% of the $\dot{V}O_2$ at lactate threshold and 50%Δ to determine MLSS. Finally, they performed four two-minute square-wave transitions at 75%Δ – 115% $\dot{V}O_2max$ with 25 min of recovery between each to determine EMG_{FT}.

The power output at CP was strongly correlated (r = .95) to, but significantly greater than the power output at MLSS (by 20 W on average). CP occurred at 71 ± 3% $\dot{V}O_2max$ whereas MLSS occurred at 65 ± 3% $\dot{V}O_2max$. Comparisons with EMG_{FT} were difficult because half of the sample size could not be computed due to an extremely variable response across participants. Blood lactate, $\dot{V}O_2$, and $\dot{V}_E$ were significantly increased during exercise intensities above MLSS,

however steady state $\dot{V}O_2$ was observed at intensities at or below the power output corresponding to MLSS.

CHAPTER THREE: METHODOLOGY

Experimental Design and Methodology

A randomized, repeated measures design was employed to examine the effectiveness of traditional and modified SIT protocols on the upper body and the trainability of metabolic and neuromuscular fatigue thresholds. Participants were randomized into one of four groups: 30 s: 4 min (30:4), 10 s: 4 min (10:4), 10 s: 2 min (10:2), or control (CON). All participants were asked to complete pre- and post-testing consisting of a graded exercise test (GXT) on day one, three constant work-rate tests on day two, and a Wingate tests on day three. Following pre-testing, participants were assigned to one of the three training protocols and underwent a two-week training intervention while the control group was instructed not to significantly alter their current activity level and only perform pre- and post-testing (Figure 1). All participants were asked to maintain their normal caloric intake habits throughout the course of the investigation.

Figure 1. Illustration of the study design

Participants

Fifty-one recreationally active men between the ages of 18 and 35 years old were recruited by word of mouth and flyers. Among the recruited participants, nine subjects withdrew (seven before training and two during training) due to scheduling conflicts, injuries sustained outside of the study, and general non-responsiveness. Consequently, 42 subjects completed all testing and training sessions and were included in the final analysis, except for one participant that did not complete post weight and body composition measures. Testing procedures were explained in full before obtaining written informed consent from each participant. Each participant was asked to complete a physical activity readiness questionnaire (PAR-Q+) in order to identify any exclusion criteria, including the inability to perform physical exercise and any chronic illness that requires continuous medical care. All participants were habitually active completing a minimum of two to three days per week for at least 30 minutes per day. In an attempt to eliminate residual fatigue, the participants were asked to refrain from any strenuous physical activity for 48 hours prior to testing.

Body Composition Measures and Familiarization Trial

Body composition was estimated using a multi-frequency bioelectrical impedance device (Inbody 720, Biospace Co., Ltd.; Seoul, Korea). Participants were asked to follow the pre-testing instructions including no exercise the day of testing, no food or drink within four hours of testing, no alcohol within 48 hours of testing, avoid large quantities of water within two hours of testing, but arrive euhydrated, void bladder immediately before the measurement, and avoid lotion or wearing accessories of any kind. Participants were required to stand on the platform with their heels placed on the circular rear sole electrode before the forefoot hit the front sole

electrode. Then the participants grasped the handles and made sure the surface of the hand electrode was placed in contact with each of the five fingers. A familiarization of the GXT was provided to each subject with an upper body cycle ergometer (Brachumera sport, Lode, Groeningen, Netherlands) before testing sessions took place. Each subject was seated with the crank arm lined up with the center of their glenohumeral joint and positioned so that their arms were extended, but not fully locked out during cranking. The researchers instructed participants to crank with minimal upper body rotation, feet planted flat on the floor, and a consistent handgrip position. Any participant that performed extraneous motions while cranking was given a warning at first, but if the movements persisted the test terminated.

Graded Exercise Test

An incremental test to volitional exhaustion was performed on a cycle ergometer (Brachumera sport, Lode, Groeningen, Netherlands) to determine peak power output (PPO) in watts (W) and peak oxygen consumption ($\dot{V}O_2peak$) in liters per minute (L/min). Prior to testing, each participant was fitted with a heart rate monitor (Heart Rate Monitor, Garmin Ltd., Schaffhausen, Switzerland), to record the participants' heart rate, and a mask around their mouth and nose to collect respiratory gases. All gas exchange data was collected using a metabolic gas analyzer (Quark CPET, Cosmed, Rome, Italy). Prior to each use, the metabolic gas analyzer was calibrated with gases of known concentration (16% O_2, 5% CO_2, and N_2 bal) and calibrated for airflow with a three-liter syringe as per the manufacturer's instruction manual. Participants underwent an incremental ramp protocol that began at an initial workload of 30 W and was increased 1 W every six seconds (10 W every minute). Participants were required to maintain a cranking cadence of 50 revolutions per minute (RPM) and continued until the participant was

unable to maintain a cadence above 50 RPM for a duration of five seconds despite verbal encouragement, or volitional fatigue. The highest power output achieved was recorded as PPO and the highest 10-s average breath-by-breath oxygen consumption rate was recorded as $\dot{V}O_2peak$.

Near Infrared Spectroscopy

To assess tissue oxygenation during the GXT, a near infrared spectroscopy (NIRS) optode (PortaLite, Artinis Medical Systems, Gelderland, the Netherlands) was placed over the biceps brachii muscle on the right arm over the muscle belly 8 cm from the elbow crease (Lusina, Warburton, Hatfield, & Sheel, 2008). The NIRS optode, which transmits light and records the reflected light within a tissue, was secured using a self-adhering bandage. The NIRS signal was measured continuously and values were averaged into 10 s bins for subsequent analysis (Muraki, Tsunawake, & Yamasaki, 2004). A modified form of the Beer-Lambert Law was used to calculate micromolar changes in oxygenated hemoglobin (OHb), deoxygenated hemoglobin (HHb), and total hemoglobin (tHb) during the graded exercise test. Tissue saturation index (TSI, expressed as a percent) was then calculated [(OHb/(HHb + tHB)) × 100] to determine the balance between oxygen supply and oxygen consumption. Deoxygenated hemoglobin was plotted over time to determine a deoxygenation breakpoint $[HHB]_{BP}$ (van der Zwaard et al., 2016).

Constant Work Rate Test Trials

Three high-intensity constant-work rate tests, at different power outputs (90%, 100%, and 120% PPO), were performed. All constant-work rate tests began with a three to five-minute

warm-up at 50 W on the cycle ergometer (Brachumera sport, Lode, Groeningen, Netherlands). Exhaustion was determined to the nearest second at the moment of volitional fatigue or failure to maintain a cranking cadence above 50 RPM for a duration of five seconds. The time-to-exhaustion (TTE; in seconds) and total work (TW; in Joules) were calculated during each trial. Linear regression was used to determine the slope of the line from the relationship between TTE and TW. The calculated slope of the work-time relationship was considered CP while the y-intercept of the regression line was considered W' from the standard multi-trial CP test (J. C. Smith, Stephens, Hall, Jackson, & Earnest, 1998).

Electromyography (EMG)

To assess muscle activity during the constant-work rate trials, a bipolar (4.6 cm center-to-center) surface electrode (Quinton Quick-Prep silver-silver chloride) arrangement was placed over the biceps brachii on the right arm. The surface electrodes were placed over the muscle belly between the medial acromion and the fossa cubit at one third the distance proximal to the fossa cubit, while the ground electrode was placed on the right wrist. Inter-electrode impedance was kept below 5,000 ohms with shaving, abrasion of the skin, and alcohol cleaning beneath the electrodes. The raw EMG signals were pre-amplified using a differential amplifier (MP150, BIOPAC Systems, Inc., Santa Barbara, CA), sampled at 1,000 Hz, and stored on a personal computer (Dell Latitude E6530, Dell Inc., Round Rock, TX) for off-line analysis. Raw EMG data was processed through a band-pass Butterworth filter (from 10 to 500 Hz) on a computerized software program (AcqKnolwedge 4.2., BIPOAC Systems, Inc., Goleta, GA, USA). Root mean square values were taken in 10s bins and plotted over time for each constant-work rate trial to obtain the slope of each trial. The power output for each constant-work rate

trial was then plotted over each fatigue slope coefficient to determine the y-intercept, which was defined as the electromyographic fatigue threshold (EMG_{FT}) (Devries et al., 1982).

Wingate test

Each participant warmed up for 3 - 5 min prior to each testing session. One Wingate test was performed on an upper body cycle ergometer (891E, Monark Upper Body Ergometer, Vansbro, Sweden) using $0.05 kg \cdot kg^{-1}$ of the participants body mass. Each participant was told to accelerate as fast as possible from the command of "GO!" and to sprint maximally for the entire 30 s duration on each test. Power output was registered using the Monark software (Monark ATS software, Vansbro, Sweden). Peak power (PP) was recorded as the highest power output generated during the test and mean power (MP) was recorded as the average power output over the entire test. Total work completed was also recorded.

Determination of Energy Systems Contribution

Estimates of oxidative, glycolytic, and ATP-PCr systems contribution were generated through oxygen uptake, blood lactate concentration, and the fast component of excess post-exercise oxygen consumption, respectively. During the Wingate test, oxygen uptake was recorded, at rest, for five minutes prior to the warm-up and the testing trial with the last 30 s as the baseline reference. Next, total oxygen consumption during exercise was calculated as the area under the curve (trapezoidal method). The aerobic contribution was calculated as the oxygen uptake during exercise minus the oxygen uptake at baseline (baseline oxygen uptake was then multiplied by the total time of exercise). The fast (ATP-PCr contribution) and slow component of exercise post oxygen consumption was analyzed, for five minutes, by the

biexponential or the monoexponential curve (using GEDAE-LaB software) (Bertuzzi et al., 2016) to see which fits the data best (see equation below) (Beneke, Pollmann, Bleif, Leithäuser, & Hütler, 2002).

$$\dot{V}O_2(t) = Ae^{t/tA} + Be^{t/tB} + (\dot{V}O_{20})$$

A = amplitude of fast component

B = amplitude of slow component

tA = fast component time constant

tB = slow component time constant

$\dot{V}O_{20}$ = the $\dot{V}O_2$ at rest

The contribution of each energy system was expressed in absolute terms (kJ), assuming the caloric quotient of 20.9 $kJ \cdot LO_2^{-1}$ (Gastin, 2001) and as a relative percentage of total metabolic work. Lastly, each system was summed to calculate total metabolic work (in kJ).

Blood samples were obtained via ear lobe prior to each Wingate test and three and five minutes following each test to determine the peak plasma lactate concentration using a lactate analyzer (Lactate Plus, Nova Biomedical, Waltham, MA). The glycolytic contribution was estimated assuming the accumulation of one $mmol \cdot L^{-1}$ of blood lactate is equivalent to three milliliters of oxygen per kilogram of body mass (di Prampero & Ferretti, 1999).

Exercise Training Protocol

A SIT program consisting of six training sessions (three sessions per week for two weeks) was employed and each session were separated by at least 48 hours. Each training

session began with a five-minute warm-up at 50 W, and then four 30-second or 10-second all-out repeated sprints using 0.05kg·kg^{-1} (or 5%) body mass loading (Franchini et al., 2016) interspersed by either two or four minutes of passive recovery (see Table 1). Training took place on a modified cycle ergometer (894E, Monark Cycle Ergometer, Vansbro, Sweden) that was placed on adjustable scaffolding for arm cranking. Subjects were instructed to perform all-out sprints trying to reach and maintain the highest power output for every sprint while strong verbal encouragement was given throughout. Training progression increased one repetition every two training sessions, thus four repetitions during the first two training sessions, five repetitions during the middle two training sessions, and six repetitions for the final two training sessions (Hazell et al., 2010). Peak power (PP, in W), mean power (MP, in W), and total work (TW, in J) were recorded. In addition, participants were asked to provide a perceived readiness rating (PRR) within 15 s prior to each sprint. The PRR is a progressive scale from one to five with one stating "Not at all ready to begin" and five stating "Completely ready to begin". Exercise Density (in J·s^{-1}) was also calculated for each participant by dividing the six session sum of TW over the sum of the inter-set recovery, in seconds, over the two-week intervention (Marston, Peiffer, Newton, & Scott, 2017).

Table 2. Training protocol for each of the three training groups

30:4

Training Session	**Number of Sprints**	**Resistance per Sprint**	**Duration of Sprints**	**Duration of Recovery**
Day 1 and 2	4	5%	30 s	4 min
Day 3 and 4	5	5%	30 s	4 min
Day 5 and 6	6	5%	30 s	4 min

10:4

Training Session	**Number of Sprints**	**Resistance per Sprint**	**Duration of Sprints**	**Duration of Recovery**
Day 1 and 2	4	5%	10 s	4 min
Day 3 and 4	5	5%	10 s	4 min
Day 5 and 6	6	5%	10 s	4 min

10:2

Training Session	**Number of Sprints**	**Resistance per Sprint**	**Duration of Sprints**	**Duration of Recovery**
Day 1 and 2	4	5%	10 s	2 min
Day 3 and 4	5	5%	10 s	2 min
Day 5 and 6	6	5%	10 s	2 min

Statistical Analysis

An analysis of covariance (ANCOVA) was performed on all testing measurements collected at post-testing to identify differences between groups for aerobic capacity, fatigue

thresholds, anaerobic performance, and energy system utilization. Associated values collected at pre were used as covariates to eliminate the possible influence of initial score variances on training outcomes. An analysis of variance (ANOVA) was used to compare average TW, PP, MP, and exercise density over the training intervention between training groups, whereas a Kruskal-Wallis analysis of variance was used for PRR due its categorical scale and non-normality. When appropriate, post hoc Bonferroni pairwise comparisons were used to examine the differences among the groups. Pearson r correlations were conducted between total lean arm mass and performance variables, CP and energy system contribution, and W' and energy system contribution; however, Spearman rho correlations were conducted when variables did not display normal distributions. Outliers were removed if they fell outside three times the median absolute deviation (MAD) for $\dot{V}O_2$peak, CP, and EMG_{FT} (Leys, Ley, Klein, Bernard, & Licata, 2013). With less than 6% of the data missing (Tabachnick & Fidell, 2013), multivariate imputation using partial least squares method was performed via JMP Pro 12 (Cary, NC, USA) on energy system contribution and training data. For effect size, the partial eta squared statistic was calculated with an interpretation of 0.01, 0.06, and 0.14 as small, medium, and large effect sizes, respectively (Cohen, 1988). Significance was established at an alpha of $p < 0.05$, whereas a trend was noted if $p < 0.10$. All data were reported as mean ± SD. Additionally, post-test measures were reported as mean ± 95% confidence intervals to indicate meaningful changes as compared with covariate adjusted pre-test values. Statistical software (IBM SPSS Statistics for Windows, Version 23.0; Armonk, NY: IBM Corp) was used for all analyses.

In addition, typical error (TE) was calculated for $\dot{V}O_2$peak, CP, and EMG_{FT} as the standard deviation of the difference scores (post-pre) divided by the square root of two (Hopkins, 2000). A responder was defined as a participant that increased or decreased more than two times

the TE, whereas a non-responder was defined as a participant that did not increase or decrease more than two times TE for each respective variable following training (Bonafiglia et al., 2016).

CHAPTER FOUR: RESULTS

Anthropometric Changes

Baseline participant characteristics are displayed in Table 3. Significant differences between groups were noted in post-test body mass ($F_{3,36} = 6.248$, $p = .002$, $\eta^2 = .342$) with an adjusted pre-test mean of 79.1 kg (Table 4). All groups were significantly heavier than 30:4 (10:2, $p = .023$; 10:4, $p = .015$; CON, $p = .002$). No significant between-group differences were noted between post-test %BF ($F_{3,36} = .165$, $p = .919$, $\eta^2 = .014$; adjusted pre-test mean = 17.2 %), RA lean mass ($F_{3,36} = 1.951$, $p = .139$, $\eta^2 = .140$; adjusted pre-test mean = 3.8 kg), LA lean mass ($F_{3,36} = 1.971$, $p = .136$, $\eta^2 = .141$; adjusted pre-test mean = 3.8 kg), or total lean arm mass ($F_{3,36} = 2.050$, $p = .124$, $\eta^2 = .146$; adjusted pre-test mean = 7.6 kg) (Table 4).

Table 3. Participant characteristics

	10:2 (n = 11)	10:4 (n = 11)	30:4 (n = 10)	CON (n = 10)
Age (yr)	22.8 ± 3.2	22.4 ± 3.2	23.1 ± 3.2	24.3 ± 3.3
Height (cm)	176.4 ± 6.9	176.2 ± 8.7	172.9 ± 7.1	174.2 ± 4.9
Body Mass (kg)	81.2 ± 9.5	83.6 ± 13.3	73.9 ± 12.1	77.0 ± 11.5

Note. Data are mean ± standard deviation (SD) and represent baseline characteristics of the participants training in the 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), or control group (CON). n = sample size.

Aerobic Capacity and Fatigue Thresholds

There were significant differences between groups in absolute $\dot{V}O_2$peak ($F_{3,37} = 5.003$, $p = .005$, $\eta^2 = .289$; adjusted pre-test mean = 2.44 $L \cdot min^{-1}$) and PPO ($F_{3,37} = 4.291$, $p = .011$, $\eta^2 = .258$; adjusted pre-test mean = 130.9 W). In regards to $\dot{V}O_2$peak, both 10:2 and 30:4 were greater than CON group ($p = .036$ and $p = .007$, respectively), while a trend was noted between 10:4 and CON ($p = .056$). In regards to PPO, only 30:4 was greater than CON group ($p = .007$)

(Figure 2 and Table 5). Due to erratic behavior in the deoxygenated hemoglobin signal (i.e. non-sigmoidal), the deoxygenation breakpoint could not be established. Due to outliers (i.e. >3MAD), four subjects were removed from EMG_{FT} analysis. No significant differences were found between groups in CP ($F_{3,37} = .748$, $p = .530$, $\eta^2 = .057$; adjusted pre-test mean = 88.2 W), W' ($F_{3,37} = .193$, $p = .900$, $\eta^2 = .015$; adjusted pre-test mean = 6.79 kJ), or EMG_{ft} ($F_{3,33} = .490$, $p = .692$, $\eta^2 = .043$; adjusted pre-test mean = 99.0 W) (Figure 3 and Table 6). Responders were classified as participants that fell beyond the range of TE×2 (±0.350 $L \cdot min^{-1}$ for $\dot{V}O_2peak$, ±17.6 W for CP, and ±45.9 W for $EMG_{FT)}$. Three participants (one from each training group) responded positively in regards to $\dot{V}O_2peak$, whereas five separate participants (three from 30:4 and two from 10:4) responded positively to CP. Although, one participant (from 10:4) responded negatively in regards to CP. Two participants (one from 30:4 and one from 10:2) responded positively in regards of EMG_{FT}. Among the responders, only one participant overlapped positively in regards to both $\dot{V}O_2peak$ and EMG_{FT}.

Relative to body weight, there was a significant difference between groups in relative $\dot{V}O_2peak$ ($F_{3,36} = 6.455$, $p = .001$, $\eta^2 = .350$) with an adjusted mean of 31.1 $ml \cdot kg^{-1} \cdot min^{-1}$. The 30:4 group was greater than CON group ($p = .001$), but a trend was noted between 10:2 and CON group ($p = .089$). Significant between group differences were noted between post-test PPO ($F_{3,36} = 7.07$, $p = .001$, $\eta^2 = .371$) with an adjusted mean of 1.68 $W \cdot kg^{-1}$. The 30:4 group was greater than the CON group ($p < .001$), and trends were noted in 10:2 ($p = .055$) and the 10:4 ($p = .097$) groups compared to the CON. However, no significant differences were observed in CP ($F_{3,36} = 2.171$, $p = .108$, $\eta^2 = .153$; adjusted pre-test mean = 1.14 $W \cdot kg^{-1}$), W' ($F_{3,36} = .331$, $p = .803$, $\eta^2 = .027$; adjusted pre-test mean = .09 $kJ \cdot kg^{-1}$), or EMG_{FT} ($F_{3,32} = 1.880$, $p = .947$, $\eta^2 = .011$; adjusted pre-test mean = 1.26 $W \cdot kg^{-1}$) relative to body weight.

Relative to total lean arm mass, there was a significant difference between groups in absolute $\dot{V}O_2$peak ($F_{3,36} = 5.390$, $p = .004$, $\eta^2 = .310$) with an adjusted mean of .325 $L \cdot min^{-1} \cdot kg^{-1}$ and PPO ($F_{3,36} = 6.666$, $p = .001$, $\eta^2 = .357$) with an adjusted mean of 17.4 $W \cdot kg^{-1}$. The 30:4 group was greater than CON group ($p = .002$) in absolute $\dot{V}O_2$peak, and greater than the 10:2 ($p = .007$) and CON ($p = .001$) groups in PPO. No significant differences were observed for W' ($F_{3,36} = .749$, $p = .749$, $\eta^2 = .033$; adjusted pre-test mean = .89 $kJ \cdot kg^{-1}$), or EMG_{FT} ($F_{3,32} = .325$, $p = .807$, $\eta^2 = .030$; adjusted pre-test mean = 13.1 $W \cdot kg^{-1}$), but a trend was noted in CP ($F_{3,36} = 2.363$, $p = .087$, $\eta^2 = .165$; adjusted pre-test mean = 11.8 $W \cdot kg^{-1}$) relative to total lean arm mass.

Wingate Performance

No significant differences were noted between groups in the upper body (UB) performance variables: UB PP ($F_{3,37} = 1.114$, $p = .692$, $\eta^2 = .043$; adjusted pre-test mean = 684.2 W), UB MP ($F_{3,37} = 1.297$, $p = .290$, $\eta^2 = .095$; adjusted pre-test mean = 394.4 W), UB TW ($F_{3,37} = .1.293$, $p = .291$, $\eta^2 = .095$; adjusted pre-test mean = 11.3 kJ) (Figure 4 and Table 7).

Relative to total lean arm mass there were no differences between groups in PP ($F_{3,36} = 1.043$, $p = .385$, $\eta^2 = .080$; adjusted pre-test mean = 90.2 $W \cdot kg^{-1}$). There were significant differences between groups in MP ($F_{3,36} = 2.98$, $p = .044$, $\eta^2 = .199$; adjusted pre-test mean = 51.9 $W \cdot kg^{-1}$) and TW ($F_{3,36} = 2.953$, $p = .045$, $\eta^2 = .197$; adjusted pre-test mean = 1.49 $kJ \cdot kg^{-1}$). In MP, 30:4 was significantly greater than CON ($p = .048$) and in TW, there was a trend for greater work completed in 30:4 compared to CON ($p = .061$).

Energy System Contribution

There were significant differences between groups in relative energy contribution for the ATP-PCr system ($F_{3,37}$ = 3.393, p = .028, η^2 = .216) with an adjusted mean of 43.5%. The 10:4 was greater than CON (p = .026). No significant differences were observed in the oxidative ($F_{3,37}$ = 1.485, p = .235, η^2 = .107; adjusted pre-test mean = 9.9 %) or glycolytic ($F_{3,37}$ = 2.084, p = .119, η^2 = .145; adjusted pre-test mean = 46.6 %) systems (Figure 5 and Table 8).

There were significant differences between groups in energy expenditure for the ATP-PCr system ($F_{3,37}$ = 3.580, p = .023, η^2 = .225) with an adjusted mean of 50.5 kJ. The 10:4 was greater than CON (p = .019). No significant differences were observed in the oxidative ($F_{3,37}$ = .952, p = .426, η^2 = .072; adjusted pre-test mean = 11.2 kJ) or glycolytic ($F_{3,37}$ = .141, p = .935, η^2 = .011; adjusted pre-test mean = 53.5 kJ) systems. No significant differences were observed in total metabolic work between groups ($F_{3,37}$ = 1.275, p = .297, η^2 = .094; adjusted pre-test mean = 115.3 kJ) (Figure 6 and Table 9).

Training

All subjects within the training groups completed 100% of the training session. Average PP was significantly different between training groups ($F_{2,31}$ = 5.35, p = .011) with the 10:2 (p = .013) and 10:4 (p = .049) groups significantly greater than the 30:4 group. Average MP was significantly different between training groups ($F_{2,31}$ = 26.637, p < .001) with the 10:2 (p < .001) and 10:4 (p < .001) groups significantly greater than the 30:4 group. Average TW was significantly different between training groups ($F_{2,31}$ = 57.489, p < .001) with the 30:4 group greater than both the 10:2 (p < .001) and the 10:4 (p = .002) groups. There was a significant difference in the distribution of ranks between groups in PRR (X^2_3 = 7.178, p = .028) with 10:4

group (M = 4.77) having a greater median value than 30:4 (M = 4.14, p = .022), but no differences with 10:2 (M = 4.25). There was a significant difference between groups in ED ($F_{2,31}$ = 50.925, p < .001) with 10:2 having a greater density than 30:4 (p = .010) and both 10:2 (p < .001) and 30:4 (p < .001) having greater densities than 10:4 (Table 10).

Correlations

Total lean arm mass was correlated to absolute $\dot{V}O_2$peak (r = .49, p = .001, PPO (r = .56, p < .001), CP (r = .39, p = .012), W' (r = .34, p = .03), EMG_{FT} (r = .65, p < .001), PP (r = .52, p < .001), MP (r = .73, p < .001), TW (r = .67, p < .001), absolute (r = .63, p < .001) and relative (r = .37, p = .017) ATP-PCr energy, absolute energy from glycolysis (ρ = .31, p = .047), and total metabolic work (ρ = .54, p = .047). W' was correlated to absolute energy derived from glycolysis (ρ = .38, p = .013). CP was correlated to absolute (r = .55, p < .001) and relative (r = .31, p = .045) energy derived from ATP-PCr and absolute energy derived from oxidative (r = .31, p = .025).

Table 4. Anthropometric measures before (pre) and after (post) training

	10:2 (*n* = 11)		10:4 (*n* = 11)		30:4 (*n* = 10)		CON (*n* = 9)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Body Mass (kg)	81.2 ± 9.5	82.2 ± 9.4	83.6 ± 13.3	84.7 ± 13.7	73.9 ± 12.1	73.0 ± 11.8	77.0 ± 11.5	78.5 ± 12.2
%BF	15.7 ± 6.2	15.7 ± 6.3	17.8 ± 3.5	18.3 ± 4.7	16.8 ± 5.9	16.7 ± 5.4	19.0 ± 7.3	18.6 ± 6.7
Total lean arm mass (kg)	8.1 ± 1.2	8.2 ± 1.1	8.2 ± 1.7	8.2 ± 1.7	7.0 ± 1.1	7.0 ± 1.2	7.3 ± 1.2	7.5 ± 1.1

Note. Data are mean ± standard deviation (SD) representing raw data measured before and after training in the 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON). *n* = sample size. %BF = percent body fat.

Table 5. Graded exercise test variables before (pre) and after (post) training

	10:2 (*n* = 11)		10:4 (*n* = 11)		30:4 (*n* = 10)		CON (*n* = 10)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
VO_2peak ($L \cdot min^{-1}$)	2.58 ± 0.35	2.53 ± 0.38	2.61 ± 0.32	2.58 ± 0.29	2.21 ± 0.30	2.36 ± 0.26	2.56 ± 0.34	2.17 ± 0.34
VO_2peak ($mL \cdot kg^{-1} \cdot min^{-1}$)	31.3 ± 3.6	31.1 ± 4.6	31.5± 3.1	31.0 ± 5.6	30.3 ± 4.4	32.8 ± 5.0	32.2 ± 4.4	28.4 ± 3.1
PPO (W)	142 ± 22	142 ± 20	129 ± 14	136 ± 12	124 ± 14	136 ± 14	135 ± 26	127 ± 22

Note. Data are mean ± standard deviation (SD) representing raw data measured before and after training in the 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON). *n* = sample size. VO_2peak = peak oxygen consumption; PPO = peak power output.

Table 6. Constant-work rate trials variables before (pre) and after (post) training

	10:2 (n = 11)		10:4 (n = 11)		30:4 (n = 10)		CON (n = 10)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
CP (W)	98.3 ± 18.0	99.2 ± 17.9	86.4 ± 13.9	95.4 ± 14.7	84.9 ± 16.7	94.9 ± 16.2	83.6 ± 15.5	85.5 ± 12.4
W' (kJ)	6.46 ± 1.65	6.78 ± 1.90	6.87± 1.67	6.34 ± 1.61	6.83 ± 2.43	6.95 ± 2.84	7.40 ± 2.79	7.12 ± 3.37
	(n = 10)		(n = 10)		(n = 10)		(n = 8)	
EMG_{FT} (W)	100.7 ± 24.8	113.1 ± 20.8	103.7 ± 13.6	112.1 ± 17.7	96.9 ± 20.1	100.6 ± 16.9	93.4 ± 19.1	104.2 ± 33.4

Note. Data are mean ± standard deviation (SD) representing raw data measured before and after training in the 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON). n = sample size. CP = critical power; W' = anaerobic working capacity; EMG_{FT} = electromyographic fatigue threshold.

Table 7. Wingate test variables before (pre) and after (post) training

	10:2 (n = 11)		10:4 (n = 11)		30:4 (n = 10)		CON (n = 10)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
PP (W)	753 ± 189	905 ± 295	666 ± 189	772 ± 195	630 ± 109	749 ± 159	682 ± 231	716 ± 224
MP (W)	427 ± 71	437 ± 75	396 ± 66	419 ± 65	372 ± 58	409 ± 69	379 ± 78	382 ± 78
TW (kJ)	12.2 ± 1.9	12.3 ± 2.2	11.3± 1.7	11.8 ± 1.7	10.8 ± 1.6	11.8 ± 1.8	10.8 ± 2.3	10.8 ± 2.3

Note. Data are mean ± standard deviation (SD) representing raw data measured before and after training in the 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON). n = sample size. PP = peak power; MP = mean power; TW = total work.

Table 8. Relative energy system contribution before (pre) and after (post) training

	10:2 (*n* = 11)		10:4 (*n* = 11)		30:4 (*n* = 10)		CON (*n* = 10)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Oxidative (%)	9.7 ± 3.7	8.9 ± 2.0	10.1 ± 2.4	10.0 ± 2.4	9.8 ± 3.3	9.5 ± 2.1	10.2 ± 3.6	10.8 ± 2.3
Glycolytic (%)	46.9 ± 6.6	43.5 ± 6.0	45.1 ± 6.9	40.1 ± 5.7	46.8 ± 6.0	44.7 ± 4.5	47.6 ± 5.7	46.1 ± 5.0
ATP-PCr (%)	43.4 ± 9.7	47.7 ± 6.2	44.8 ± 7.9	49.9 ± 5.1	43.4 ± 6.1	45.8 ± 2.9	42.3 ± 4.7	43.1 ± 5.3

Note. Data are mean ± standard deviation (SD) representing raw data measured before and after training in the 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON). *n* = sample size.

Table 9. Absolute energy system contributions before (pre) and after (post) training

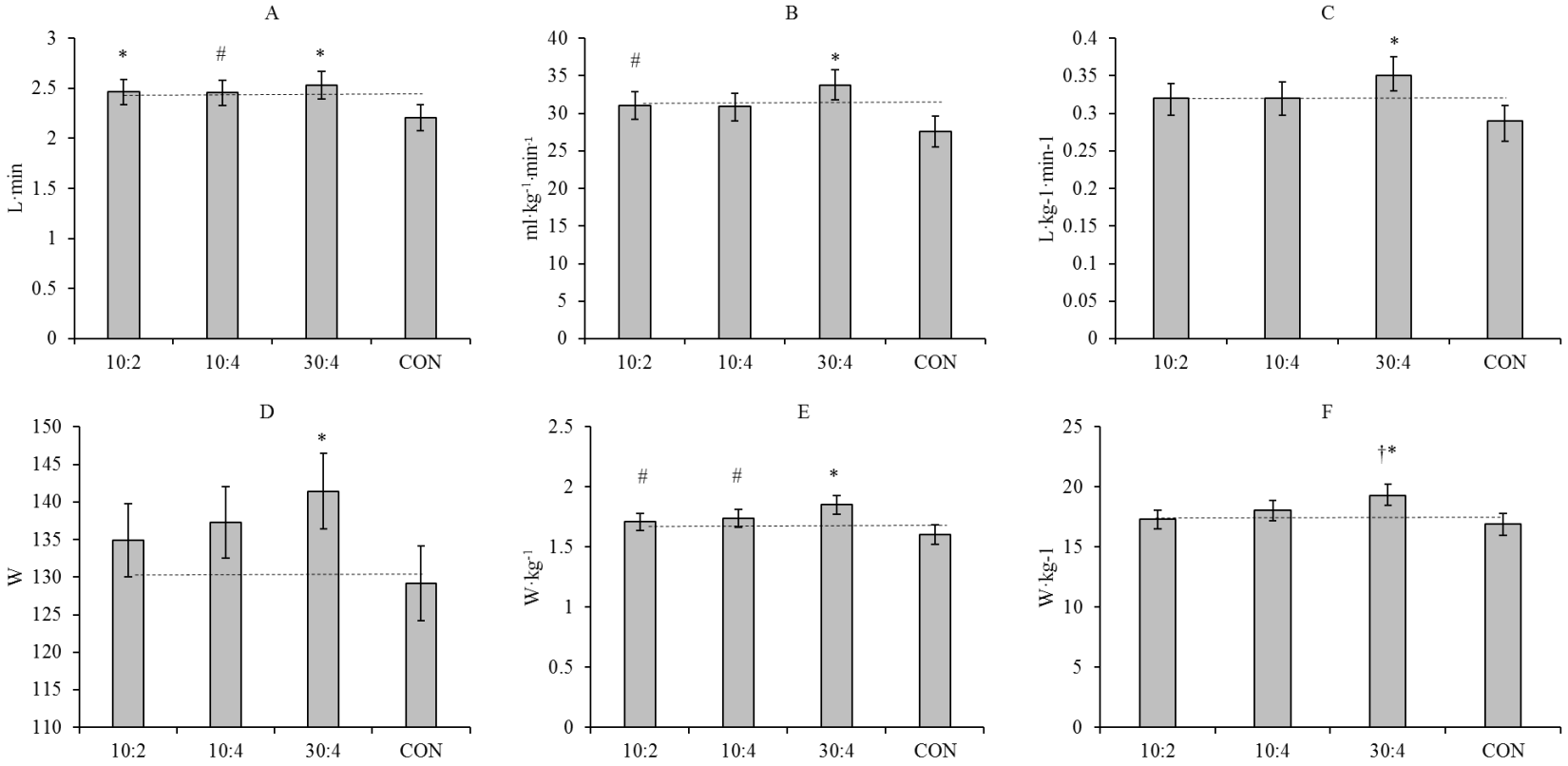
	10:2 (*n* = 11)		10:4 (*n* = 11)		30:4 (*n* = 10)		CON (*n* = 10)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Oxidative (kJ)	11.6 ± 2.9	11.0 ± 2.5	11.1 ± 2.3	12.2 ± 2.5	10.8 ± 3.0	10.6 ± 2.0	11.3 ± 4.1	11.7 ± 3.3
Glycolytic (kJ)	57.2 ± 8.6	55.4 ± 19.3	50.3 ± 11.1	49.8 ± 12.1	52.5 ± 9.5	51.7 ± 12.9	54.1 ± 14.6	50.1 ± 10.0
ATP-PCr (kJ)	54.8 ± 17.6	59.8 ± 14.9	49.8 ± 11.1	62.1 ± 14.1	49.2 ± 11.7	52.6 ± 10.5	47.8 ± 12.8	47.2 ± 11.1

Note. Data are mean ± standard deviation (SD) representing raw data measured before and after training in the 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON). *n* = sample size.

Table 10. Performance variables during training

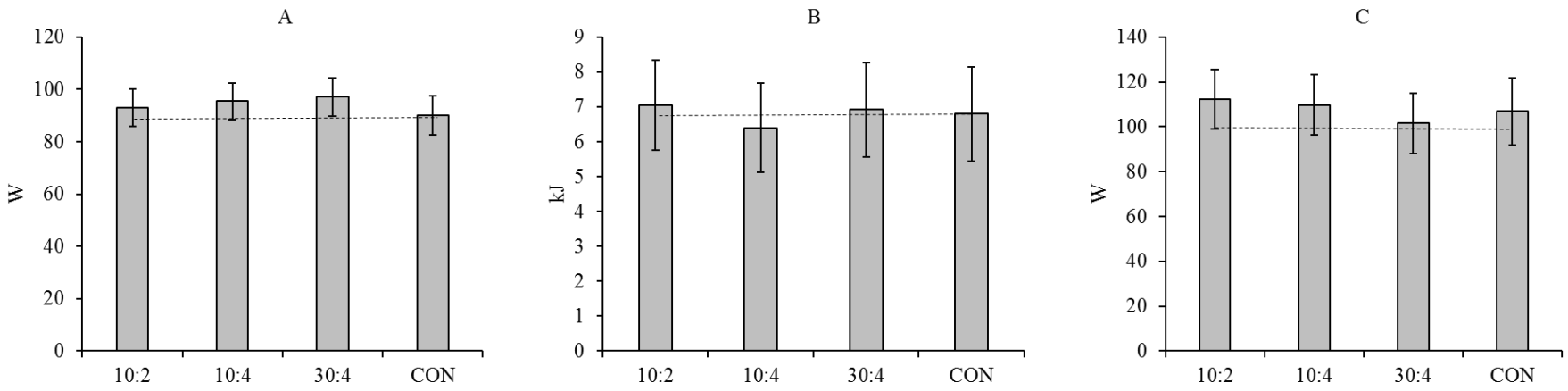
	10:2 (*n* = 11)	10:4 (*n* = 11)	30:4 (*n* = 10)
PP (W)	638 ± 147*	611 ± 113*	482 ± 73
MP (W)	495 ± 85*	488± 73*	300 ± 38
TW (kJ)	149 ± 26*	147 ± 21*	248 ± 27
ED ($J \cdot s^{-1}$)	51.7 ± 8.9*#	25.5± 3.7	43.1 ± 4.6

Note. Data are mean ± standard deviation (SD) from training in the 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON). *n* = sample size. PP = average peak power; MP = average mean power; TW = average total work; PRR = perceived readiness rating; ED = exercise density. *Significantly different than 30:4 ($p<.05$). #Significantly different than 10:4 ($p<.05$).



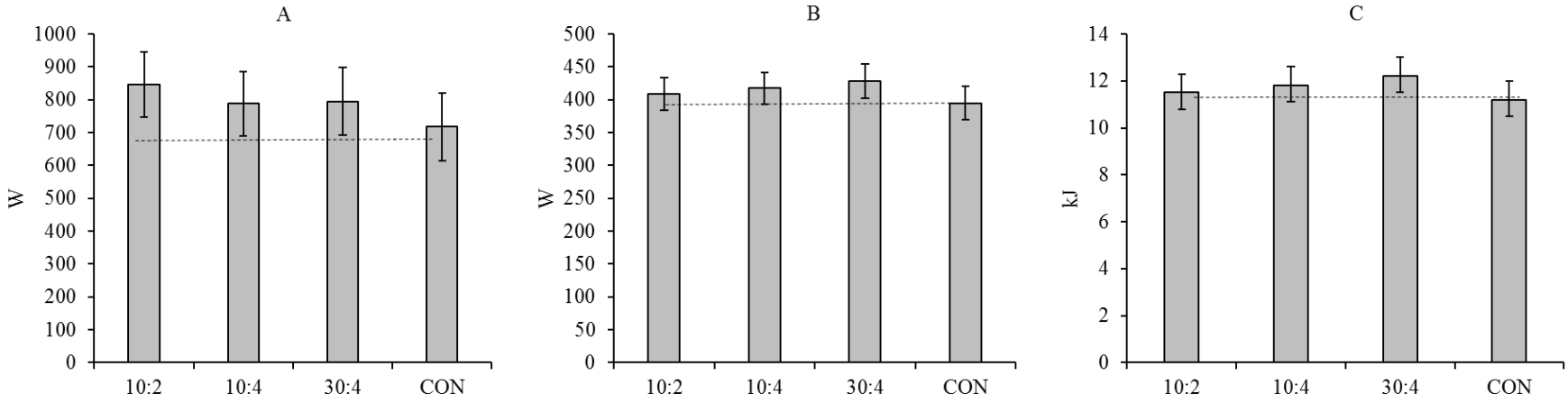
Note. Mean values (±95% confidence interval) for posttest adjusted for initial differences in pretest (dashed line) for 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON): A. Maximal oxygen uptake ($\dot{V}O_2$peak; covariate: adjusted pretest mean = 2.44 $L \cdot min^{-1}$); B. Maximal oxygen uptake relative to body weight ($\dot{V}O_2$peak; covariate: adjusted pretest mean = 31.3 $ml \cdot kg^{-1} \cdot min^{-1}$); C. Maximal oxygen uptake relative to total lean arm mass ($\dot{V}O_2$peak; covariate: adjusted pretest mean = .325 $L \cdot kg^{-1} \cdot min^{-1}$); D. Peak power output (PPO; covariate: adjusted pretest mean = 130.9 W); E. Peak power output relative to body mass (PPO; covariate: adjusted pretest mean = 1.68 $W \cdot kg^{-1}$); F. Peak power output relative to total lean arm mass (PPO; covariate: adjusted pretest mean = 17.4 $W \cdot kg^{-1}$). *Significantly different from CON ($p<.05$). #Trend compared to CON ($p<.10$). †Significantly different from 10:2 ($p<.05$).

Figure 2. Graded exercise testing variables after 2 weeks of training



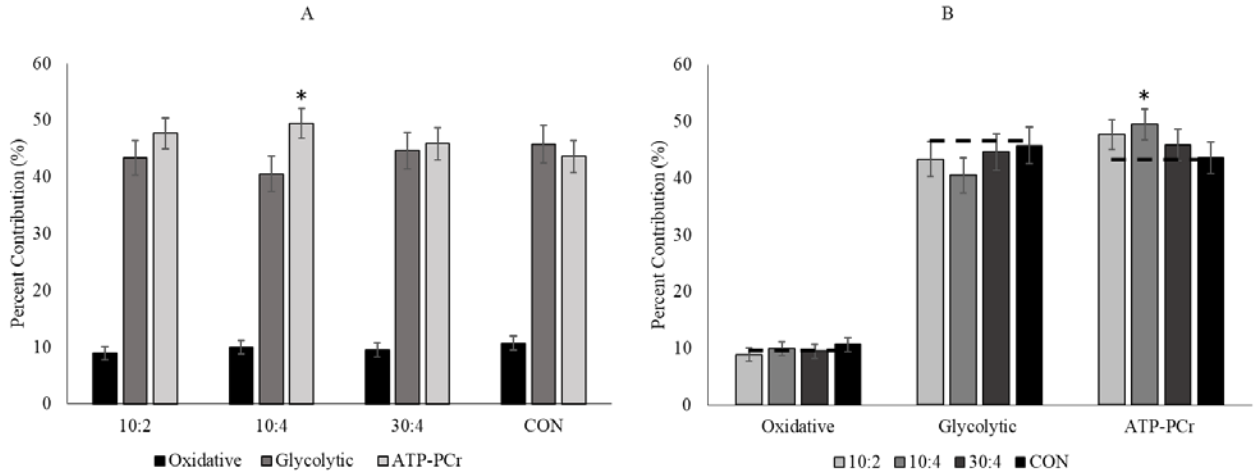
Note. Mean values (±95% confidence interval) for posttest adjusted for initial differences in pretest (dashed line) for 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON): A. Critical power (CP; covariate: adjusted pretest mean = 88.2 W); B. Anaerobic working capacity (W'; covariate: adjusted pretest mean = 6.8 kJ); C. Electromyography fatigue threshold (EMG_{FT}; covariate: adjusted pretest mean = 99 W)

Figure 3. Constant-work rate testing variables after 2 weeks of training



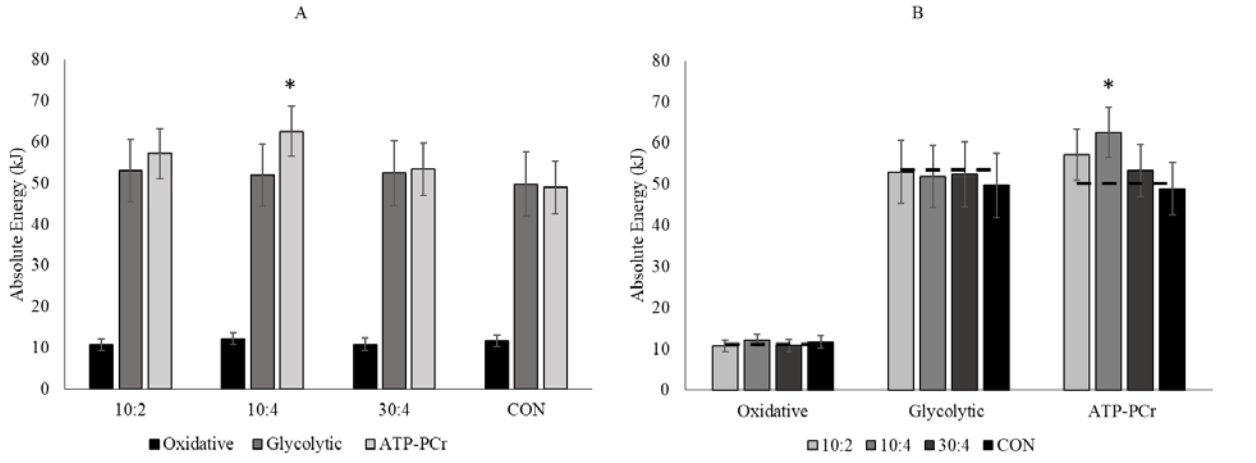
Note. Mean values (±95% confidence interval) for posttest adjusted for initial differences in pretest (dashed line) for 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON): A. Peak Power (PP; covariate: adjusted pretest mean = 684.2 W); B. Mean Power (MP; covariate: adjusted pretest mean = 394.4 W); C. Total Work (TW; covariate: adjusted pretest mean = 11.3 kJ)

Figure 4. Wingate testing performance variables after 2 weeks of training



Note. Mean values (±95% confidence interval) for posttest adjusted for initial differences in pretest (dashed line) for 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON): A. Relative contribution between energy systems; B. Relative energy contribution between groups (Oxidative; covariate: adjusted pretest mean = 9.9 %; Glycolytic: covariate: adjusted pretest mean = 46.6 %, ATP-PCr; covariate: adjusted pretest mean = 43.5 %). *Significantly different from CON (p<.05)

Figure 5. Relative energy system contribution



Note. Mean values (±95% confidence interval) for posttest adjusted for initial differences in pretest (dashed line) for 10 s 2 min group (10:2), 10 s 4 min group (10:4), 30 s 4 min group (30:4), and control group (CON): A. Relative contribution between energy systems; B. Relative energy contribution between groups (Oxidative; covariate: adjusted pretest mean = 11.2 kJ; Glycolytic: covariate: adjusted pretest mean = 53.5 kJ; ATP-PCr; covariate: adjusted pretest mean = 50.5 kJ). *Significantly different from CON (p<.05)

Figure 6. Absolute energy system contribution

CHAPTER FIVE: DISCUSSION

The purpose of this study was to compare different work-to-rest ratios utilized during SIT on upper body aerobic capacity and fatigue thresholds, anaerobic performance, and energy system contribution. The major finding of this study was that a two-week period of SIT could be effective in enhancing upper body aerobic capacity. It appears that completing a greater amount of total work with at least a moderate level of metabolic stress, quantified via ED, provides a more optimal stimulus for upper body aerobic adaptations. Alternatively, SIT did not bring about delays in fatigue thresholds (i.e. CP and EMG_{FT}) or improvements in anaerobic performance. This appears to be the first study to examine changes in energy system utilization after upper body SIT. The novel finding was that individuals undergoing a modified SIT protocol utilizing 10 s with four minutes of rest were able to draw a greater amount of energy from the ATP-PCr system during a 30-second anaerobic task as compared to a control group.

In agreement with previous research examining similar work-to-rest ratio SIT protocols, aerobic capacity was improved (Gillen et al., 2014; Hazell et al., 2010; Zelt et al., 2014; Zinner et al., 2016). Although the current results of this study indicate that training with a reduced duration work bout (i.e. 10 s) does not diminish aerobic adaptations, as observed by Zelt and colleagues (2014) and Hazell and colleagues (2010), the duration of rest appears to be influential. Both 10:2 and 30:4 resulted in significant improvements in absolute $\dot{V}O_2$peak over CON, whereas 10:4 showed a trend towards greater improvement compared to CON. The 30:4 protocol elicited a positive response in $\dot{V}O_2$peak and PPO in terms of absolute, relative to body weight, and relative to lean arm mass values than CON. Additionally, 30:4 attained a greater PPO relative to lean arm mass than 10:2. Adaptations from shorter work-to-rest ratios could be

due to the greater metabolic demand within the 30:4 and 10:2 groups (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995; Buchheit et al., 2012; Glaister, 2005). The greater metabolic demand may be supported by the currently reported lower average feeling of perceived readiness within participants from 30:4 as compared to 10:4. Zinner and colleagues (2016) examined adaptations in the arm muscles from two weeks of a combined upper and lower body SIT protocol, however their protocol solely consisted of the traditional (30 s sprints) SIT protocol with four minutes of rest rather than the modified 10 s protocols used in the current investigation. Our findings are contrary to previous reports employing the same SIT protocols in the lower body by Hazell and colleagues (2010) which found 30:4 and 10:4 more beneficial for improvements in $\dot{V}O_2$peak compared to CON; however, a trend was noted for improvements in 10:2 compared to CON. In particular, SIT has been shown to increase PGC-1α mRNA expression and protein content (Burgomaster et al., 2008; Gibala et al., 2009; Scalzo et al., 2014), which could induce a phenotypic expression characteristic of slow-twitch muscle fibers (Lin et al., 2002), and given the upper body's fast-twitch dominance (Sanchis-Moysi et al., 2010; Zinner et al., 2016) may result in a large potential for aerobic adaptation. It has been shown that SIT can induce a muscle fiber transition from either type I or type IIb towards type IIa, thereby increasing the relative percentage within the muscle (Parcell et al., 2005; Ross & Leveritt, 2001). Given the differences between the upper and lower body musculature, the upper body may benefit from larger work-to-rest ratios that provide more work to be accomplished while maintaining a high exercise density. A greater exercise density may indirectly limit recovery of severely depleted PCr stores, which in turn, will rely heavily on aerobic metabolism to compensate (Bogdanis et al., 1995). This added metabolic cost along with greater exercise densities may distinguish the adaptations observed in the 30:4 and 10:2 protocols from the 10:4 protocol. Further, lower body

improvements in $\dot{V}O_2$peak due to SIT may be attributed to increased muscle oxygen extraction or capillarization in the active musculature (Vollaard, Metcalfe, & Williams, 2017). Additionally, two weeks of lower body SIT and HIT has been shown to increase muscle oxidative capacity, resting muscle glycogen levels, muscle buffering capacity, and aerobic and anaerobic enzymatic activity (Gibala et al., 2006; Little, Safdar, Wilkin, Tarnopolsky, & Gibala, 2010; Rodas, Ventura, Cadefau, Cussó, & Parra, 2000).

This appears to be the first study to examine changes in the work-time relationship before and after a two-week SIT protocol in the upper body. Despite improvements in maximal aerobic capacity, two weeks of SIT did not stimulate increases in CP or W'. In contrast to the current findings, Zelt and colleagues (2014) found a main effect for training over four weeks with two SIT protocols during lower body cycling. However, the authors utilized 30 s work bouts with longer rest periods (i.e. 4.5 min and 4.75 min) than the current investigation. Despite the relationship between $\dot{V}O_2$max and CP (Jenkins & Quigley, 1993; Moritani, Nagata, deVries, & Muro, 1981), reported increases in $\dot{V}O_2$max can occur without changes in CP during lower body high-intensity interval training (Graef et al., 2009; Jenkins & Quigley, 1993; Kendall et al., 2009). Gaesser and Wilson (1988) suggested that changes in CP are not dependent on changes in $\dot{V}O_2$peak, and despite both measures purported to be measures of aerobic function, training-based improvements are not mutually exclusive. Since CP is a submaximal parameter of aerobic function (Moritani et al., 1981; Poole et al., 1990), and the current investigation found CP to be positively correlated with energy derived from the oxidative and ATP-PCr system, the maximal nature of SIT may have a greater impact on maximal (i.e.$\dot{V}O_2$peak) rather than submaximal measures.

Relatively few explorations have determined the influence of high-intensity intermittent exercise on W' derived from the work-time relationship with divergent findings (Jenkins & Quigley, 1993; Poole et al., 1990). Moreover, previous studies demonstrate conflicting reports on the relationship between anaerobic performance (TW, PP and MP), from upper and lower body Wingate tests, and W' (Bulbulian, Jeong, & Murphy, 1996; Jenkins & Quigley, 1993; Zagatto, Papoti, & Gobatto, 2008). Hence, some investigations argue the validity of W' as an estimation of anaerobic capacity (Dekerle, Sidney, Hespel, & Pelayo, 2002; Poole, Burnley, Vanhatalo, Rossiter, & Jones, 2016). Although, the current investigation did find significant correlations among W' and anaerobic performance (PP, MP, and TW) with no changes in either following SIT. Only the energy derived from glycolysis was related to W' demonstrating that W' may be related to fatigue-related metabolites and glycogen stores (Black et al., 2017; Jones, Wilkerson, DiMenna, Fulford, & Poole, 2008; Miura, Sato, Sato, hipp, & Fukuba, 2000). Future research is needed to clearly define the components within the work-time relationship for the upper body; however, the current findings support that W' is a representation of anaerobic capabilities.

Sprint interval training and HIIT using the lower body has shown to increase motor unit recruitment and delay the onset of neuromuscular fatigue over four and six weeks, respectively (Creer et al., 2004; Smith et al., 2009). An increase in EMG_{FT} may reflect improvements in muscle buffering capacity and may be sensitive to central and peripheral adaptions (Moritani et al., 1993; O'Leary, Collett, Howells, & Morris, 2017). In the current study, SIT did not delay neuromuscular fatigue in the biceps brachii via EMG_{FT}, which coincides with lack of improvements following SIT on any of the other anaerobic parameters measured. Previous studies have reported no training related increases in muscle activation after lower body sprint

training (Sleivert, Backus, & Wenger, 1995). This may be due to the incorporation of many muscles, including antagonists, during upper body arm cranking and this load sharing may inhibit an adequate activation response from a single muscle (Hug, Nordez, & Guével, 2009; Lusina et al., 2008). Stimulation of the type II fibers are evident above EMG_{FT}, but SIT may not induce a greater expression of type I, IIa, or IIx in the upper body after two weeks (Zinner et al., 2016), which may partially explain the lack of change in the onset of neuromuscular fatigue in the current study. Furthermore, training at the fast speeds (i.e. all-out), such as those utilized in the SIT protocols, may not evoke greater motor unit activation at slow speeds (i.e. 50RPM) (Cormie, McGuigan, & Newton, 2011), such as those utilized in the constant work-rate trials. Lastly, this investigation observed a significant intra-individual variation of EMG response in certain subjects, which may also be related to the slow constant-load crank rate (Foss & Hallén, 2005; Takaishi, Yasuda, Ono, & Moritani, 1996).

During the investigation, determination of HHB_{BP} was not possible because approximately 43% of the trials displayed non-sigmoidal patterns where a plateau could not be credibly established. This may again be due to the slow crank rate during the GXT which can exert a greater isometric component of grip force, especially at the higher relative workloads during the GXT, and may impede blood flow (Smith, Price, & Doherty, 2001; Tschakovsky, Sujirattanawimol, Ruble, Valic, & Joyner, 2002). Alternatively, a faster crank rate reduces the force required to pedal (Hagberg, Mullin, Giese, & Spitznagel, 1981) and may increase blood flow in the working muscles (Armstrong & Peterson, 1981; Takaishi et al., 1996). In addition, Price and colleagues (2007) found that arm cranking at slower rates (50 RPM) versus faster rates (70 and 90 RPM) elicited lower oxygen consumption, earlier onset of fatigue, and greater range of motion from the trunk down. This may further support the potential variations in the engaged

muscular that may have occurred during the GXT despite attempting to control for any extraneous movements. The authors suggested that a slow crank rate could induce longer and more forceful contractions causing a greater force requirement owing to pre-mature exhaustion prior to maximal oxygen supply and delivery (Price et al., 2007).

The variability within individual response to SIT has warranted investigation (Astorino & Schubert, 2014; Bonafiglia et al., 2016; Gurd et al., 2016). It has been shown that higher rates of non-responders occur at a frequency below four days per week (Gurd et al., 2016). In a meta-analysis by Bacon and colleagues (2013) it was found that longer high-intensity work bouts have a more profound positive effect on $\dot{V}O_2$max. Furthermore, Ross and colleagues (2015) showed that increasing the amount of exercise at high intensities reduced the number of non-responders, which would increase the amount of work completed and help to explain the increases in $\dot{V}O_2$peak and PPO for the 30 s group. In the current study, most of those classified as responders to SIT showed favorable improvements according to changes in either $\dot{V}O_2$peak, CP, or $EMG_{FT;}$ however, they were not mutually exclusive. Similarly, Bonafiglia and colleagues (2016) observed individual patterns of response showing increases in $\dot{V}O_2$peak without related increases in lactate threshold. Thus, individuals can be considered responders in one variable, but not in another (Bonafiglia et al., 2016; Gurd et al., 2016). Given the demanding physical exertion of SIT, it is susceptible to individual effort and fatigue rates; therefore, it is difficult to ascertain if a subject put forth their greatest effort every visit on each sprint (Gurd et al., 2016). The effect of training is influenced by the highly variable amount of metabolic stress during 'all-out' exercise (Mann, Lamberts, & Lambert, 2014), thereby potentially limiting to the ability to tolerate critical values of metabolite accumulation (Foster et al., 2004). Therefore, individuals may restrain themselves during repeated 'all-out' efforts to avoid critical metabolic levels as a type of neural

control regulator (Foster et al., 2004; Gibson, Lambert, & Noakes, 2001). Perhaps future studies should implement strategies that quantify metabolic stress during training to ensure whether their participants are working maximally.

In contrast to previous investigations on anaerobic performance following lower body SIT (Astorino, Allen, Roberson, & Jurancich, 2012; Bayati, Farzad, Gharakhanlou, & Agha-Alinejad, 2011; Burgomaster et al., 2006; Harmer et al., 2000; Hazell et al., 2010; Zinner et al., 2016), two-weeks did not elicit improvements in upper body PP, MP, or TW. Although relative to lean arm mass, 30:4 was the only group significantly greater than CON in terms of MP. For example, Zinner and colleagues (2016) found greater PP and MP in the upper body over two-weeks of combined upper and lower body SIT (30:4) despite no changes in oxidative enzyme activity, muscle glycogen content, proportion of muscle fiber types, or cross sectional area of the triceps brachii. Additionally, the authors did not implement a control group to account for a training effect and all participants performed upper body SIT alongside lower body SIT (Zinner et al., 2016). Furthermore, Zelt and colleagues (2014) observed a training effect in PP and MP for two SIT protocols (30 s vs. 15 s work bouts), but were unable to differentiate between groups over a four-week lower body SIT intervention. Alternatively, previous studies have reported increases in $\dot{V}O_2$max and PPO with no increases in anaerobic performance following a two-week lower body cycling intervention (Rodas et al., 2000) or minimal increases following a four-week running-based SIT intervention (McKie et al., 2017). Hazell and colleagues (2010) observed increases in PP and MP in both 30:4 and 10:4 protocols over two weeks in the lower body, whereas our current findings did not find the 10:4 protocol to be beneficial for increasing upper body MP. The authors attributed the initial production of power output to SIT adaptations; however, our results concluded that total work completed during training was more crucial for

the improvements seen in this investigation. Interestingly, Hazell and colleagues (2010) implemented a load equivalent to 10% of the subject's body weight (kg) during training, which is greater than the traditional 7.5% recommended for a Wingate test. Astorino and colleagues (2012) found significant improvements in PP and MP despite no change in muscular force production. The mechanisms behind positive anaerobic adaptations in the upper body are unclear given the lack of changes in muscle morphology (including muscle fiber types and cross sectional area), muscle enzymes, or glycogen content (Zinner et al., 2016). Lastly, perhaps a greater resistance during repeated efforts (Forbes, Kennedy, Boule, & Bell, 2014) or a longer SIT intervention is needed to further stimulate anaerobic performance in the upper body.

Energy System Contribution

This appears to be the first investigation comparing the influence of SIT on energy system contribution. Following the intervention, energy derived from the ATP-PCr system was greater in 10:4 than CON; however, glycolytic and oxidative energy was not different. In contrast, SIT did not alter energy expenditure or relative contribution in 10:2 or 30:4 compared to CON.

It has been reported that sprint training can increase resting PCr concentrations (Parra, Cadefau, Rodas, Amigo, & Cusso, 2000; Rodas et al., 2000). During lower body cycling, a 10 s sprint can reduce resting PCr concentrations by ~55% (Bogdanis, Nevill, Lakomy, & Boobis, 1998), whereas a 30 s sprint can reduce PCr concentrations by 55-83% (Bogdanis, Nevill, Boobis, & Lakomy, 1996; Parra et al., 2000). After a 30-s lower body sprint, PCr concentrations can be replenished by ~47% in two minutes and ~76% in four minutes of resting values (Bogdanis et al., 1996). In conjunction with a reduction in PCr concentrations, levels of muscle

pH can drop considerably after one 30 s sprint with further reductions in subsequent sprints (Bogdanis et al., 1996). In this investigation, 30:4 was perceived as more challenging than the 10 s protocols via PRR. Participants within 30:4 had the largest work-to-rest ratio with moderate exercise density which likely resulted in greater blood lactate concentrations (Little & Williams, 2007). These metabolic conditions were likely the cause of lower average PP, MP, and PRR values in 30:4 compared to the two 10 s protocols over the course of training. Therefore, given that 10:4 had the lowest ED, the associated metabolic stress may have allowed for greater resynthesis of PCr and greater removal of lactate during training, thereby increasing ATP-PCr utilization.

Chronic training adaptations are suggested to influence the unique energy system profiles of sprinters and endurance athletes (Gastin, 2001). During an 'all-out' 10-s sprint in the lower body, aerobic energy yield is ~13%, whereas glycolysis may yield within 55 to 75% of the total metabolic energy (Bogdanis et al., 1998). Meanwhile, work-to-rest ratios of ~1:6 have been suggested to develop the ATP-PCr system (Bompa & Haff, 2009). Although the SIT protocols used in this study employed work-to-rest ratios smaller than 1:6 [10:4 (1:24), 10:2 (1:12), and 30:4 (1:8)], the physiological demands of repeated 'all-out' sprinting can rapidly induce fatigue despite a constant work-to-rest ratio (Abt, Reaburn, Holmes, & Gear, 2003). In addition, 10:4 had the lowest ED, while 10:2 had the greatest ED, indicating the least and greatest amount of sessional intensity, respectively. Therefore, based upon performance and intensity metrics, 10:4 appeared to be the ideal protocol for developing energy yield from the ATP-PCr system.

There does not appear to be a consensus within the literature on energy yield over the course of a 30 s Wingate test in the upper body. The current investigation calculated on average,

~9-11% oxidative, ~40-46% glycolytic, and ~43-50% ATP-PCr. In contrast, Harvey and colleagues (Harvey et al., 2015) reported ~11% oxidative, ~28% glycolytic, and ~60% ATP-PCr in the upper body, however the authors recruited sedentary individuals and used an alternative method of calculating the energy derived from ATP-PCr. Previous investigations reported oxidative and glycolytic energy contribution to contribute 21% and 46% (Franchini et al., 2016), respectively, and ~11% and ~60% (Lovell et al., 2013), respectively, of total metabolic energy in a 30 s upper body sprint. However, discrepancies in relative contribution between our study and previous investigations (Franchini et al., 2016; Lovell et al., 2013) may lie within the training experience of the recruited participants, sensitivity of the instruments utilized, and mechanical versus electromagnetically braked ergometers. Further research is needed to establish energy system requirements within the upper body.

Conclusions

This investigation offers a novel examination of the upper body with traditional and modified SIT and shows that the traditional protocol (30:4) elicits positive aerobic performance in a relatively short-training period. However, the current findings were unable to support the underlying mechanism of aerobic improvement with an examination of local oxygen delivery. Despite this limitation, the total amount of active muscle mass during upper body cycling is difficult to quantify while taking into consideration the lower limb and trunk stabilization. In order to better account for these discrepancies participants may need to be restrained to avoid any undesired movement that can influence the outcome variables.

In conclusion, larger work-to-rest ratio SIT protocols induce enhanced aerobic adaptions in the upper body over a short-term two-week intervention. However, there was no improvement

in submaximal performance as denoted by fatigue thresholds or anaerobic performance via Wingate assessment. A faster crank rate may have increased blood flow and increased muscle efficiency to determine fatigue thresholds more clearly. This is also the first study to indicate that a smaller work-to-rest ratio SIT protocol may enhance ATP-PCr utilization during an anaerobic exercise bout. Perhaps future studies should investigate the progression of larger work-to-rest ratios in order to increase ED rather than increasing the number of repetitions.

Practical Applications

Upper body SIT training may provide a time-efficient form of exercise that provides similar health benefits as traditional endurance training. Metabolic health may be maintained despite the mode in which SIT is performed (Francois et al., 2017). In fact, SIT has been shown to positively influenced individuals with cardio-metabolic disorders (Gibala, Little, MacDonald, & Hawley, 2012; Gillen et al., 2016; Hicks et al., 2003; Maire et al., 2004). However, the intensity and duration may be too stressful for the general and clinical populations (Bayati et al., 2011); therefore, shorter work bouts may serve as precursors to larger work-to-rest ratios as progression ensues. On the other hand, the performance benefits of SIT may benefit upper body dominant athletes (e.g. sailing, kayaking, cross-country skiing, judo, wrestling or paralympic) or individuals enduring acute lower body injuries. Therefore, upper body SIT may be advantageous for athletes, or for those who may not be able to partake in more traditional forms of exercise training.

APPENDIX: UCF IRB LETTER

University of Central Florida Institutional Review Board
Office of Research & Commercialization
12201 Research Parkway, Suite 501
Orlando, Florida 32826-3246
Telephone: 407-823-2901 or 407-882-2276
www.research.ucf.edu/compliance/irb.html

Approval of Human Research

From: **UCF Institutional Review Board #1**
FWA00000351, IRB00001138

To: **Michael B. La Monica** and Co-PI: **David Fukuda**

Date: **August 22, 2017**

Dear Researcher:

On 08/22/2017 the IRB approved the following human participant research until 08/21/2018 inclusive:

Type of Review:	Submission Response for UCF Initial Review Submission Form Expedited Review
Project Title:	Examining work-to-rest-ratios to optimize upper body interval training
Investigator:	Michael B. La Monica
IRB Number:	SBE-17-13210
Funding Agency:	
Grant Title:	
Research ID:	N/A

The scientific merit of the research was considered during the IRB review. The Continuing Review Application must be submitted 30days prior to the expiration date for studies that were previously expedited, and 60 days prior to the expiration date for research that was previously reviewed at a convened meeting. Do not make changes to the study (i.e., protocol, methodology, consent form, personnel, site, etc.) before obtaining IRB approval. A Modification Form **cannot** be used to extend the approval period of a study. All forms may be completed and submitted online at https://iris.research.ucf.edu .

If continuing review approval is not granted before the expiration date of 08/21/2018, approval of this research expires on that date. When you have completed your research, please submit a Study Closure request in iRIS so that IRB records will be accurate.

Use of the approved, stamped consent document(s) is required. The new form supersedes all previous versions, which are now invalid for further use. Only approved investigators (or other approved key study personnel) may solicit consent for research participation. Participants or their representatives must receive a copy of the consent form(s).

All data, including signed consent forms if applicable, must be retained and secured per protocol for a minimum of five years (six if HIPAA applies) past the completion of this research. Any links to the identification of participants should be maintained and secured per protocol. Additional requirements may be imposed by your funding agency, your department, or other entities. Access to data is limited to authorized individuals listed as key study personnel.

In the conduct of this research, you are responsible to follow the requirements of the Investigator Manual.

On behalf of Sophia Dziegielewski, Ph.D., L.C.S.W., UCF IRB Chair, this letter is signed by:

Signature applied by Patria Davis on 08/22/2017 10:54:09 PM EDT

IRB Manager

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