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Physiological and Biomechanical Factors of Cycling Performance

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Physiological and Biomechanical Factors of Cycling Performance

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Dedication

To my wife, Miriam. For all of your love, patience, and support.

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Abstract

Physiological and Biomechanical Factors of Cycling Performance

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The two primary predictors of cycling performance are maximal oxygen consumption and lactate threshold. However, several physiological and biomechanical factors influence these variables. The purpose of study one was to investigate relative joint contribution, muscle activation, and muscle oxygenation differences between high (HLT) and low (LLT) lactate threshold cyclists with similar maximal oxygen consumption capabilities (VO_{2max}). While there were no differences in muscle oxygenation, the HLT group had greater relative hip contribution at 90% of VO_{2max} compared to the LLT group, as well as decreased vastus medialis EMG activation during exercise at 60 and 70% of VO_{2max} ($p<0.05$). These findings suggesting the HLT cyclists place a greater emphasis on the hip compared to the knee joint to generate power while cycling. The purpose of study two was to investigate the effects of short-term maximal power training on cycling peak oxygen consumption ($VO_{2peak\text{-}cycling}$) in non-cyclists. Over the course of 5 days, the training group performed 10 maximal sprints a day each lasting ~4 seconds with two-minutes rest between each sprint. This protocol was designed to maximize recruitment of muscles involved in cycling while

minimizing cardiovascular stress during training. Following training, absolute and relative $VO_{2peak\text{-}cycling}$ was 5.9 ± 1.6% and 5.6 ± 1.9% greater compared to pre-training ($p<0.05$), while in the control group $VO_{2peak\text{-}cycling}$ did not change ($p>0.05$). The improvement in $VO_{2peak\text{-}cycling}$ was accompanied by a 6.3 ± 2.5% increase (Pre: 228 ± 18 W vs. Post: 242 ± 19 W) in peak work rate achieved during post-testing in the training group ($p<0.05$). This suggests that $VO_{2peak\text{-}cycling}$ can be increased through maximal power training in non-cyclists likely as a result of increased ability to recruit additional muscle mass during intense cycling exercise. Taken together, these studies indicate that biomechanical muscle recruitment 'strategies' can influence both submaximal (i.e. LT_{VO2}) and peak oxygen consumption (i.e. $VO_{2peak\text{-}cycling}$) during cycling.

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Chapter I: General Introduction

Human endurance performance is governed by the highest rate of oxygen consumption that can be maintained for the duration of exertion. The upper limit to this rate of oxygen consumption is dictated by maximum oxygen consumption (VO_{2max}), and is a function of the exercise intensity at which lactate begins to accumulate in the muscle and then blood (i.e. lactate threshold). Improvements in oxygen delivery and muscle oxygen utilization allow athletes to exercise at higher rates of oxygen consumption at lactate threshold (LT_{VO2}).

Chronic endurance training elicits improvements in skeletal muscle oxidative capacity (Chi et al. 1983, Holloszy et al. 1984) which increases the relative and absolute exercise intensity at which lactate threshold occurs (Ivy et al. 1980). Although a majority of the increases in lactate threshold result from improved oxygen delivery and muscle oxidative capacity, previous research has suggested that lactate threshold may also be improved by distributing power production to a greater amount of active muscle (Coyle et al. 1988). In theory, by spreading the work to more muscle mass, the work performed by any individual muscle would be reduced thereby delaying the onset of lactate production and lactate threshold.

Active muscle mass plays an important role in achieving VO_{2max} while cycling. For example, individuals without cycling experience demonstrate ~10% lower VO_{2max} values while cycling (i.e. $VO_{2peak)}$ than those achieved during treadmill VO_{2max} testing (Tanaka 1994). This lower $VO_{2peak\text{-}cycling}$ has been attributed to lower muscle mass recruitment in untrained individuals (Tanaka et al. 1987, Tanaka 1994, Sloniger et al. 1997, Sloniger et al. 1997). Greater active muscle mass during exercise is related to greater maximal oxygen consumption (Sloniger et al. 1997). Having subjects stand up towards the end of maximal exercise cycling results in higher values of maximal oxygen consumption compared to seated cycling which can be attributed to an increased

recruitment of active muscle mass (Tanaka et al. 1987). The mechanism driving this relationship has been attributed to increases in peak cardiac output from more active muscle tissues receiving oxygenated blood (Reybrouck et al. 1975). Therefore, we sought to determine whether $VO_{2peak\text{-}cycling}$ could be improved in non-cyclists, specifically through short-term inertial load training designed to elicit maximal muscle recruitment and neuromuscular power while minimizing cardiovascular stress.

Muscle deoxygenation is reflective of the metabolic stress of a particular muscle group during exercise (Ryan et al. 2014, Skovereng et al. 2016, Skovereng et al. 2016). Previous research has shown reduced stress, as reflected by less glycogen depletion, on the vastus lateralis of well-trained cyclists with high lactate thresholds compared with equally well-trained cyclists with relatively low lactate thresholds during submaximal cycling (Coyle et al. 1988). Accompanying this reduced stress was an estimated ~2kg increase in the estimated total muscle mass used during cycling in the high lactate threshold cyclists. Authors attributed the reduction in knee extensor glycogen utilization to a better spreading of the metabolic work across more muscle mass and multiple power generating joints (i.e. hip and knee), however, direct investigations are needed to test this hypothesis.

Therefore, the primary objectives of this dissertation are to determine 1) whether biomechanical differences occur between highly trained cyclists with high and low LT and if these differences are associated with changes in deoxygenation of the vastus lateralis during submaximal cycling and 2) whether short-term inertial load power training increases $VO_{2peak\text{-}cycling}$ and reduces stress on the vastus lateralis during submaximal exercise reflective of a possible 'learned ability' to recruit additional muscle while cycling.

A variety of biomechanical and physiological techniques were implemented to investigate

the cycling strategy used by well-trained cyclists. Techniques included electromyography (EMG) to determine relative muscle activation and near-infrared spectroscopy (NIRS) to assess changes in deoxygenation of the vastus lateralis during submaximal cycling in HLT and LLT cyclists. Kinematics (i.e. motion analysis) and kinetics (i.e. pedal forces) were used to determine the relative contribution of the hip, knee, and ankle joints to the cycling movement. The unique combination of local physiological stress (i.e. NIRS) and biomechanical data will provide a detailed description of the cycling stress and technique in well-trained cyclists (HLT) and whether it reduces the deoxygenation of knee extensors. These data will describe the cycling biomechanics of athletes with high lactate thresholds who are capable of exercising at higher steady state rates of energy expenditure.

Furthermore, short-term inertial-load power training designed to elicit maximal neuromuscular improvement was used to investigate whether improvements in $VO_{2peak\text{-}cycling}$, $LT_{VO2\text{-}cycling}$, and muscle oxygenation can be made without significant cardiovascular stress during training. By utilizing this training technique on subjects who fail to achieve VO_{2max} when cycling but rather only obtain $VO_{2peak\text{-}cycling,}$ elicited improvements to cycling maximal and submaximal performance can occur independent of aerobic exercise training or possible changes in the maximal values of cardiac output and/or A-VO_{2diff}. We hypothesize that inertial-load power training will increase $VO_{2peak\text{-}cycling}$, $LT_{VO2\text{-}cycling}$, and lower deoxygenation of the vastus lateralis.

Together, these two studies attempt to investigate the adaptations other than cardiovascular or mitochondrial function of cycling performance, specifically, those possible biomechanical improvements that might contribute to improved VO_{2peak} and LT_{VO2}.

Chapter II: Statement of Problem

Classically, the limiting factors of VO_{2max} and LT_{VO2} have been examined from a systems physiology perspective with an emphasis on the relationship between the maximal capabilities of cardiac output and skeletal muscle oxidative capacity. However, the variation in both VO_{2max} and LT_{VO2} in cyclists can be attributed to other factors other than oxygen delivery and oxidative capacity. Therefore, the following studies were designed to determine novel determinants of cycling endurance performance (i.e. VO_{2max} and LT_{VO2}). This series of studies answers the following specific questions:

1. Do individuals with similar VO_{2max}, but differing cycling LT_{VO2} (i.e. high and low lactate threshold) differ in relative joint contribution, muscle oxygenation, and muscle activation during submaximal cycling?
2. Does short-term inertial-load power training lead to improvements in $VO_{2peak\text{-}cycling}$, $LT_{VO2\text{-}cycling}$, and muscle oxygenation in individuals without cycling experience?

Chapter III: Purpose and Hypothesis

Study #1: The purpose of this study was to examine the physiological (as assessed by NIRS) and biomechanical (as assessed by absolute/relative joint power contributions) differences between high LT_{VO2} (HLT) and low LT_{VO2} (LLT) cyclists across work rates in competitive cycling. We hypothesized that compared with LLT cyclists, HLT cyclists will exhibit greater relative power contribution from the hip and lower relative contribution from the knee during submaximal cycling resulting in lower physiological stress as evidenced by lower deoxygenation and EMG activation on the knee extensors.

Study #2: The purpose of this study was to examine changes in $VO_{2peak\text{-}cycling}$ and $LT_{VO2\text{-}cycling}$ after a short-term, inertial-load maximal power cycling training program that maximized neuromuscular power and minimized cardiovascular stress. We hypothesized that recreationally trained individuals would demonstrate improved cycling $VO_{2peak\text{-}cycling}$, $LT_{VO2\text{-}cycling}$, and reduced muscle deoxygenation of their knee extensors.

Chapter IV: Study #1

RELATIVE LOWER EXTREMITY JOINT POWER AND MUSCLE DEOXYGENATION IN HIGH AND LOW LACTATE THRESHOLD CYCLISTS

Abstract

Background: The physiological and biomechanical differences between high LT_{VO2} (HLT) and low LT_{VO2} (LLT) cyclists have yet to be completely described. Therefore, the objective of the present study is to investigate whether, compared with low LT_{VO2} cyclists, high LT_{VO2} cyclists are able to reduce the stress on the knee extensor muscles and relative joint contribution by increasing the relative contribution of the hip extensor muscles during high intensity cycling.

Methods: Sixteen well-trained endurance athletes completed cycling and running VO_{2max} and cycling and running lactate threshold (LT_{VO2}) testing, and were separated into two groups based on cycling LT_{VO2} (HLT: n=8) and (LLT: n=8). During submaximal cycling (60-90% VO_{2max}), near-infrared spectroscopy (NIRS) measured oxygenated hemoglobin (O_2Hb), deoxygenated hemoglobin (HHb), %saturation, and total hemoglobin (THb) in the vastus lateralis; EMG measured activity of muscles in the lower extremity; and hip, knee, and ankle absolute and relative joint powers (the percent contribution to total joint powers) were compared between groups.

Results: Sixteen subjects were separated into two groups based on cycling LT_{VO2}: HLT (n=8) and LLT (n=8) with similar VO_{2max} between groups. Blood lactate concentration increased with work rate and was lower in the HLT group at 80 and 90% of VO_{2max} ($p<0.05$). Groups did not differ in O_2Hb, HHb, or THb ($p>0.05$). Vastus medialis activation was higher in the LLT group at 60 and 70% VO_{2max} ($p<0.05$). There were no differences between groups in either hip, knee, or ankle absolute joint specific power across work rates ($p>0.05$), but relative hip contribution was significantly greater in the HLT group at 90% VO_{2max} compared to the LLT group ($p<0.05$).

Conclusion: HLT cyclists have a greater relative hip contribution during submaximal cycling and reduced stress on the knee extensors (i.e. lower VM activation) compared to LLT cyclists.

Introduction

The mechanisms surrounding maximal aerobic capacity (VO_{2max}) and lactate threshold (LT_{VO2}) are a complex combination of cardiovascular, muscular, and neural adaptations that improve oxygen delivery and muscle oxidative capacity (Holloszy et al. 1984, Bassett et al. 2000). Typically, VO_{2max} and LT_{VO2} are specific to the mode of exercise used in training (Millet et al. 2009). For example, well-trained cyclists have high LT_{VO2} capabilities while cycling (Coyle et al. 1988), but runners and triathletes typically have lower LT_{VO2} while cycling compared with running uphill (Millet et al. 2009).

When comparing cycling and inclined treadmill running in well-trained cyclists, two groups have emerged in prior research: 1) those cyclists with equally high LT_{VO2} while cycling and running uphill and 2) cyclists with low LT_{VO2} while cycling, but high LT_{VO2} while running uphill (closely matching those of the high LT_{VO2} cyclists) (Coyle et al. 1988). Traditionally, mitochondrial enzyme activity/skeletal muscle oxidative capacity would explain differences in LT_{VO2} between groups of cyclists; however, mitochondrial enzyme activity was found to be similar between these two groups suggesting the low LT_{VO2} cyclists have a capacity for high LT_{VO2} that is not attained during cycling. Previous research investigating physiological measures as possible causes found no evidence of glucose or glycogen utilization nor oxidative metabolism as the driving mechanism for these differences. Moreover, the high LT_{VO2} used approximately 1.8 kg more calculated muscle mass during cycling compared with low LT_{VO2} cyclists despite having similar VO_{2max} (Coyle et al. 1988, Coyle 1995). The authors attributed the higher LT_{VO2} to the improved ability to spread the metabolic work to a greater amount of muscle mass effectively increasing the number of mitochondria sharing in the metabolic work. Others have speculated this

could be accomplished, at least in part, by specific technical adaptations, such cycling skill and/or muscle utilization patterns (Millet et al. 2009).

Experienced cyclists tend to reduce the stress on their knee extensors and generate more force on the down-stroke of pedaling, presumably through increased joint contribution from the hip extensors (Coyle et al. 1988, Coyle et al. 1991, Takaishi et al. 1998). Direct comparisons of relative joint contribution between experienced and novice cyclists (Hoshikawa 2007, Bini et al. 2014, Aasvold 2017) have found inconsistent results, with some studies finding higher (Aasvold 2017), lower (Hoshikawa 2007), or no difference in relative hip contribution (Bini et al. 2014). The discrepancy in these results are likely a result of the work rates chosen for comparison, as prior studies have made relative joint contribution comparisons at different absolute and relative work rates and/or relative exercise intensities. To date no study has compared, between high and low LT_{VO2} cyclists with similar VO_{2max}, the relative joint contribution at similar absolute and relative work rates commonly attained during high level competitive cycling.

Although absolute and relative joint powers calculated through inverse dynamics have often been used interchangeably to describe muscular power, these joint power calculations are independent of the mechanical energy expenditure of working skeletal muscle during exercise and differ from individual muscle contribution (Kautz et al. 1994, Neptune et al. 1998, Kautz et al. 2002). Electromyography (EMG) allows for the assessment of muscle activation and can be used to determine relative intensity of muscle activation and patterns of muscle recruitment. Prior research has shown increased muscle EMG activation of the vastus lateralis in novice compared with experienced cyclists (Takaishi et al. 1998). Additionally, near-infrared spectroscopy (NIRS) can assess the oxygenation of working muscle by calculating oxygenated (O_2Hb) and deoxygenated (HHb) hemoglobin. Changes in HHb in response to changes in work rate and

cadence during cycling have been used as indicators of local muscle stress (Boone et al. 2015, Skovereng et al. 2016, Skovereng et al. 2016)and, in healthy adults, decreased relative knee contribution at higher work rates is accompanied by a plateau in deoxygenation of the vastus lateralis. However, changes in deoxygenation have not been used to compare the physiological stress of the working muscle between low and high LT_{VO2} cyclists.

Therefore, the objective of the present study is to investigate whether, compared with low LT_{VO2} cyclists, high LT_{VO2} cyclists are able to reduce the stress on the knee extensor muscles and relative joint contribution by increasing the relative contribution of the hip extensor muscles during high intensity cycling. We propose to examine the physiological (as assessed by NIRS and EMG) and biomechanical (as assessed by absolute/relative joint contributions) differences between high LT_{VO2} (HLT) and low LT_{VO2} (LLT) cyclists. We hypothesized that compared with LLT cyclists, HLT cyclists will exhibit greater relative hip power contribution and lower relative knee power contribution during submaximal cycling resulting in lower physiological stress as evidenced by lower deoxygenation and EMG activation of the knee extensors.

Methods

Fifty-two well-trained endurance athletes were originally recruited to take part in this research study. Those who did not obtain similar running and cycling VO_{2max} values were excluded (cycling VO_{2max} no less than 0.2 L/min compared with treadmill VO_{2max}). The remaining 16 well-trained endurance athletes were separated into two groups: High LT_{VO2} (HLT, n=8) and Low LT_{VO2} (LLT, n=8) based on cycling LT_{VO2} (HLT: cycling LT_{VO2} no less than 0.2L/min compared with treadmill LT_{VO2}) while controlling for cycling VO_{2max} to ensure no differences between groups in maximal oxygen consumption capabilities.

A total of 6 testing visits were required. Visit 1 measured VO_{2max} while cycling and Visit 2 measured VO_{2max} while running uphill. Visit 3 was used to determine LT_{VO2} while cycling and Visit 4 was used to determine LT_{VO2} while running. Visit 5 and 6 measured muscle deoxygenation, muscle activation, and joint specific power during submaximal cycling.

Maximal Oxygen Consumption While Cycling and Running

Subjects exercised for 8-12 minutes with increasing intensity until exhaustion, as described previously (Costill 1970). Briefly, subjects exercised for four minutes at a moderate intensity (~75% VO_{2max}) and thereafter work rate was progressively increased every two minutes until exhaustion. During the cycling test, wattage was increased on the cycle ergometer (Excaliber Sport, Lode, Groningen, The Netherlands). During the treadmill test, subjects ran at a constant, speed which elicited ~75% of VO_{2max}. After 4 minutes, grade was increased to 4% and increased 2% every 2 minutes thereafter. Subjects typically reached VO_{2max} at a grade of 8-10%. For both cycling and running, a successful VO_{2max} test was determine based on ACSM criteria: RER>1.1, Max Heart Rate ± 10 bpm of predicted max heart rate (Tanaka et al. 2001), plateau of ≤ 150 mL/min VO_2, and an RPE >17 (Pescatello et al. 2014). Heart rate was measured continuously by a monitor worn around the chest (Suunto, Vantaa, Finland).

Respiratory analyses were determined using oxygen and carbon dioxide analyzers (Applied Electrochemistry, Models S-3A/I and CD-3A, respectively) while the participants breathed through a one-way valve (Hans Rudolph, Kansas City, MO). Ventilation was measured via an inspiratory pneumotachometer (Hans Rudolph, Kansas City, MO). VO_2, VCO_2, and RER was continuously monitored throughout the exercise test. The highest 30 second average of VO_2 was used as the measurement of VO_{2max}.

Lactate Threshold Testing

This protocol required 30 minutes of continuous exercise at submaximal intensities (5 min at each stage of approximately 40, 50, 60, 70, 80, 90% of maximal oxygen consumption). During cycling test, wattage was adjusted to increase work rate and cadence was maintained at 80-100 RPM. However, during treadmill testing subjects ran at a constant 10% grade and speed was increased (from a walk ~80 m/min to a jog/run ~215 m/min) to increase work rate as uphill running has been shown to use similar muscles as cycling (Costill et al. 1974). Lactate threshold was determined from analysis of a series of blood samples obtained between minutes 4 and 5 in each stage. Blood samples were immediately deproteinized by placing in a 10% perchloric acid solution and lactic acid levels were measured on the supernatant. Enzymatic analysis was used to determine blood lactate concentration, as described previously (Farrell et al. 1979, Coyle et al. 1988). The lactate threshold was defined as the exercise intensity that elicits a 1mM increase above baseline in blood lactate concentration (Coyle et al. 1988, Coyle et al. 1991). VO_2, VCO_2, and RER were continuously monitored throughout the exercise test and were averaged over the final 4 and 5 minutes of each stage. Heart rate was measured continuously by a monitor worn around the chest and taken as the average during minutes 4 and 5 of each stage (Suunto, Vantaa, Finland), during this time subjects were also asked to report RPE.

Muscle Deoxygenation

Near-infrared spectroscopy (OxiplexTS, ISS, Champaign, IL) was used to measure oxygenated hemoglobin [O_2Hb] and deoxygenated hemoglobin [HHb] in the vastus lateralis. NIRS uses the feature that the chromophores of O_2Hb and HHb have different optical properties of absorbing near-infrared (wave length: 690 nm, 830 nm). This enabled NIRS to measure the absolute concentrations of O_2Hb and HHb in μM in real-time in a noninvasive manner (Ryan et al. 2012, Boone et al. 2015, Skovereng et al. 2016). O_2Hb and HHb serve as markers of

'physiological stress' and were compared between groups. The O_2Hb and HHb data during the last 1 minute of each stage were averaged and compared as a %change from resting values.

Electromyography

Bagnoli™ Desktop EMG system (Delsys INC., Natick, MA) was used to determine muscle activity of the: gluteus maximus (Gmax), rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF), gastrocnemius (Gast), soleus (Sol), and tibialis anterior (TA). Skin sites were cleaned and shaved with an alcoholic prep wipe and disposable razor. Electrodes were placed along the skin surface to run in parallel with the skeletal muscle fibers, and secured with double sided tape and athletic wrap. Raw EMG signals were smoothed using a fourth-order, band-pass Butterworth filter with a frequency range set between 20 and 500Hz. Onset and offset of EMG activity were determined when the signal with an amplitude above two standard deviations beyond the mean of the quiescent phase between EMG bursts (Diefenthaeler et al. 2012). All EMG data were visually inspected and data manually selected to determine periods of quiescence before and after EMG bursts. EMG data were normalized as the percentage of the highest value which occurred during testing.

Joint Specific Powers

Subjects exercised on a stationary cycle ergometer while wearing 3M reflective markers. The markers were secured to the subjects using double-sided tape and placed on 9 sites (acromion process, mid-axillary, greater trochanter, mid-femur, lateral epicondyle of knee, mid-shank, lateral malleolus, toe of shoe, and heel of shoe). The Vicon Nexus motion analysis system uses infrared technology to collect data on the position of the markers in a 3-dimensional space and provides coordinates that can later be used for data processing and analysis (Vicon Motion Systems Ltd., Lake Forest, CA). Normal and tangential components of force applied to the pedal were collected

using a custom-designed force pedal with two piezo electric force transducers (Kistler, model 9251AQ01) at a sampling rate of 1200Hz. Pedal forces were filtered by a third-order low-pass Butterworth filter with a cutoff frequency of 10Hz (Coyle et al. 1991, Diefenthaeler et al. 2012). Data were paired to match the kinematic data obtained through motion analysis. Using the pedal angle obtained in the coordinate data, forces were transformed from the reference frame of the pedal into the inertial reference frame. Data were used for inverse dynamics calculations that determined the joint moments/power from the hip, knee, and ankle (Hull et al. 1985, Skovereng et al. 2016, Skovereng et al. 2016). Data were processed using a custom MatLab (MathWorks Inc., Natick, MA, USA) program.

Joint powers at the hip, knee, and ankle were derived using standard inverse dynamics techniques (Hull et al. 1985). Rigid segment models of the crank, foot, leg, and thigh were generated. Hip position was determined based on the location of the anterior superior iliac spine assuming constant offset measured during the static trials (Neptune et al. 1995). Linear and angular velocities and accelerations of the limb segments were determined by finite differentiation of position data with respect to time. Segmental mass proportions, center of mass locations, and radii of gyration were estimated from anthropometric tables (de Leva 1996). Joint moments of the ankle, knee, and hip were calculated through use of angular accelerations of the segments, normal and tangential pedal forces, and acceleration of segmental center of gravity. Joint powers were then calculated by the product of joint angular velocities and joint moments. Relative joint contribution was then calculated as the percentage contribution to the total joint powers.

Statistical Analysis

Descriptive statistics are reported as Mean ± SE. Descriptive statistics were compared using Students t-test (α= 0.05). Between group differences in VO_{2max} and lactate threshold were determined by Two-Way ANOVA (Group X Exercise Mode). Differences in muscle oxygenation, muscle activation, and joint contribution during submaximal cycling were determined through two-way repeated measures ANOVA (Group X Work Rate). Least Significant Difference post hoc comparison were performed for all significant main effects (α= 0.05).

Results

Subject Characteristics

There was no difference in age (HLT: 30.3 ± 1.0 yrs.; LLT: 26.3 ± 3.0 yrs.), height (HLT: 175.4 ± 1.5 cm; LLT: 176.0 ± 2.7 cm) or weight (HLT: 76.1 ± 3.2 kg; LLT: 67.9 ± 2.4 kg) between groups (p>0.05).

Within Group Differences in VO_{2max} (i.e. Ergometer vs. Treadmill)

During maximal exercise, there were no differences in absolute or relative oxygen consumption, HR, RER, or RPE between cycling and running in the HLT group (p>0.05). Similarly, the LLT group did not differ in absolute or relative maximal oxygen consumption, RER, or RPE on the cycle ergometer compared with treadmill running (p>0.05); However, HR_{max} was significantly higher while cycling compared with treadmill running in the HLT group (Table 1).

Between Group Differences in VO_{2max} (i.e. HLT vs LLT)

During cycling VO_{2max} testing there were no differences between groups in absolute (L/min) or relative (mL/kg/min) oxygen consumption, RER, Watts, or RPE (p>0.05). However, cycling HR_{max} was higher in the HLT group compared with the LLT group (p<0.05). During treadmill VO_{2max} testing absolute oxygen consumption (L/min), HR_{max}, RER, or RPE did not differ

between groups. However, treadmill relative oxygen consumption (mL/kg/min) was lower in the HLT group compared with the LLT group ($p<0.05$) (Table 1).

Within Group Differences in LT_{VO2} and LT_{HR} (i.e. Bike vs Treadmill)

Within the HLT group there were no differences in LT_{VO2} or LT_{HR} while cycling compared with uphill running ($p>0.05$). However, in the LLT group, LT_{VO2} and LT_{HR} were significantly lower while cycling compared with uphill running ($p<0.05$) (Table 2).

Between Group Differences in LT_{VO2} and LT_{HR} (i.e. HLT vs LLT)

Oxygen consumption and work rate at lactate threshold were significantly higher in the HLT group compared with the LLT group ($p<0.05$) (Table 2 and Figure 1). However, there was no difference in cycling or running LT_{HR} between the HLT and LLT groups ($p>0.05$). Furthermore, no differences between groups were found for uphill running LT_{VO2} or LT_{HR} ($p>0.05$) (Table 2).

Submaximal Measures During Lactate Threshold Testing

Cycling

Blood lactate concentration increased with increasing work rate in both groups, with the HHLT group having lower blood lactate concentration while cycling at 80 and 90% of VO_{2max} compared with the LLT group ($p<0.05$) (Figure 1). Additionally, as work rate progressively increased oxygen consumption, HR, RPE, RER in both groups ($p<0.05$) with each successive increase in intensity ($p<0.05$). However, there were no differences between groups ($p>0.05$) (Figure 2).

Uphill Running

As work rate increased all physiological measures (i.e. blood lactate, oxygen consumption, HR, RPE, and RER) increased for both groups ($p<0.05$), with each successive increase in exercise

intensity. ($p<0.05$). However, there was no difference between groups in any measure ($p>0.05$) (Figure 3).

NIRS, EMG, and Joint Powers

Near-Infrared Spectroscopy

Percent change (%Δ) in %Saturation of oxygen within the vastus lateralis was not statistically different across work rates or between groups ($p>0.05$). However, difference in %Δ in THB, O_2Hb, and HHb increased with work rates ($p<0.05$) with no differences between groups ($p>0.05$) (Figure 4).

Electromyography

A summary of the EMG results can be found in Table 3. Briefly, there were no differences between LLT and HLT across work rates for the RF, BF, Gast, or TA in % Activation ($p>0.05$). However, VM activation was lower in the HLT group compared with the LLT group at 60 and 70% VO_{2max} with no differences at higher work rates ($p<0.05$). VL and Gmax activation increased with work rate ($p<0.05$). In addition, the average Sol activation was higher in the HLT group compared with the LLT group ($p<0.05$).

Joint Power

There were no significant differences between the HLT and LLT groups in either Hip or Knee joint specific power at any work rate ($p>0.05$) (Figure 4). However, in both the HLT and LLT groups, absolute hip joint specific power increased with increases in work rate ($p<0.05$). Knee joint specific power increased with increases in work rate in the LLT group ($p<0.05$), but did not change in the HLT group ($p>0.05$). Ankle joint specific power did not differ between groups ($p>0.05$).

Relative hip contribution was significantly greater in the HLT group at 90% VO_{2max} compared with the LLT group ($p<0.05$). Relative knee contribution was not significantly different between groups or across work rates ($p>0.05$). In addition, relative ankle contribution was not different in the HLT group compared with the LLT group (Figure 5).

Discussion

The present study sought to determine the physiological and biomechanical differences between high LT_{VO2} (HLT) and low LT_{VO2} (LLT) cyclists. In well-trained cyclists with equally high VO_{2max}, those with low LT_{VO2} during cycling can sometimes achieve a higher LT_{VO2} during uphill treadmill running which suggests that the reduced LT_{VO2} is cycling-specific in some highly trained cyclists (Coyle et al. 1988). Prior research has shown HLT cyclists have reduced glycogen utilization of the knee extensors which was speculated to be due to shifts in biomechanical strategies during submaximal cycling (Coyle et al. 1988); specifically, the authors hypothesized that HLT cyclists distribute power production across multiple joints which reduces stress on the knee extensors. Therefore, we hypothesized that compared with LLT cyclists, HLT cyclists would exhibit greater relative hip power contribution and lower relative knee power contribution during submaximal cycling resulting in lower physiological stress as evidenced by lower deoxygenation and EMG activation of the knee extensors. In the present study, we observed that HLT cyclists have a greater contribution from the hip joint when cycling at 90% VO_{2max} and lower EMG activation of the VM compared with LLT cyclists.

Differences between groups in relative hip joint contribution emerged at 80% ($p=0.07$) and 90% of VO_{2max} ($p<0.05$): the HLT group had greater relative contribution of power from the hip joint compared with the LLT cyclists. For knee joint power there was a cross-over effect: with increases in work rate the LLT group significantly increased knee joint power, but there were no

changes in knee joint power in the HLT group (Figure 5). In the LLT group, since both the absolute hip joint and knee joint power increased with increasing work rates, there were no changes in relative hip joint or knee joint contribution over time. However, absolute hip joint power increased at 80 & 90% of VO_{2max} in the HLT group without changes in knee power leading to greater relative hip joint contribution at 90% of VO_{2max} in the HLT group compared with the LLT group. This finding suggests the HLT group relies more on increasing hip joint power to accommodate increases in work rate and provides further evidence that HLT cyclists have adapted a biomechanical strategy to rely more on their hip for power production during submaximal cycling (Coyle et al. 1988, Coyle et al. 1991).

The higher relative hip joint contribution in HLT compared with LLT cyclists supports the work of Aasvold et al. (2017) who compared elite cyclists with amateur recreational cyclists (Aasvold 2017). However, other studies which have attempted to compare joint powers between groups of cyclists have found inconsistent results (Hoshikawa 2007, Bini et al. 2014), likely due to the subject population and work rates chosen for comparison. As such, prior research has found greater relative knee contribution and lower relative hip contribution in cyclists compared with recreationally active adults (Hoshikawa 2007) as well as no differences in relative hip or knee joint contribution between well-trained cyclists and triathletes (Bini et al. 2014). Prior studies have used groups which differed in both maximal oxygen consumption and lactate threshold (Aasvold 2017) or failed to report either (Hoshikawa 2007, Bini et al. 2014). Therefore, subjects either exercised at different absolute workloads (Aasvold 2017) or potentially different relative oxygen consumptions (Hoshikawa 2007, Bini et al. 2014). In the current study, and in the work by Aasvold et al. 2017, subjects were compared at work rates relative to maximal oxygen consumption or lactate threshold and both found greater relative hip joint contribution in the more 'experience'

cyclists. Conversely, when studies compared experienced and novice cyclists at the same absolute work rates the experienced cyclists had a higher relative knee joint contribution and lower relative hip joint contribution (Hoshikawa 2007). However, when using absolute work rates the relative exercise intensity would be lower for trained cyclists and therefore, a higher relative hip joint contribution may not have been necessary to accomplish the given task. By controlling for maximal oxygen consumption, the current study allowed direct comparisons between groups at the same submaximal work rates thereby limiting the confounding effects absolute work rate can have on joint powers (Ericson 1986, Ericson 1988). The present study adds to the current literature examining joint power differences among individuals who vary in cycling ability and is unique its ability to: 1) compare groups of cyclists that differ in lactate threshold (which is cycling specific) rather than cycling experience and 2) control for relative exercise intensity (%VO_{2max})/absolute work rate (W).

No differences were found in muscle deoxygenation between the HLT and LLT groups suggesting no significant differences between groups in the oxidative stress placed on the knee extensors during submaximal cycling. However, differences in hip and knee joint contribution can have direct effects on deoxygenation of the knee extensors while cycling (Skovereng et al. 2016). Previous work has shown that knee extensors' muscle oxygen consumption, assessed by the vascular occlusion technique using near-infrared spectroscopy, is related to absolute and relative knee joint specific power during submaximal cycling (Skovereng et al. 2016). Therefore, lower relative stress, assessed by higher muscle oxygenation, could be reflective of lower relative knee joint contributions. The lack of differences between groups in muscle oxygenation could be due to the variations in muscle oxygenation in response to changes in work rate (Chin et al. 2011) or the location (proximal versus distal) of sensor placement on a given muscle (Spencer et al. 2014). The

oxygenation of the VL may not be truly reflective of the combined effect of the muscles crossing the knee joint which could partially explain the discrepancies between the present study and prior investigations.

In addition to muscle oxygenation status, the present study assessed muscle activation with electromyography to determine relative intensity of muscle activation and patterns of muscle recruitment. While prior research has shown increased muscle activation of the vastus lateralis in novice compared with experienced cyclists (Takaishi et al. 1998), the present study found little difference in vastus lateralis muscle activation between groups. These discrepancies could be attributed to the relatively low work rate chosen by Takaishi et al. 1998 or the differences in cycling experience between novice and experienced cyclists which highlighted differences in VL muscle activation. By including only experienced cyclists the present study restricted the range of 'cycling skill' and perhaps the ability to detect differences in VL activation between groups. Importantly, in the present study, the HLT group did have lower activation of another knee extensor, the vastus medialis (VM), suggesting a reduced stress on the vastii group. Additionally, the HLT group demonstrated increased SOL activation compared with the LLT group which is consistent with fine wire EMG studies on lower extremity muscle activation in experienced and novice cyclists (Chapman et al. 2006, Chapman et al. 2008) and may reflect a less skilled pattern of SOL recruitment during cycling in the LLT group. The Gmax and vastii muscle groups account for ~80% of the power production during cycling; however, only approximately 55% of that power production is transferred directly to the pedal while the rest (45%) is transferred to the limb segments (Raasch et al. 1997, Neptune et al. 2000). Therefore, plantar flexion muscle activation is necessary to ensure the remaining 45% of the power going to the limbs can assist in the acceleration of the crank, rather than transfer to knee extension and dorsiflexion (Raasch et al.

1997). Thus, the greater SOL activation in the HLT cyclists may assist in the transfer of hip power towards crank acceleration, and agrees with prior studies which suggest triathletes and novice cyclists differ in muscle recruitment of the plantar/dorsi flexors and pedaling technique compared with experienced cyclists (Chapman et al. 2006, Chapman et al. 2008, Chapman et al. 2009).

This is the first study to compare relative joint contribution, muscle deoxygenation, and muscle activation, differences among high and low LT_{VO2} cyclists with similar VO_{2max}. Therefore, the confounding effects of different work rates on relative joint contributions were eliminated allowing physiological and biomechanical measures to be compared at the same absolute and relative submaximal work rates. Prior research has hypothesized that an increased ability to recruit additional muscle mass during submaximal cycling can increase LT_{VO2}, and total active muscle mass explains ~20% of the variance in LT_{VO2} (Coyle et al. 1988, Coyle 1995). As more muscle mass is recruited, there is a larger amount of muscle mass able to share in the metabolic work which equates to lower oxygen demand per mitochondria, lesser reliance on glycogenolysis, and a lower overall lactate production during submaximal exercise (Holloszy et al. 1984, Coyle et al. 1988). Therefore, the higher relative hip joint contribution at 80% ($p=0.07$) and 90% of VO_{2max} ($p<0.05$) in the HLT cyclists could be reflective of an increased ability to 'spread the metabolic work' by increasing the contribution of the hip to power production leading the lower blood lactate concentrations in the HLT group at 80 and 90% of VO_{2max} compared with the LLT cyclists. Without a direct measure of total active muscle mass, we are unable to determine whether the HLT group's lower blood lactate concentrations while cycling was due to their ability to spread the work to more active muscle.

The present study is unique in its ability to determine whether HLT group exhibits greater relative contribution from the hip during submaximal cycling and whether these differences are

associated with lower physiological stress of the knee extensors (assessed by muscle deoxygenation and EMG activation). The HLT cyclists exhibited lower EMG activation of knee extensors (i.e. VM) and increased relative power production from their hip. Therefore, it appears that HLT cyclists rely more on their hip to produce power during submaximal cycling compared with LLT cyclists, which is reflected in higher relative hip power and lower knee extensor activation during submaximal cycling. These results add to the growing evidence suggesting the traditional limiters of endurance performance (i.e. VO_{2max} and LT_{VO2}) can be influenced by cycling biomechanics patterns.

Tables and Figures

Table 1. Results of maximal oxygen consumption testing between High Lactate Threshold (HLT) and Low Lactate Threshold (LLT) groups during cycling and treadmill running.

	HLT		LLT	
	Cycling	Treadmill	Cycling	Treadmill
Absolute VO_{2max} (L/min)	4.57 ± 0.17	4.47 ± 0.13	4.42 ± 0.15	4.49 ± 0.16
Relative VO_{2max} ($mL \cdot kg^{-1}min^{-1}$)	60.3 ± 2.0	58.7 ± 1.3*	64.9 ± 1.6	65.9 ± 2.2
Heart Rate Max (beats/min)	189 ± 2*†	184 ± 3	179 ± 3	179 ± 3
Maximal RER	1.10 ± 0.01	1.09 ± 0.02	1.10 ± 0.01	1.07 ± 0.02
Maximal RPE	18 ± 1	18 ± 1	18 ± 1	18 ± 1
Work Rate at VO_{2max} (W)	359 ± 16	------	326 ± 17	------

*significant difference between HLT & LLT within same exercise mode. †significant difference between Cycling and Treadmill within same group. RER: respiratory exchange ratio; RPE: rating of perceived exertion

Table 2. Results of lactate threshold testing between High Lactate Threshold (HLT) and Low Lactate Threshold (LLT) groups during both cycling and uphill running.

	HLT		LLT	
	Cycling	Treadmill	Cycling	Treadmill
LT Threshold VO_2 (L/min)	3.68 ± 0.21*	3.73 ± 0.18	3.10 ± 0.15†	3.71 ± 0.13
LT Threshold $\%VO_{2max}$	80.2 ± 2.1	82.3 ± 2.5	70.3 ± 2.9*†	82.8 ± 2.3
LT Threshold Heart Rate (beats/min)	162 ± 4	164 ± 3	152 ± 7†	168 ± 4
LT Threshold Work Rate (W)	263 ± 9*	------	221 ± 14	------

*significant difference between HLT & LLT within same exercise mode.
†significant difference between Cycling and Treadmill within same group.

Table 3. Electromyography (EMG) results. Between group differences in percent activation (% Act) of lower extremity muscles during submaximal cycling of increasing work rate in the High (HLT) and Low (LLT) threshold cyclists (Mean ± SE).

		%VO_{2max}			
		60	**70**	**80**	**90**
Vastus Lateralis (% Act)	HLT	37.4 ± 2.5	39.5 ± 2.2	41.4 ± 2.9	47.1 ± 2.7[a,b,c]
	LLT	36.7 ± 2.2	35.8 ± 1.5	45.5 ±4.7[b]	44.6 ± 6.3[a,b]
Rectus Femoris (% Act)	HLT	29.3 ± 5.1	32.7 ± 5.2	30.6 ± 2.5	35.8 ± 2.4
	LLT	24.3 ± 0.9	23.2 ± 1.1	26.7 ± 3.2	33.2 ± 7.8
Vastus Medialis (% Act)	HLT	29.6 ± 1.5*	32.5 ± 1.9*	33.2 ± 3.1	38.8 ± 2.7
	LLT	50.3 ± 8.5	52.0 ± 9.6	41.4 ± 4.0	40.8 ± 2.5
Biceps Femoris (% Act)	HLT	40.9 ± 3.5	41.7 ± 4.1	41.7 ± 2.5	43.9 ± 2.9
	LLT	35.2 ± 3.4	36.6 ± 2.5	35.9 ± 2.5	39.1 ± 1.8
Gluteus Maximus (% Act)	HLT	49.3 ± 3.7	55.4 ± 3.7	66.2 ± 4.9[a]	69.8 ± 1.8[a]
	LLT	41.6 ± 5.9	46.9 ± 5.4	50.1 ± 6.4	49.4 ± 5.8
Soleus (% Act)	HLT	52.5 ± 7.5*	54.2 ± 5.3	51.5 ± 2.0	50.4 ± 2.4
	LLT	40.8 ± 3.7	40.3 ± 3.3	44.4 ± 4.8	42.4 ± 2.5
Gastrocnemius (% Act)	HLT	36.7 ± 4.5	35.8 ± 2.9	36.1 ± 2.1	34.3 ± 1.8
	LLT	29.3 ± 5.5	26.6 ± 3.8	27.4 ± 4.3	27.1 ± 3.1
Tibialis Anterior (% Act)	HLT	54.6 ± 7.4	48.4 ± 3.8	40.1 ± 3.6	48.7 ± 3.9
	LLT	33.3 ± 8.1	23.1 ± 6.5	34.1 ± 11.4	36.1 ± 6.3

*=significant difference between the HLT and LLT group, a=significant difference compared with the 60% work rate, b= significant difference compared with the 70% work rate.

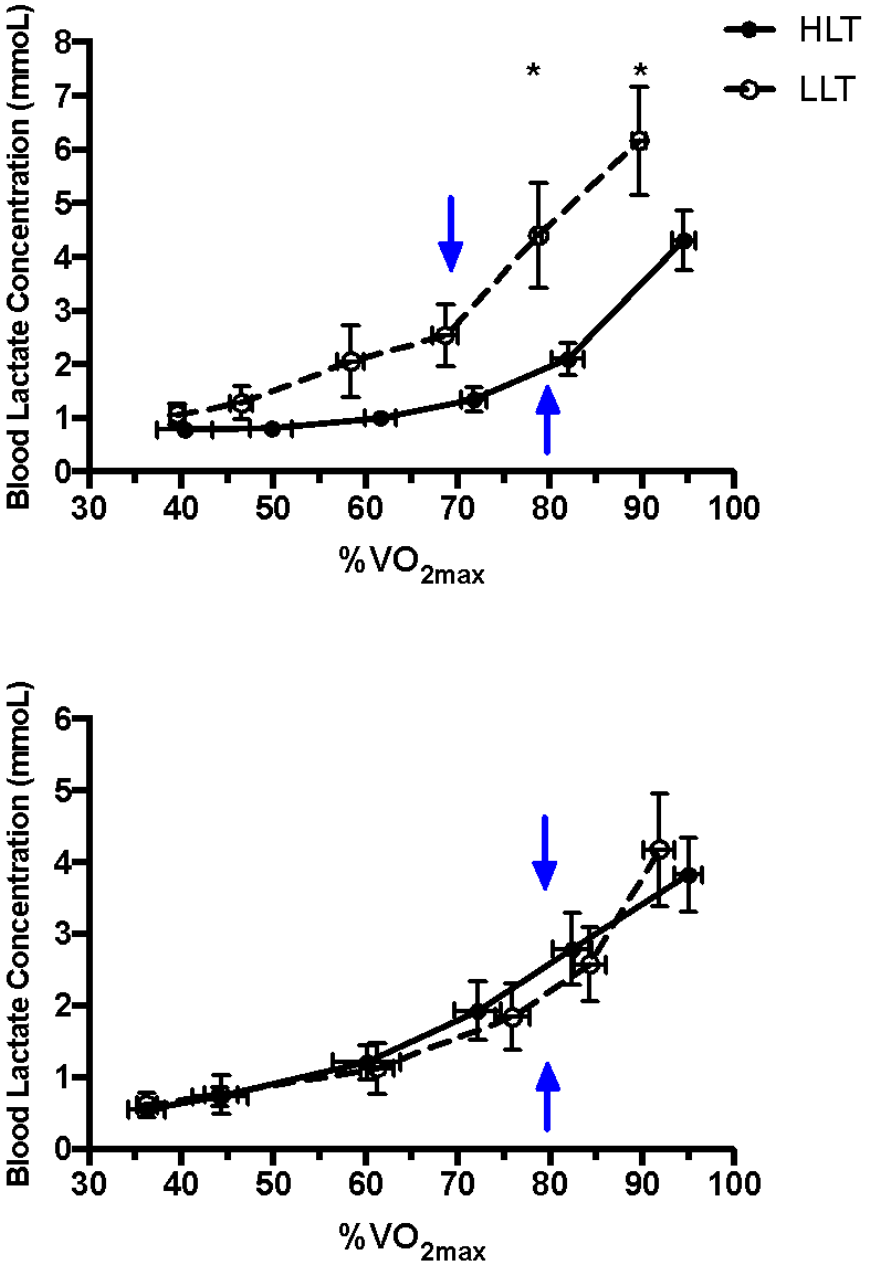

Figure 1. Blood lactate concentration during lactate threshold testing during A) Cycling and B) Uphill Running (Mean ± SE). Solid lines with closed symbols represent the High Lactate Threshold (HLT) group and dashed lines with open symbols represent the Low Lactate Threshold (LLT) group. Blue arrows indicate LT_{VO2}. *significant difference between groups at that work rate.

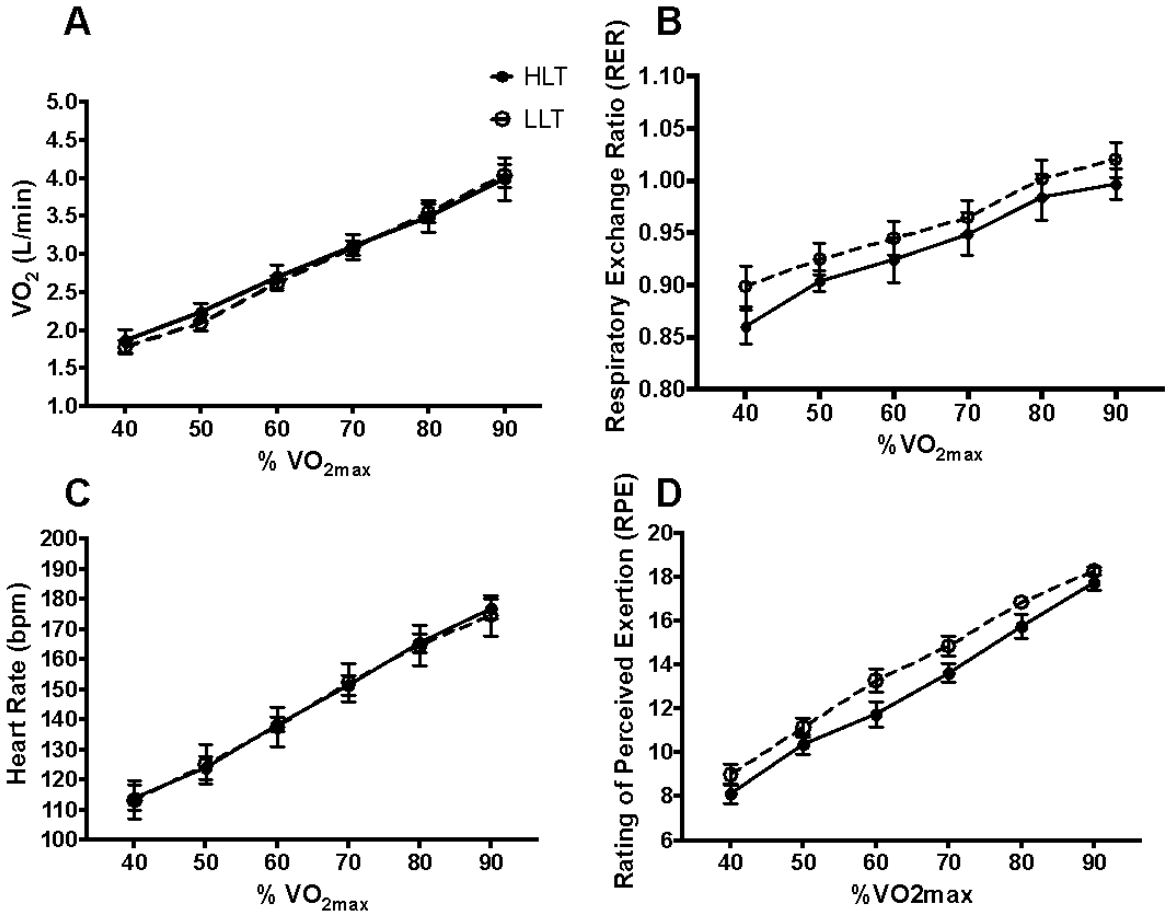

Figure 2. Physiological measurement during the cycling LT_{VO2} testing in both the High Lactate Threshold (HLT) and Low Lactate Threshold (LLT) groups (Mean ± SE). Solid lines with closed bars represent the HLT group and dashed line with open symbols represent the LLT group. There were no significant differences between groups at any work rate (i.e. $\%VO_{2max}$) ($p>0.05$).

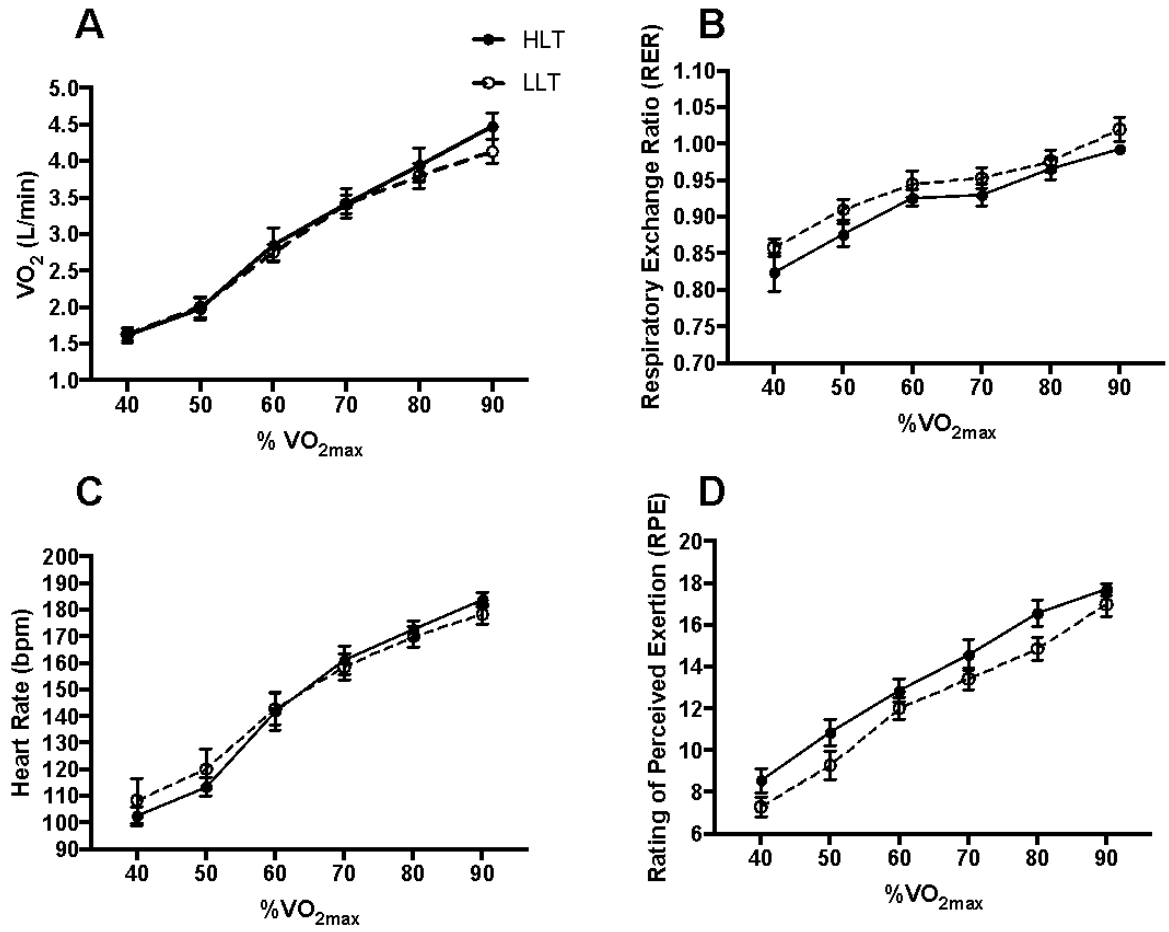

Figure 3. Physiological measurement during the uphill running LT_{VO2} testing in both the High Lactate Threshold (HLT) and Low Lactate Threshold (LLT) groups (Mean ± SE). Solid lines with closed symbols represent the HLT group and dashed line with open symbols represent the LLT group. There were no significant differences between groups at any work rate (i.e. $\%VO_{2max}$) ($p>0.05$).

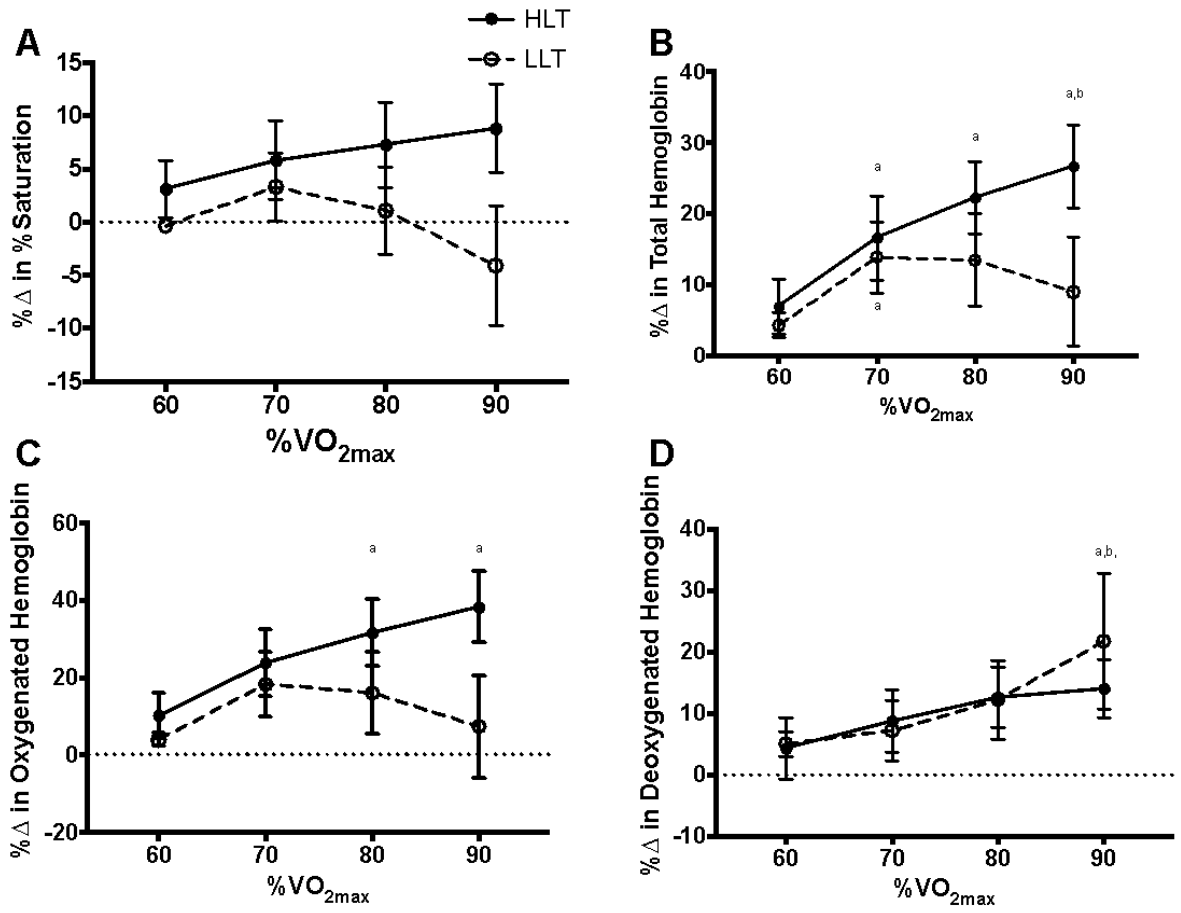

Figure 4. Near-infrared spectroscopy (NIRS) measurements of the vastus lateralis during submaximal cycling between High Lactate Threshold (HLT) and Low Lactate Threshold (LLT) groups. Calculated as the percent change from resting oxygenation. Solid lines with closed symbols represent the HLT group and dashed lines with open symbols represent the LLT group. There were no significant differences between groups ($p>0.05$); however, there were significant differences across work rates ($p<0.05$). [a]significant difference compared with the 60% VO_{2max} work rate within that group. [b]significant difference compared with the 70% VO_{2max} work rate within that group.

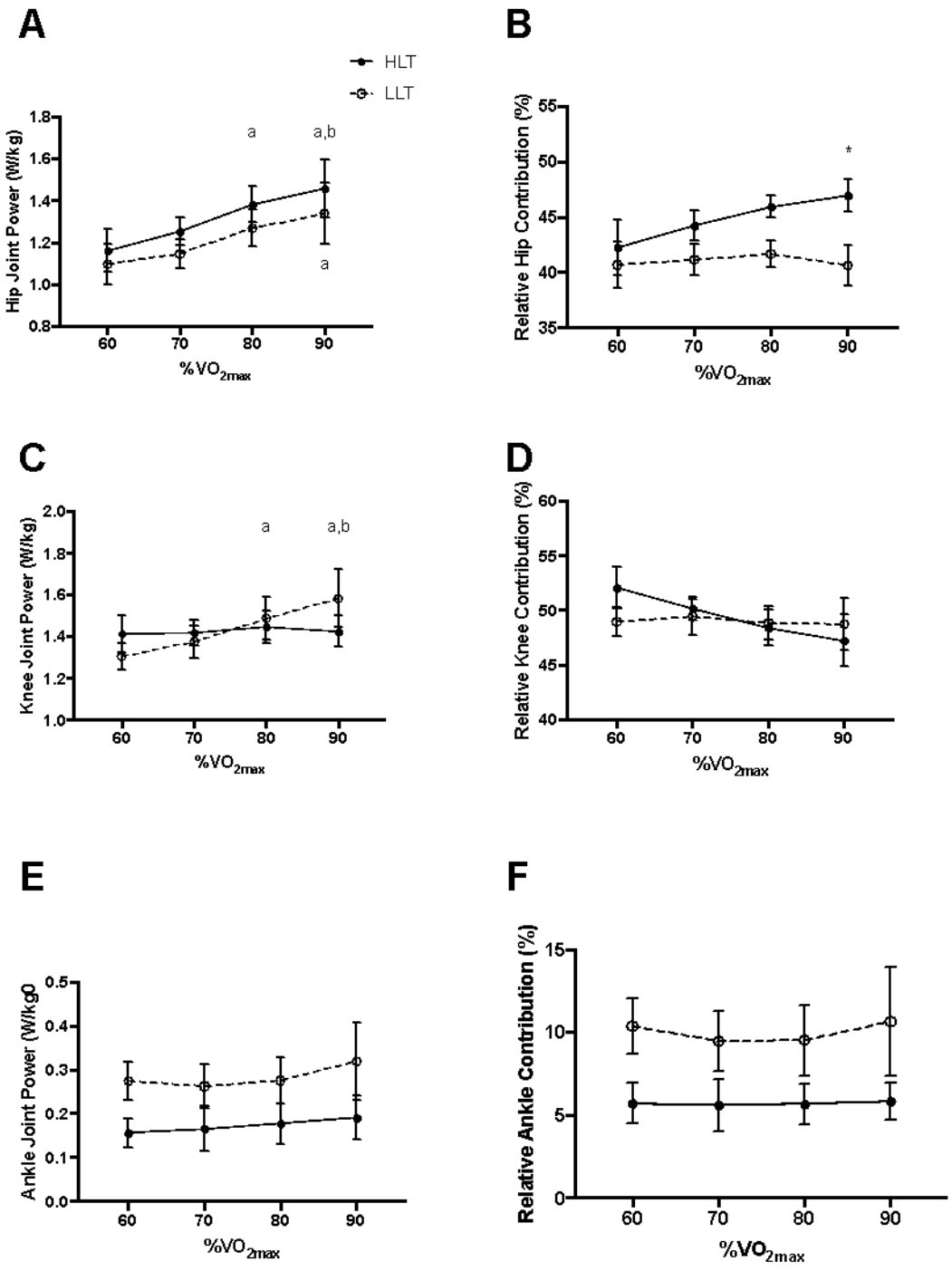

Figure 5. Between group comparison of joint powers and relative joint contribution with increases in work rate (Mean ± SE). A&B) Hip, C&D) Knee, E&F) Ankle. Solid lines with closed symbols represent the High Lactate Threshold (HLT) group and dashed line with open symbols represent the Low Lactate Threshold (LLT) group. *between group differences at that work rate. [a]significant difference between 60% VO_{2max} work rate, [b]significant difference compared with the 70% VO_{2max} work rate.

Chapter V: Study #2

EFFECTS OF SHORT-TERM MAXIMAL POWER TRAINING ON VO_{2PEAK} AND LACTATE THRESHOLD IN NON-CYCLISTS

Abstract

Background: The present study tested whether short-term inertial-load training, designed to achieve maximal neuromuscular recruitment with minimal aerobic stress, could improve measures of aerobic metabolism while cycling using measures of $VO_{2peak\text{-}cycling}$, lactate threshold and oxygenation (NIRS) of the vastus lateralis (VL) in individuals without cycling experience. **Methods:** Twenty subjects (9 males) completed pre-assessments (Day 1: $VO_{2max\text{-}treadmill}$; Day 2: $VO_{2peak\text{-}cycling}$ and LT+NIRS) assessments and were randomly allocated into a Training (n=10) group or Control group (n=10). Training group performed 10 bouts of maximal acceleration for 4 seconds with 2 minutes rest between bouts each day of training. Control group received no intervention. All subjects completed post-assessments ($VO_{2peak\text{-}cycling}$; LT+NIRS) on Day 9. **Results:** Groups did not differ in absolute or relative $VO_{2max\text{-}treadmill}$ or cycling $VO_{2peak\text{-}cycling}$ ($p>0.05$). Maximal power or RPM @ maximal power did not differ across training days ($p>0.05$). Training group absolute $VO_{2peak\text{-}cycling}$, relative $VO_{2peak\text{-}cycling}$, peak work rate increased following training ($p<0.05$) without differences (pre to post-training) in maximal RER, heart rate, or RPE ($p>0.05$). Groups did not differ in pre and post-intervention regarding $VO_{2peak\text{-}cycling}$ ($p>0.05$) or other variables. Post training $LT_{VO2\text{-}cycling}$ did not differ from pre-training in the Control or Training groups ($p>0.05$). Muscle oxygenation (NIRS) did not differ pre-training vs post-training ($p>0.05$). **Conclusion:** $VO_{2peak\text{-}cycling}$ can be improved through maximal power cycling training over a 5-day period without significant cardiovascular stress during training in individuals without prior cycling experience.

Introduction

Maximal oxygen uptake (VO_{2max}) and lactate threshold oxygen consumption (LT_{VO2}) are predictors of endurance performance among athletes (Tanaka et al. 1993, Coyle 1995, Coyle 1999, Bassett et al. 2000, Joyner et al. 2008). Adaptations to endurance training that lead to improvements in VO_{2max} are primarily central in nature (i.e. increases in stroke volume and cardiac output) which effect the delivery of blood and, more specifically, oxygen to the working muscles (Ekblom et al. 1968, Saltin et al. 1968). Peripheral adaptations (i.e. mitochondrial density and increased capillarization) with endurance training play a smaller role in influencing VO_{2max}, but are still significant determinants of submaximal performance (i.e. LT_{VO2}) (Holloszy et al. 1984, Saltin 1985, Bassett et al. 2000). While VO_{2max} sets the upper limit of submaximal endurance performance, the determinants of LT_{VO2} are complex and represent a combination of central and peripheral adaptations that lead to improvements in oxygen delivery and muscle oxidative capacity (Holloszy et al. 1984, Coyle 1995, Bassett et al. 2000).

VO_{2max} is specific to the mode of exercise used in training and the recruitment of a large enough quantity of muscle to surpass the ability of the cardiovascular system to deliver blood and oxygen to the working muscles (Tanaka 1994, Millet et al. 2009). Untrained individuals and experienced runners have values ~10% lower on a cycle ergometer (i.e. $VO_{2peak\text{-}cycling}$) compared with treadmill running (Astrand et al. 1961, Withers et al. 1981, Mikesell et al. 1984). Compared with running, the lower $VO_{2peak\text{-}cycling}$ has been attributed to lower cardiac output and/or lower arteriovenous oxygen difference ($A\text{-}VO_{2difference}$) (Hermansen et al. 1969, Hermansen et al. 1970, Faulkner et al. 1971). However, $VO_{2peak\text{-}cycling}$ values may also be due to lower total muscle mass used while cycling compared with running and thus a less than maximal stress on cardiac output (Faulkner et al. 1971). In support of this, $VO_{2peak\text{-}cycling}$ has been shown to be higher when untrained

men perform maximal testing on a cycle ergometer while standing as compared with sitting, which was attributed to greater total active muscle mass (Tanaka et al. 1987).

Non-cyclists can have a relatively low $LT_{VO2\text{-}cycling}$ compared with running, suggesting that test specificity or prior experience in the exercise modality plays an important role in determining LT_{VO2} (Mazzeo et al. 1989, Bouckaert et al. 1990). Some high $LT_{VO2\text{-}cycling}$ cyclists display less vastus lateralis glycogen depletion during exercise compared with low $LT_{VO2\text{-}cycling}$ cyclists. This has been attributed to differences in cycling technique, specifically, that high $LTvo_{2\text{-}cycling}$ cyclists are theorized to place less stress on knee extensors (Coyle et al. 1988). Recently, near-infrared spectroscopy has been used to determine muscle oxygenation and deoxygenation of the vastus lateralis (Skovereng et al. 2016, Skovereng et al. 2016), which has served as an indicator of the physiological stress placed on the working muscle during exercise. With this method, the breakpoint in muscle deoxygenation has been shown to correspond with $LT_{VO2\text{-}cycling}$ (Wang et al. 2012). Therefore, in addition to the typical predictors of LT_{VO2} (i.e. mitochondrial number/activity), $LT_{VO2\text{-}cycling}$ might be partially explained by cycling experience and learning which could allow for increased amount of active muscle mass during cycling. This suggests that untrained individuals might improve $LT_{VO2\text{-}cycling,}$ and $VO_{2peak\text{-}cycling}$ without traditional physiological adaptations (i.e. increases in mitochondrial number/activity), but simply by gaining cycling experience. However, to date, no study has yet examined whether maximal neuromuscular power training increases $VO_{2peak\text{-}cycling}$ and $LT_{VO2\text{-}cycling}$ by reducing stress (i.e. HHb) on the vastii muscles during cycling exercise.

Inertial-load cycling elicits maximal neuromuscular power production in a 4 second bout of sprinting (Martin et al. 1997, Martin et al. 2000, McLean et al. 2012). This type of exercise maximally activates the muscles necessary for cycling with only small increases in oxygen

consumption when adequate rest is given between bouts. Recreationally active individuals demonstrate ~7% increases in maximal power production over ~3 training sessions performing repeated sprint bouts with 1-2 min rest using the inertial load method (Martin et al. 2000). This suggests that maximal power production while cycling is, in part, a partially learned skill that can be acquired relatively quickly (Martin et al. 2000). Heavy strength training has been shown to increase $VO_{2peak\text{-}cycling}$, but this study was prolonged (10 weeks), and was accompanied by increases in lean body mass (Hickson et al. 1980). Therefore, it cannot be determined whether the increases in absolute $VO_{2peak\text{-}cycling}$ are attributed to increased total body mass or an improved ability to recruit additional muscle mass while cycling. Therefore, the present study employed a training protocol that would maximize neuromuscular power, minimize cardiovascular stress, and determine whether maximal neuromuscular power training can lead to improvements in $VO_{2peak\text{-}cycling}$ and $LT_{VO2\text{-}cycling}$ through the improved ability to recruit additional muscle mass.

Currently, it is unknown whether maximal power training elicits improvements in $VO_{2peak\text{-}cycling}$ or $LT_{VO2\text{-}cycling}$. Therefore, we propose to examine changes in $VO_{2peak\text{-}cycling}$ and $LT_{VO2\text{-}cycling}$ and after a short-term, inertial-load maximal power cycling training program. We hypothesize that recreationally trained individuals will demonstrate improved cycling $VO_{2peak\text{-}cycling}$, $LT_{VO2\text{-}cycling}$, and reduced muscle deoxygenation on their knee extensors.

Methods

Study Overview

Twenty recreationally-active, college-aged men and women without cycling experience were recruited to participate in this study. Subjects were randomly allocated into either a Training group (n=10) or a Control group (n=10) and, over a one week period, visited the laboratory either three (Control) or eight (Training) times. The study timeline can be found below (Figure 6).

Day 1,3&10: *Treadmill Maximal Oxygen Consumption and Cycling Peak Oxygen Consumption*

On the first day of testing (Day 1) subjects performed an incremental treadmill $VO_{2max\text{-}treadmill}$ test lasting 8-12 minutes during which time, incline was increased by 2% every 2 minutes (Costill 1970). VO_2, VCO_2, and heart rate (HR) was monitored throughout the test, and the highest 30 second VO_2 average was recorded for maximal oxygen consumption. ACSM criteria for VO_{2max} was used to determine a successful $VO_{2max\text{-}treadmill}$ test; these criteria are: respiratory exchange ratio (RER) >1.1, Max Heart Rate (HR_{max}) ± 10 bpm of predicted maximal heart rate (Tanaka et al. 2001), oxygen consumption plateau of ≤ 150 mL/min, and rating of perceived exertion (RPE) >17 (Pescatello et al. 2014). The values for treadmill $VO_{2max\text{-}treadmill}$ served two purposes: 1) to confirm subjects have no cycling experience (compared with cycling $VO_{2peak\text{-}cycling}$) 2) to determine workloads for the cycling lactate threshold and $VO_{2peak\text{-}cycling}$ testing procedures. On Day 3 & 10 subjects performed a peak oxygen consumption test on the cycle ergometer, initial workloads were estimated from the results of the treadmill $VO_{2max\text{-}treadmill}$ test. The cycling $VO_{2peak\text{-}cycling}$ test on Day 10 used the same protocol as used during Day 3 of testing.

Day 3&10: *Lactate Threshold Testing and Muscle Deoxygenation*

On Day 3, subjects performed a 25-minute submaximal cycling test consisting of five 5-minute stages of progressive intensities (40,50,60,70, & 80% of VO_{2peak}) estimated from treadmill $VO_{2max\text{-}treadmill}$testing. VO_2, VCO_2, and HR were monitored throughout the test, and averaged over the final minute of each stage. During the final minute of each stage approximately 1mL of blood was drawn from a venous catheter and used later for plasma lactate analysis. When subjects returned for Day 10 of testing they performed the same submaximal cycling protocol utilizing the workloads of Day 3.

Respiratory and Cardiovascular Measurements During Maximal Exercise

During exercise the participants breathed through a two-way non-rebreathing valve (Hans Rudolph, Kansas City, MO). Ventilation was measured via an inspiratory pneumotachometer attached to the two-way valve (Hans Rudolph, Kansas City, MO). Expired gas samples were taken from a mixing chamber which was directly connected via capillary tubing to oxygen and carbon dioxide analyzers (Applied Electrochemistry, Models S-3A/I and CD-3A, respectively). MOXUS metabolic software (Applied Electrochemistry) was then used to continuously analyze VO_2, VCO_2, and RER throughout the exercise trials. Gross efficiency was calculated as the ratio of the energy produced on the ergometer to the rate of caloric expenditure as previously described (Coyle et al. 1992, Horowitz et al. 1994).

Near-Infrared Spectroscopy (Muscle Deoxygenation)

Near-infrared spectroscopy (OxiplexTS TS, ISS, Champaign, IL) was used to measure oxygenated hemoglobin [O_2Hb] and deoxygenated hemoglobin [HHb] in the vastus lateralis during the lactate threshold testing procedure. The chromophores of O_2Hb and HHb have different optical properties of absorbing near-infrared light (wave length: 690 nm, 830 nm). This enables NIRS to measure the absolute concentrations of O_2Hb and HHb in μM in real-time noninvasively (Ryan et al. 2012, Boone et al. 2015, Skovereng et al. 2016, Skovereng et al. 2016). NIRS serves as a marker of 'physiological stress' and was compared between testing sessions. The NIRS data during the last 1 minute of each stage were averaged and recorded.

Days 4-8: *Control or Training Intervention*

Control Intervention

Subjects in the Control group were instructed to maintain their normal diet and exercise programs over the course of Days 5-9. Subjects were instructed to refrain from vigorous activity on Day 2 and 9 of testing to minimize effects of fatigue on $VO_{2peak\text{-}cycling}$ testing on Days 3 and 10.

Training Intervention

Subjects in the Training group visited the lab on 5 successive days to perform maximal inertial-load cycling training (i.e.; days 5-9). For the training program, subjects performed 10 bouts of maximal acceleration on the Inertial Load Ergometer with 2-minutes resting recovery between trials. Subjects started from a resting crank angle of approximately 45° from top dead center (0°), and accelerated maximally for approximately 3-4 s with standardized verbal encouragement. Seat height was self-selected by each subject, and the same height was used for all trials. Subjects were instructed to remain seated throughout the duration of each bout (Martin et al. 1997). All trials were analyzed for absolute and relative maximal power as well as maximum pedal revolutions, and RPM at max power. Subjects in the Training group were instructed to maintain their normal diet and exercise programs in addition to completing the inertial-load training program.

Statistical Measures

Descriptive statistics are reported as Mean ± SE. Between group differences pre and post training on cycling $VO_{2peak\text{-}cycling}$ testing measures were compared with two-way repeated measures ANOVA (Treatment x Time). In order to determine differences in subject characteristics pre-intervention, two-tailed unpaired t-tests were performed (α=0.05). Within and between group differences in submaximal cycling measures were compared through multiple two-way repeated measures ANOVA (Treatment x Work Rate). Tukey's least significant difference test were used for post hoc comparisons on significant main effects (α=0.05).

Results

Subject Characteristics

Twenty recreationally active individuals (9 males; 11 females) were recruited to participate in this research study. There were no significant differences between groups in subject characteristics including: age, height, or weight pre-intervention (See Table 4) ($p>0.05$).

Treadmill Results

The groups were similar in absolute and relative maximal oxygen consumption during pre-training treadmill testing (Table 4). Additionally, there were no differences in maximal heart rate or RPE during the treadmill test ($p>0.05$). However, pooled subject data revealed treadmill absolute $VO_{2max\text{-}treadmill}$ (3.47 ± 0.26 L/min) was 11.2 ± 2.9% higher than cycling $VO_{2peak\text{-}cycling}$ (3.06 ± 0.19 L/min) ($p<0.05$) and maximal heart rate during treadmill testing (190 ± 2 bpm) was 4.9 ± 1.1 % higher compared with cycling peak heart rate (180 ± 2 bpm) ($p<0.05$).

Inertial-Load Training Results

In the training group, there was no treatment effect for maximal power, relative maximal power, or RPM @ peak power ($p>0.05$) (Table 5). In addition, there were no differences in average heart rate, average peak heart rate, or resting heart rate across days of the training protocol ($p>0.05$). The daily average heart rate during training ranged from the lowest daily average of (93 ± 8 bpm) to a high of (98 ± 2 bpm) ($p>0.05$. While the average peak heart rate during training ranged from 121 ± 3 bpm to 123 ±2 bpm ($p>0.05$).

Cycling VO_{2peak} Results

Pre-training measures of cycling absolute $VO_{2peak\text{-}cycling}$ (L/min), relative $VO_{2peak\text{-}cycling}$ (mL/kg/min), RER, heart rate, work rate, or RPE did not differ between the Control and Training

group (Table 3) ($p>0.05$). The Control group did not differ in pre and post training measures during cycling $VO_{2peak\text{-}cycling}$ testing (Table 6) ($p>0.05$). However, in the training group, compared with pre, post intervention, absolute $VO_{2peak\text{-}cycling}$ and relative $VO_{2peak\text{-}cycling}$ increased by 5.9 ± 1.6% and 5.6 ± 1.9%, respectively ($p<0.05$) (Table 6). Additionally, in the training group, the maximal work-rate achieved during the cycling peak VO_2 test increased by 6.3 ± 2.5 % (Pre: 228 ± 18 W vs Post: 242 ± 19W) following training ($p<0.05$) (ES: 0.23, $p<0.05$) without differences (pre to post-training) in maximal RER, heart rate, or RPE ($p>0.05$) (Table 6).

Cycling Submaximal Responses

Post-training $LT_{VO2\text{-}cycling}$ did not differ from pre-training in the Control or Training groups ($p>0.05$) (Figure 7 and Figure 8A, B). The average heart rate during $LT_{VO2\text{-}cycling}$ testing was not significantly different pre vs post-training in either the Control (Pre: 150 ± 4 vs Post: 148 ± 4, $p>0.05$) or Training groups (Pre: 144 ± 4 vs Post: 141 ± 4, $p=0.07$). In addition, heart rate at lactate threshold did not change following the intervention in either the Control (Pre: 149 ± 5 bpm; Post: 151 ± 3 bpm) or Training group (Pre: 146 ± 5 bpm; Post: 139 ± 4bpm) ($p>0.05$). There was no significant main effect for TREATMENT in either the Control or Training group (Pre vs Post-training) for VO_2, RER, lactate, heart rate, or RPE ($p>0.05$) (Table 7).

There were no main effects for either WORKRATE or TREATMENT (pre-training *vs.* post-training) for % Sat for the Control group ($p>0.05$). There was a significant main effect for WORKRATE ($p<0.05$) for [total Hb], [HHb], and [O_2Hb] in the Control Group($p<0.05$) (Figure 9 A-D). For the Training group, there was a significant main effect for WORKRATE for % Saturation, [total Hb], and [HHb]($p<0.05$). However, there was no significant main effect for WORKRATE with [O_2Hb] ($p>0.05$). There were no significant main effects for TREATMENT

(pre-training *vs.* post-training) or significant interaction (TREATMENT x WORKRATE) for any of the NIRS measures ($p>0.05$) (Figure 10 A-D).

Discussion

The present study tested whether short-term inertial-load training for maximal power could improve measures of aerobic metabolism while cycling using measures of $VO_{2peak\text{-}cycling}$, lactate threshold and oxygenation of the vastus lateralis. The training protocol allowed subjects to achieve maximal neuromuscular recruitment with minimal aerobic stress in order to determine the independent effects of muscle recruitment on $VO_{2peak\text{-}cycling}$, lactate threshold, and oxygenation of the vastus lateralis. The primary findings of this study were that $VO_{2peak\text{-}cycling}$ (absolute and relative) and peak work rate were significantly improved by ~5% and ~6%, respectively, following short-term maximal power training. Inertial-load training had no effect on $LT_{VO2\text{-}cycling}$ or the muscle oxygenation responses to matched-work rate submaximal cycling.

In the present study, peak oxygen consumption and peak work rate while cycling increased following the training intervention without measurable differences in lactate threshold or muscle oxygenation. In agreement with previous findings, during the pre-measurements our subjects had ~10% lower cycling $VO_{2peak\text{-}cycling}$ compared with treadmill running. These differences might be attributed to lower total muscle mass recruitment while cycling (Tanaka et al. 1987, Tanaka 1994). $VO_{2peak\text{-}cycling}$ has previously been improved by having subjects stand during maximal cycling testing, likely as a result of increased muscle mass recruitment while standing compared to seated cycling (Tanaka et al. 1987). Additionally, prior research utilizing heavy resistance training over a period of 10 weeks has shown improvements in cycling $VO_{2peak\text{-}cycling}$ which was attributed to an improved ability to recruit additional muscle mass following training (Hickson et al. 1980). However due to the study design the subjects also increased lean body mass which could have led

to the improvements in $VO_{2peak\text{-}cycling}$. In the current study, it is likely that the increases in cycling $VO_{2peak\text{-}cycling}$ following only 5 days of maximal power training resulted in were due to an improved ability to recruit additional muscle mass while cycling.

Studies which have examined the effects of active muscle mass on $VO_{2peak\text{-}cycling}$ have done so by adding arm cycling with maximal leg cycling. Some (Gleser et al. 1974, Reybrouck et al. 1975), but not all (Astrand et al. 1961) studies using this approach have found increases in cycling $VO_{2peak\text{-}cycling}$ from the addition of arm cycling to maximal leg cycling above that achieved with maximal leg cycling alone. The cycling $VO_{2peak\text{-}cycling}$ increase found with the addition of arm muscles has been attributed to increases in cardiac output (Reybrouck et al. 1975). Traditionally, VO_{2max} values while cycling and running have been used to differentiate cyclists from runners and recreationally active individuals: well-trained cyclists can attain similar maximal oxygen consumptions while cycling and running while runners and recreationally active individuals have higher maximal oxygen consumptions while running compared with cycling. Importantly, Reybrouck et al. 1975 noted that $VO_{2peak\text{-}cycling}$ did not increase acutely with the addition of arm cycling in subjects who were "conditioned for leg ergometry" (Reybrouck et al. 1975). Since our subjects had no cycling experience and were capable of achieving ~10% higher values of oxygen consumptions while running compared with cycling, it is likely the increases in cycling specific $VO_{2peak\text{-}cycling}$ following maximal power training were a result of increased muscle mass recruitment. However, data from the current study cannot conclusively determine if the improvements in $VO_{2peak\text{-}cycling}$ is a result of increased muscle mass recruitment attributed to neuromuscular training.

Since cardiac output was not measured in the present study, we cannot determine whether increases in cardiac output or A-V $O_{2difference}$ are responsible for the increase in cycling $VO_{2peak\text{-}}$

$_{cycling}$. As previously mentioned, increasing muscle mass while cycling leads to increases in $VO_{2peak\text{-}cycling}$ through increases in cardiac output (Reybrouck et al. 1975). However, other potential factors can lead to increases in cardiac output following short-term training, with the primary factor being plasma volume expansion. Plasma volume expansion has been shown to increase VO_{2max} and time to fatigue in untrained individuals (Coyle et al. 1990). However, those studies which have shown increases in plasma volume following short-term endurance training have typically used moderate intensity exercise (60-80% VO_{2max}) for long durations 30-120 minutes (Convertino et al. 1980, Convertino et al. 1980, Green et al. 1991, Luetkemeier et al. 1994). In the present study, the average heart rate while training was ~93-98 bpm suggesting that the cardiovascular stress during the training protocol was low. Indeed, average peak heart rate during the training protocol was 121-123 bpm, corresponding to 65% of maximal heart rate for this population. While it is unlikely that 4 second intervals with the highest heart rate at 65% heart rate max for a few seconds would be sufficient to generate increases in plasma volume and cardiac output, it cannot be ruled out.

Previous research has suggested that in addition to $VO_{2peak\text{-}cycling}$, $LT_{VO2\text{-}cycling}$ is also influenced, at least in part, by the total amount of active muscle mass while cycling (Coyle et al. 1988). Since the inertial-load training protocol was designed to elicit maximal neuromuscular recruitment while cycling, we hypothesized that the duty cycle of a given motor unit would be reduced during submaximal exercise by having a larger pool of potential motor units following training. Therefore, we theorized that potential increases in the total active muscle mass recruitment might allow for reductions in blood lactate for a given workload as well as reductions in the physiological stress of the vastus lateralis, as assessed by HHb of the VL, at given submaximal work rate. Potential mechanisms leading to improvements in lactate threshold and

endurance performance are through reductions in the physiological stress placed on the vastii for a given absolute workload (Coyle et al. 1988). Muscle oxygenation of the vastii is related to total external work (watts) as well as joint specific work of the knee extensors (Skovereng et al. 2016, Skovereng et al. 2016). Therefore, we expected to find reduced HHb following training reflecting reduced physiological stress on the vastii and increased recruitment of additional muscle mass. Prior research utilizing heavy progressive resistance strength training alone or concurrently with endurance training over a period of a few weeks has found improvements in short-duration and long-duration cycling endurance performance in untrained and well-trained cyclists (Hickson et al. 1988, Marcinik et al. 1991, Vikmoen et al. 2016). This is usually, but not always, accompanied by improvements in lactate threshold (Marcinik et al. 1991, Vikmoen et al. 2016). Therefore, it is possible that the lack of training effect on lactate threshold and the NIRS responses during submaximal testing could be explained by the short nature of our protocol.

Interestingly, during the inertial-load training protocol maximal power and RPM at maximal power remained constant across training days. Prior research from our laboratory using a similar subject group found improvements of ~4% in relative maximal power after only one day of inertial load training and another 2.5% by the third day. However, in the present study there were increases within the first day of testing for maximal power (W/kg) with all but the 4th and 6^{th} bouts having higher maximal power compared with the 1st bout. This is in accordance with prior research using inertial-load maximal power training which found increases in maximal power over the course of the first day of testing (Martin et al. 2000). consider

The increases in cycling $VO_{2peak\text{-}cycling}$ following power training were likely the result of an improved ability to recruit additional muscle mass, leading to greater cardiac output and oxygen consumption. However, the current study had no direct measure of total active muscle mass

limiting the ability to confirm muscle mass recruitment as the source of $VO_{2peak\text{-}cycling}$ improvements. Additionally, with no direct measure of cardiac output, we lack the ability to discern whether cycling $VO_{2peak\text{-}cycling}$ increased as a result of changes in maximal cardiac output or A-$VO_{2difference}$. However, a strength of the present study was the low peak and average heart rate during maximal power training which likely minimized other training induced adaptations including plasma volume expansion which has been shown to increase cardiac output and VO_{2max} (Coyle et al. 1990); while maximal power training still allowed for neuromuscular adaptations and subsequent improvements in $VO_{2peak\text{-}cycling}$ through increases in muscle mass recruitment. Future research is still necessary to determine if short-term maximal neuromuscular power training has the ability to increase the physiological maximum of cardiac output and/or A-V $O_{2difference}$, or if the early changes in $VO_{2peak\text{-}cycling}$ following maximal power training can be attributed to a learning effect which increases the peak cardiac output during maximal cycling exercise as we suspect.

To date, differences in cycling $VO_{2peak\text{-}cycling}$ and treadmill VO_{2max} in individuals without cycling experience have been attributed to decreased muscle mass recruitment and reductions in cardiac outputs during cycling (Hermansen et al. 1969, Hermansen et al. 1970, Faulkner et al. 1971). The present study demonstrates that cycling $VO_{2peak\text{-}cycling}$ and cycling performance can be improved through maximal power cycling training over a 5-day period in individuals without cycling experience.

Tables and Figures

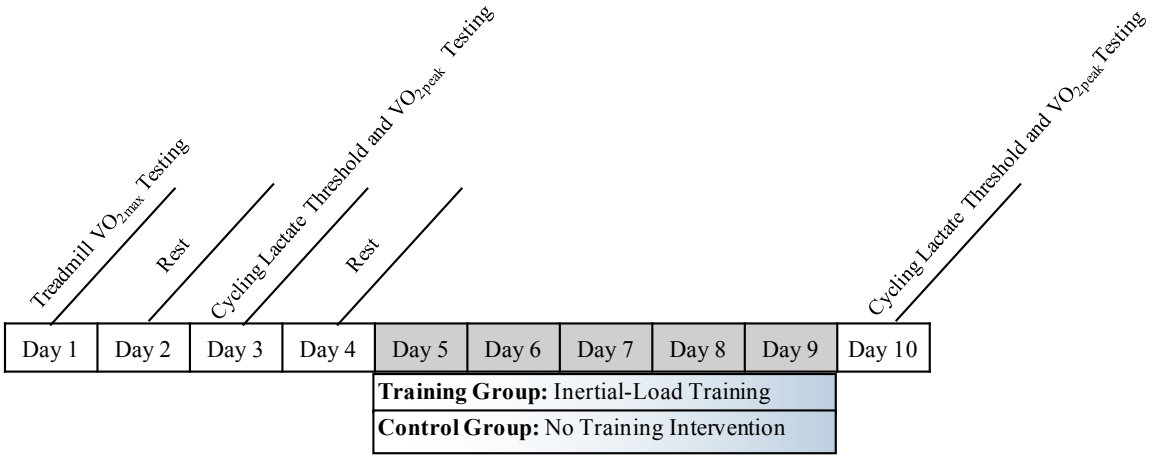

Figure 6. Study design. Subjects were separated into two groups (Control or Training) and compared during treadmill maximal and cycling lactate threshold and peak testing.

Table 4. Descriptive statistics and treadmill VO_{2max} testing results of the two groups (i.e. Control and Training) at the beginning of the study. All data are reported as Mean ± SE.

Descriptives	**Control**	**Training**
Age (yrs)	26 ± 1	27 ± 2
Height (cm)	171.0 ± 4.0	171.5 ± 4.0
Weight (kg)	76.4 ± 7.2	72.1 ± 5.5
Sex (N)	M: 4; F: 6	M: 5; F: 5
Treadmill Running		
Absolute VO_{2max}(L/min)	3.52 ± 0.31	3.44 ± 0.32
Relative VO_{2max} (mL/kg/min)	46.4 ± 2.0	48.8 ± 4.8
Heart Rate Max (bpm)	192 ± 3	188 ± 4
RPE	18 ± 1	17 ± 1

Table 5. Power and heart rate measure during the inertial-load training intervention for the Training group over the course of the 5 training days. All data reported as Mean ± SE.

	Day 1	**Day 2**	**Day 3**	**Day 4**	**Day 5**
Peak Power (Watts)	984 ± 121	1005 ± 114	987 ± 124	993 ± 122	994 ± 118
Relative Max Power (W/kg)	13.4 ± 0.9	13.7 ± 1.0	13.4± 1.0	13.5 ± 1.0	13.5 ± 0.9
RPM @ Max Power	125 ± 4	128 ± 4	127 ± 3	124 ± 4	127 ± 4
Average Heart Rate (bpm)	93 ± 3	96 ± 3	95 ± 4	97 ± 2	98 ± 2
Average Peak Heart Rate (bpm)	121 ± 3	121 ± 3	122 ± 4	121 ± 3	123 ± 2
Average Resting Heart Rate (bpm)	74 ± 4	75 ± 4	77 ± 4	77 ± 3	78 ± 3

Table 6. Cycling VO_{2peak} testing results in both groups (i.e. Control and Training) both before (Pre) and after intervention (Post). All results are reported Mean ± SE.

	Control		**Training**	
	Pre	*Post*	*Pre*	*Post*
Absolute $VO_{2peak\text{-}cycling}$ (L/min)	2.97 ± 0.27	2.99 ± 0.27	3.16 ± 0.28	3.32 ± 0.27*
Relative $VO_{2peak\text{-}cycling}$ (mL/kg/min)	39.2 ± 2.1	39.3 ± 2.0	44.1 ± 3.3	46.4 ± 3.3*
RER	1.10 ± 0.02	1.10 ± 0.01	1.10 ± 0.01	1.10 ± 0.01
Heart Rate (bpm)	182 ± 2	183 ± 1	179 ± 3	181 ± 3
Work Rate (W)	217 ± 19	224 ± 20	228 ± 18	242 ± 19*
RPE	18 ± 0	18 ± 0	18 ± 0	18 ± 0

*= significant difference between Pre and Post-training.

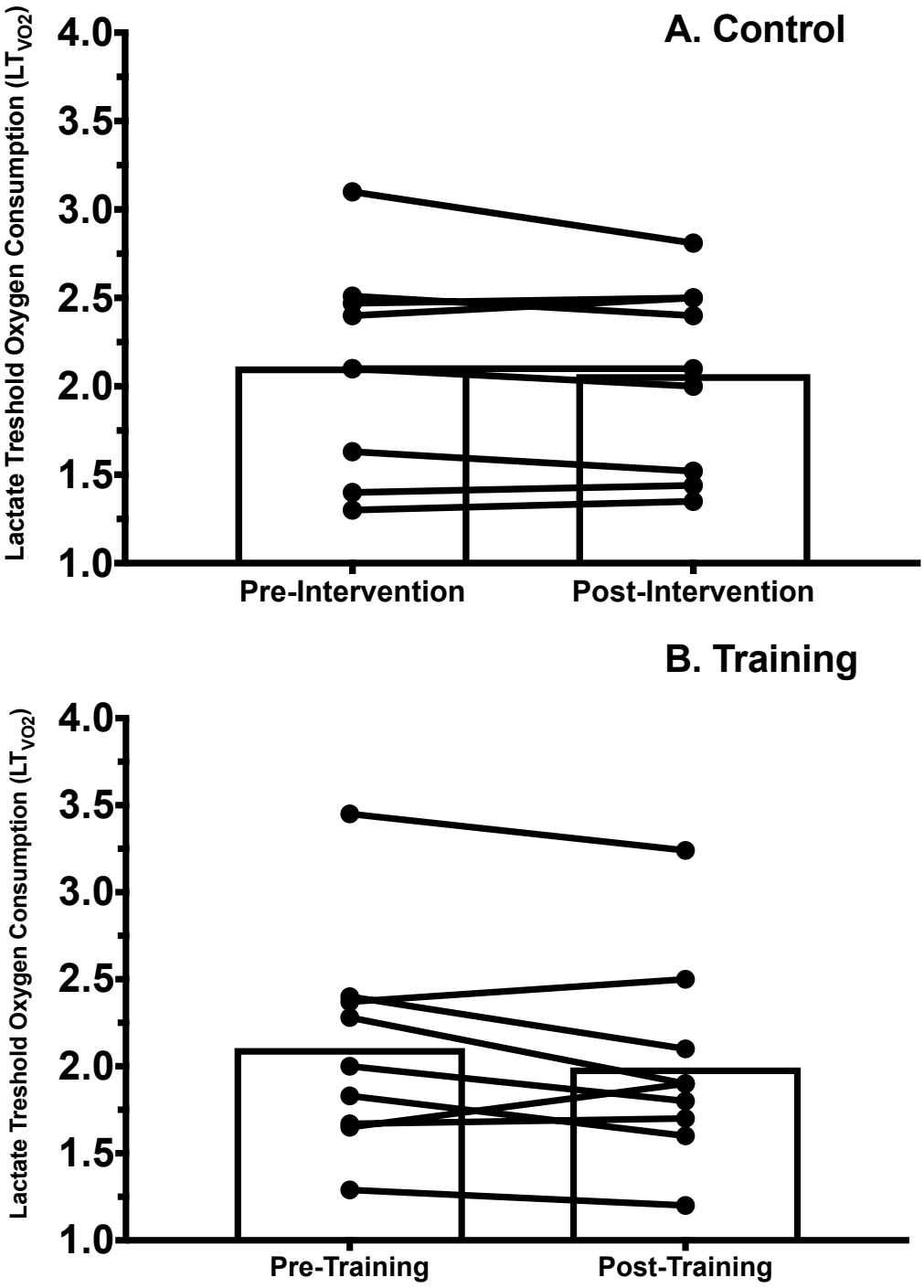

Figure 7. Lactate threshold oxygen consumption (LT_{VO2}) while cycling in the A) Control and B) Training groups before and after the intervention. Open bar represents group mean and lines represent individual subject data. No significant difference in LT_{VO2} between pre vs post measures in either group ($p>0.05$).

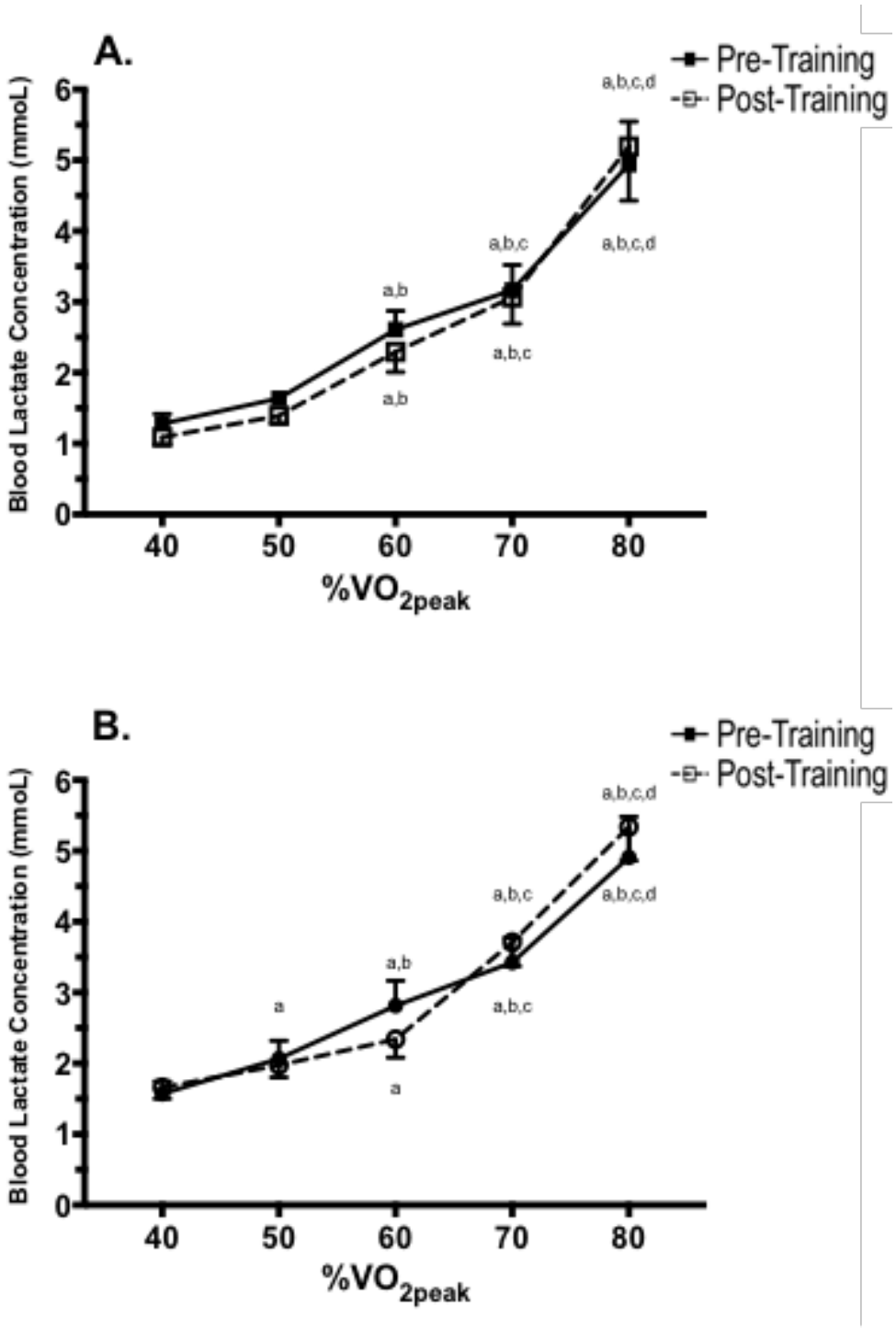

Figure 8. Mean blood lactate concentration during the cycling LT_{VO2} test in both A) Control and B) Training groups (Mean ± SE). Solid lines and closed symbols represent pre-training values. Dashed line with open symbols represent post-training values. Blood lactate concentration generally increased with increases in work rates ($p<0.05$): a>40%; b>50%; c>60%; d>70%.

Table 7. Group comparisons of physiological measurements during the cycling LT_{VO2} test both pre and post-training (Mean ± SE).

	Work Rate (%$VO_{2peak\text{-}cycling}$)									
	40		**50**		**60**		**70**		**80**	
	Pre	*Post*	*Pre*	*Post*	*Pre*	*Post*	*Pre*	*Post*	*Pre*	*Post*
VO_2 (L/min)										
Control	1.52 ± 0.18	1.51 ± 0.16	1.80 ± 0.21[a]	1.76 ± 0.19[a]	2.07± 0.24[a,b]	2.03 ± 0.22[a,b]	2.40 ± 0.29[a,b,c]	2.35 ± 0.27[a,b,c]	2.64 ± 0.33[a,b,c,d]	2.61 ± 0.29[a,b,c,d]
Training	1.43 ± 0.12	1.41 ± 0.13	1.69 ± 0.15[a]	1.65 ± 0.16[a]	1.95 ± 0.17[a,b]	1.87 ± 0.19[a,b]	2.31 ± 0.22[a,b,c]	2.18 ± 0.24[a,b,c]	2.69 ± 0.27[a,b,c,d]	2.53 ± 0.28[a,b,c,d]
Gross Efficiency (%)										
Control	13.7 ± 0.8	13.0 ± 1.1	15.6 ± 0.8	15.0 ± 1.1	17.1 ± 1.0[a]	16.5 ± 1.1[a]	18.1 ± 1.1[a,b]	17.4 ± 1.3[a,b]	19.8 ± 1.4[a,b,c]	18.4 ± 1.2[a,b]
Training	13.6 ± 1.0	13.5 ± 0.9	15.3 ± 1.0	15.2 ± 0.9	16.7 ± 0.9[a]	16.6 ± 0.9[a]	17.2 ± 0.6[a,b]	17.5 ± 0.7[a,b]	17.3 ± 0.6[a,b]	17.9 ± 0.7[a,b]
RER										
Control	0.93 ± 0.02	0.93 ± 0.02	0.95 ± 0.01[a]	0.96 ± 0.01[a]	0.97 ± 0.02[a,b]	0.98 ± 0.01[a,b]	0.98 ± 0.02[a,b,c]	0.99 ± 0.01[a,b,c]	1.00 ± 0.02[a,b,c,d]	1.00 ± 0.01[a,b,c,d]
Training	0.96 ± 0.02	0.94 ± 0.02	0.97 ± 0.02	0.97 ± 0.01[a]	0.98 ± 0.02[a]	0.99 ± 0.01[a,b]	1.00 ± 0.02[a,b,c]	1.00 ± 0.01[a,b,c]	1.00 ± 0.02[a,b,c]	1.00 ± 0.01[a,b,c]
Heart Rate										
Control	123 ± 4	119 ± 4	137 ± 4[a]	136 ± 4[a]	150 ± 4[a,b]	149 ± 4[a,b]	163 ± 4[a,b,c]	163 ± 4[a,b,c]	175 ± 4[a,b,c,d]	175 ± 5[a,b,c,d]
Training	116 ± 5	113 ± 4	132 ± 5[a]	127 ±4[a]	145 ± 4[a,b]	141 ±4[a,b]	158 ± 4[a,b,c]	154 ± 4[a,b,c]	171 ± 4[a,b,c,d]	168 ± 4[a,b,c,d]
RPE										
Control	10 ± 1	10 ± 1	12 ± 1[a]	12 ± 1[a]	14 ± 1[a,b]	14 ± 1[a,b]	16 ± 1[a,b,c]	16 ± 1[a,b,c]	17 ± 1[a,b,c,d]	17 ± 1[a,b,c,d]
Training	9 ± 1	9 ± 1	11 ± 1[a]	11 ± 1[a]	13 ± 1[a,b]	13 ± 1[a,b]	15 ± 1[a,b,c]	15 ± 1[a,b,c]	16 ± 1[a,b,c,d]	16 ± 1[a,b,c,d]

a=significant difference compared with 40% work rate, b=significant difference compared with 50% work rate, c=significant difference compared with 60% work rate, d= significant difference compared with 70% work rate.

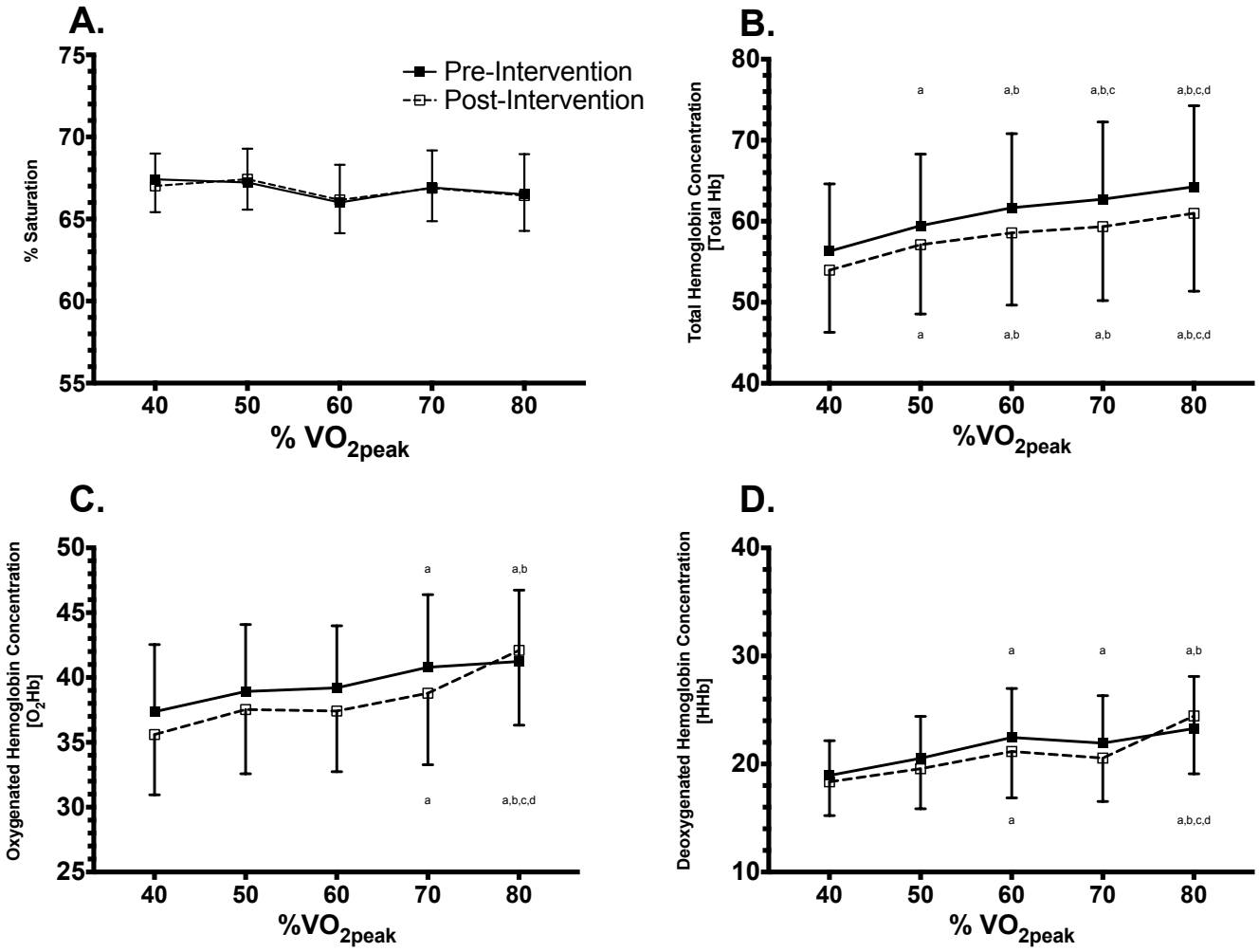

Figure 9. Near-Infrared Spectroscopy (NIRS) measurements pre vs post-intervention in the Control group (Mean ± SE). Solid lines with closed symbols represent pre-intervention. Dashed lines with open symbols represent post-intervention. There were no differences pre vs post-intervention ($p>0.05$). However, there were significant differences across work rates ($p<0.05$) a>40%; b>50%; c>60%; d>70%

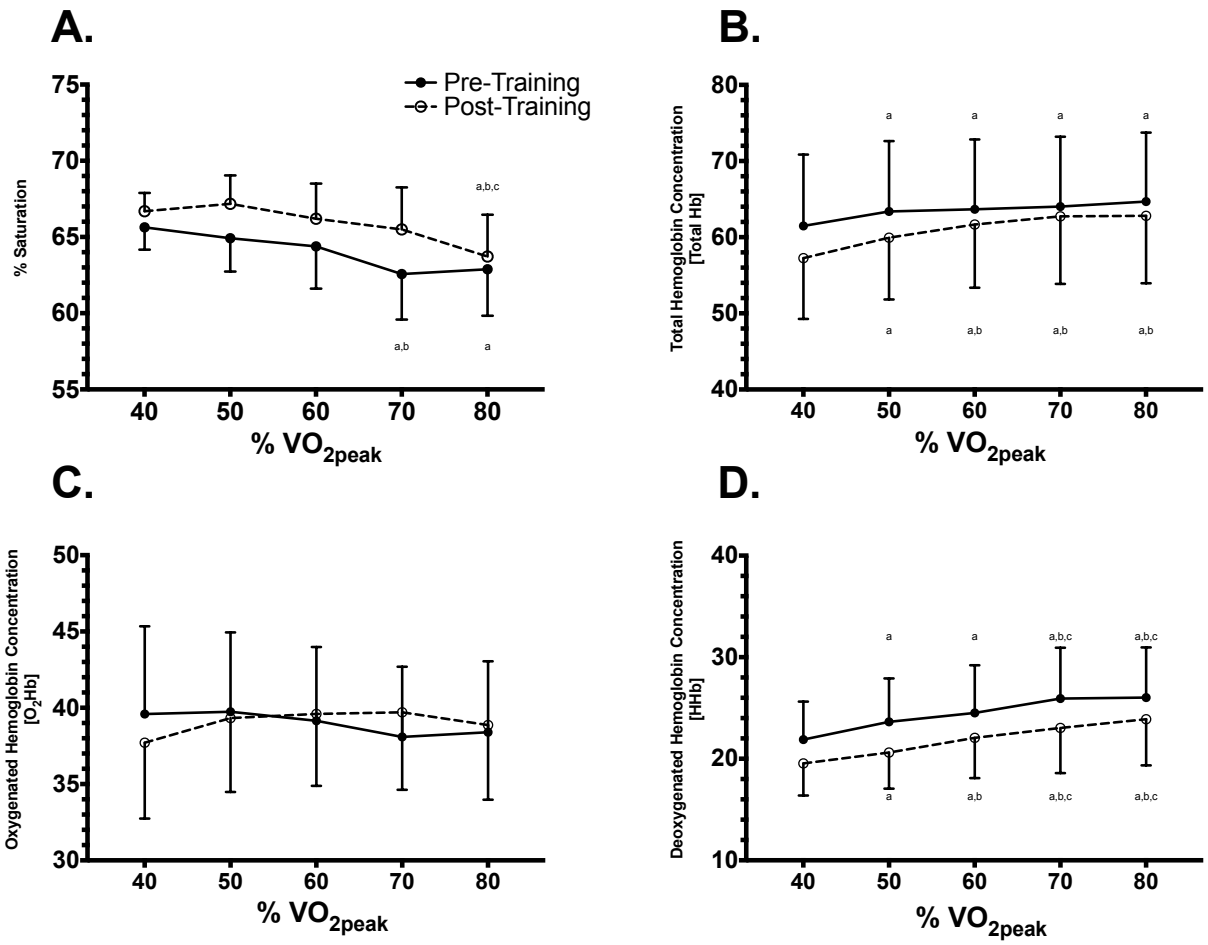

Figure 10. Near-Infrared Spectroscopy (NIRS) measurements pre vs post-intervention in the Training group (Mean ± SE). Solid lines with closed symbols represent pre-intervention. Dashed lines with open symbols represent post-intervention. There were no differences pre vs post-intervention ($p>0.05$). However, there were significant differences across work rates ($p<0.05$) a>40%; b>50%; c>60%; d>70%

Chapter VI: General Summary

The purpose of this dissertation was to identify novel factors that influence cycling lactate threshold (LT_{VO2}) and peak oxygen consumption ($VO_{2peak\text{-}cycling}$) in well-trained cyclists as well as non-cyclists. The specific aims were to determine: 1) the relative joint power contribution, muscle activation, and muscle oxygenation differences between high (HLT) and low (LLT) LT_{VO2} cyclists and 2) the effect of short-term (5 days) maximal power training on $VO_{2peak\text{-}cycling}$ in non-cyclists.

In study 1, it was shown HLT cyclists have an increased relative hip joint power contribution and decreased EMG activation of the knee extensors. This agrees with the idea that spreading the metabolic work rate reduces lactate production because recruiting a larger mass of muscle for a given work rate reduces the stress per kg of muscle activated in HLT compared with LLT cyclists. In study 2, it was demonstrated that non-cyclists have a lower maximal oxygen consumption while cycling (i.e. $VO_{2peak\text{-}cycling}$) compared with treadmill running (i.e. VO_{2max}) and maximal-power training effectively increased $VO_{2peak\text{-}cycling}$. This type of training required maximal neuromuscular recruitment while limiting the cardiovascular training stimulus, and therefore any changes found in $VO_{2peak\text{-}cycling}$ following the training program were likely a result of an increased ability to recruit additional muscle mass while cycling. Taken together these studies suggest both submaximal LT_{VO2} in trained cyclists and peak oxygen consumption ($VO_{2peak\text{-}cycling}$) in non-cyclists can be influenced by 'cycling strategy' of muscle recruitment. Future studies should directly assess the changes in relative joint power contribution and active muscle mass recruitment following traditional aerobic exercise regimens and the role they play in LT_{VO2} and VO_{2max}. This is especially important in studies which have non-cyclists begin a cycling aerobic training intervention as the initial increases could potentially be increases in VO_{2peak} rather than VO_{2max}.

In summary, in well-trained and non-cyclists, both LT_{VO2} and VO_{2peak} are influenced by the cycling strategy. These findings provide guidance for future research integrating physiological and biomechanical methodologies of human cycling performance.

Chapter VII: Review of Literature

DETERMINANTS OF CYCLING ENDURANCE PERFORMANCE

Endurance cycling performance is primarily determined by the oxygen consumption at lactate threshold (LT_{VO2}) which is largely a function of maximal oxygen uptake (VO_{2max}) and skeletal muscle oxidative capacity. The power/velocity that an athlete maintains during competition is influenced by the integration of physiological and biomechanical factors. Identifying novel factors that determine an individual's VO_{2max} and LT_{VO2} has wide-ranging implications for cycling performance (Coyle 1995, Joyner et al. 2008).

Maximal aerobic capacity sets the upper limit of endurance performance. Endurance performance can be predicted by an individual's VO_{2max} in a heterogeneous group of athletes (Farrell et al. 1979). However, a weak relationship between VO_{2max} and endurance performance exists when VO_{2max} is homogenous among subjects (Coyle et al. 1991). Instead, the LT_{VO2} that can be sustained for the duration of the endurance event dictates performance when VO_{2max} is homogenous across subjects (Coyle et al. 1991).

For endurance events lasting longer than five minute the majority of the race is performed below an individual's VO_{2max}. Endurance athletes can exercise for long periods of time at a steady state value of oxygen consumption with little accumulation of blood lactate so long as the intensity remains low (i.e. below 70% of VO_{2max}) (Costill et al. 1973). It has been previously shown that LT_{VO2} is significantly related to endurance performance, and is a better indicator of endurance performance when the VO_{2max} of a group of athletes becomes more homogeneous (Coyle et al. 1991). As endurance athletes progress through training they can improve VO_{2max} and LT_{VO2} (Hickson et al. 1977, Hickson et al. 1981); these improvements are a result of cardiovascular and skeletal muscle adaptation aimed at improving oxidative capacity (Holloszy et al. 1984).

Maximal oxygen consumption (VO_{2max}) is the highest rate at which the body can use oxygen during intense, whole body exercise, and is frequently used as an indicator of cardiorespiratory fitness(Astrand et al. 1961). Factors central (i.e. pulmonary diffusing capacity, cardiac output, and oxygen carrying capacity of the blood) and peripheral (i.e. skeletal muscle oxidative capacity) in nature determine VO_{2max} (Bassett et al. 2000). This review will discuss those factors limiting VO_{2max} in apparently healthy individuals exercising at sea level, as well as the ability of VO_{2max} to determine endurance performance.

Cardiac Output

Differences in VO_{2max} between untrained and trained individuals is largely due to differences in maximal cardiac outputs, with higher VO_{2max} values seen in trained individuals (Saltin et al. 1968). The higher cardiac outputs in trained compared with the untrained state are due to variations in stroke volume rather than variations in maximal heart rate (Ekblom et al. 1968). Less of the variation in VO_{2max} is due to oxygen extraction by active skeletal muscle as little differences in oxygen extraction exist between trained and untrained individuals. Therefore, the large increases in VO_{2max} which occur following aerobic endurance training are accompanied by increases in maximal cardiac output, with only small changes in A-$VO_{2difference}$ (Ekblom et al. 1968, Saltin et al. 1968, Saltin 1985).

Available evidence suggests cardiac output is the primary limiter of VO_{2max} in healthy individuals. During single leg knee extensor exercise the oxygen uptake of the knee extensors is 2-3 times greater than knee extensor oxygen consumption during whole body maximal exercise (Saltin et al. 1976). During whole body exercise, working skeletal muscle has the capacity for increasing blood flow and oxygen consumption beyond the capacity of the heart (i.e. cardiac output), suggesting VO_{2max} is limited by the ability to deliver oxygen to the working muscle and

rather than the ability of the mitochondria to consume oxygen. Taken together, this data further indicates maximal cardiac output is the principle factor limiting VO_{2max} in healthy individuals.

Mitochondrial Adaptations

Following endurance training a ~2-fold increase in mitochondrial enzyme activity is associated with only a ~30% increase in VO_{2max} (Saltin et al. 1977). Rather, the increases in mitochondrial enzymes following training results in increases fat oxidation and decreases lactate production, for a given work rate, as opposed to improvements in VO_{2max} (Holloszy et al. 1984). This is consistent with the theory that VO_{2max} is limited by oxygen delivery (i.e. cardiac output) rather than oxygen utilization (i.e. mitochondrial adaptations). Further, the mitochondrial adaptations following endurance training have larger effects on endurance performance rather than VO_{2max}, as athletes with similar VO_{2max} values can vary up to two-fold in mitochondrial enzymes (Coyle et al. 1991). Mitochondrial adaptations and their relationship to endurance performance and submaximal exercise will be discussed in detail later in this review.

Differences in VO_{2max} between Cycling and Running

In recreationally active individuals VO_{2max} on a cycle ergometer is ~10% lower than the VO_{2max} achieved during treadmill running (Astrand et al. 1961, Withers et al. 1981, Mikesell et al. 1984). It is generally accepted that the lower VO_{2max} while cycling compared to running is due to less active muscle mass while cycling. Importantly, the reduced muscle mass recruitment while cycling likely results in a reduced cardiac output leading to lower VO_{2max} (Faulkner et al. 1971, Reybrouck et al. 1975). However, research investigating whether differences in VO_{2max} between cycling and running are a result of reduced cardiac output or A-$VO_{2difference}$ have found mixed results (Hermansen et al. 1969, Hermansen et al. 1970, Faulkner et al. 1971).

When comparing VO_{2max} between treadmill running and cycling, among well-trained runners and cyclists it appears that training significantly influences the maximal capacity achieved with each test (Millet et al. 2009). Trained runners tend to have higher VO_{2max} values while treadmill running compared to cycling, while trained cyclists can achieve VO_{2max} values while cycling approaching or equaling those while treadmill running (Millet et al. 2009). Therefore, that ability of well-trained cyclists to reach high VO_{2max} values while cycling has been attributed to their ability to recruit large muscle mass while cycling (Tanaka et al. 1987, Tanaka 1994). Additionally, in individuals with VO_{2max} values that are lower on a cycle ergometer compared to treadmill running increasing the *maximal* values of cardiac output and/or A-$VO_{2difference}$ *per se* are not necessary for improvements in the VO_{2max} while cycling. Rather, the 'narrowing of the gap' between cycling VO_{2max} and treadmill VO_{2max}, can be achieved through increases in muscle mass recruitment leading to increases in *peak* cardiac output while cycling.

Endurance Performance

Endurance performance can be predicted by VO_{2max} among individuals with heterogeneous VO_{2max} values (Bassett et al. 2000, Joyner et al. 2008). However, individuals with homogenous VO_{2max} values restricts the range of VO_{2max} values and therefore the ability for VO_{2max} to predict endurance performance (Coyle et al. 1988, Coyle et al. 1991). Rather, among athletes with similar VO_{2max} the 'performance VO_2,' or highest steady state of oxygen consumption that can be maintained for the duration of the endurance event, is the primary predictor of performance (Coyle et al. 1988).

Oxidative Capacity and Lactate Threshold

As exercise intensity increases blood lactate concentration progressively increases as muscles contract to meet exercise demand. ATP is converted to ADP and Pi which are then used

to drive metabolic reactions in the cell to meet the demand of the exercising muscle. The increase in ADP concentration increases glycolysis and carbohydrate oxidation leading to accumulation of pyruvate and NADH, and subsequently and increase in lactate production/accumulation (Holloszy et al. 1984). Improving skeletal muscle oxidative capacity through endurance training delays the production and subsequent accumulation of lactate for a given submaximal work rate leading to increases in lactate threshold (Ivy et al. 1980, Holloszy et al. 1984). Therefore, increases in lactate threshold are accomplished primarily through the increased oxidative capacity of skeletal muscle (Ivy et al. 1980).

A muscle's oxidative capacity and it's absolute and relative lactate thresholds are highly correlated (Ivy et al. 1980). Improved oxidative capacity in response to chronic endurance training is due to increases in mitochondria number, size, and enzyme activity in response to chronic endurance exercise (Holloszy 1967, Holloszy 1973, Ivy et al. 1980). These mitochondrial adaptations lead to increased pyruvate and fat oxidation at both relative and absolute work rates eliciting reduced carbohydrate oxidation in the trained state (Holloszy 1973, Holloszy et al. 1976, Holloszy et al. 1984).

The absolute oxygen requirements for a given submaximal work rate may be similar between trained and untrained individuals, but there can be more than a two-fold difference in muscle oxidative capacity. Thus, to maintain the same rate of oxygen utilization, the oxidative work done per mitochondria is less in trained individuals. Consequently, lower ADP concentration, higher ATP, and higher phosphocreatine concentrations are present in trained muscle at the same absolute work rates (Karlsson et al. 1972). As a result of the higher ATP/ADP ratio skeletal muscle glycolysis is less activated, allowing well-trained athletes to exercise at higher relative work rates for a given level of intramuscular lactate compared with untrained counterparts (Hurley et al.

1984). Additionally, research examining the adaptations to detraining have shown parallel decreases in oxidative enzyme capacity and LT_{VO2}, further supporting the role of skeletal muscle oxidative capacity in determining LT_{VO2} (Coyle et al. 1985).

Due to the tight coupling of oxidative capacity and LT_{VO2} researchers have suggested that skeletal muscle fiber type may determine differences in LT_{VO2} of endurance athletes. In untrained individuals, the oxidative capacity of Type I muscle fibers is nearly double that of Type II suggesting fiber type composition may play a role in determining LT_{VO2} in an untrained population (Chi et al. 1983). In fact, long distance runners tend to have a greater percent Type I fiber compared with sprinters (Gollnick et al. 1972). However, the oxidative capacity of Type I & Type II fibers are similar in well-trained endurance athletes suggesting fiber type percentage plays little to no role in determining LT_{VO2} in this population (Figure 11)(Chi et al. 1983).

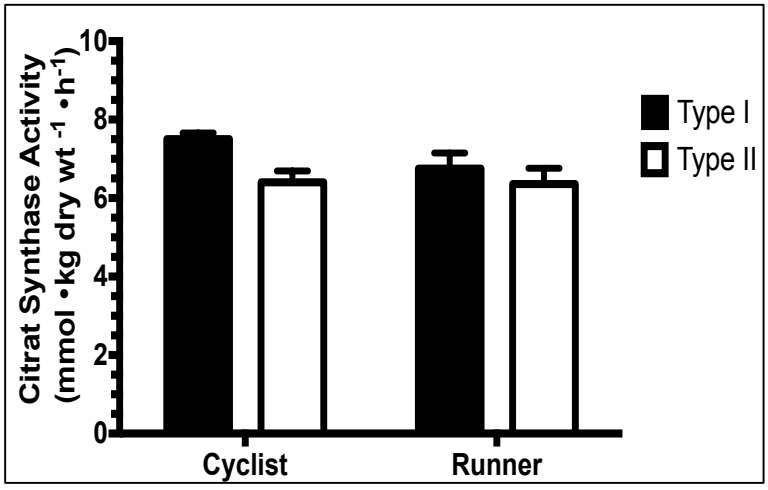

Figure 11. Oxidative capacity comparisons of Type I and Type II fibers in well-trained endurance athletes (Chi et al. 1983).

In summary, the two primary physiological adaptations to endurance training are increases in the oxygen delivery to and oxidative capacity of the skeletal muscle leading to improvements in VO_{2max} and LT_{VO2}. VO_{2max} is a determinant of endurance performance in a heterogeneous group

of athletes, but less predictive in a group of well-trained athletes that present with similar maximal oxygen consumption (Coyle et al. 1991). Furthermore, LT_{VO2} is coupled to skeletal muscle oxidative capacity as evidenced by parallel decreases in oxidative enzyme capacity and LT_{VO2} following detraining (Coyle et al. 1985). Although improvements in oxidative capacity explain a large percentage of adaptations to endurance training, muscle recruitment, active muscle mass, and mode of exercise appear also to play a role in determining and endurance athletes LT_{VO2}.

Motor Unit Recruitment and Lactate Threshold

Available evidence suggests that the appearance of lactate in the blood occurs in conjunction with a progressive recruitment of larger motor units (i.e. motor units with greater glycolytic capacity). Compelling evidence for this phenomenon is demonstrated by a non-linear increase in integrated EMG signal corresponding to the point of blood lactate threshold in cycling and running (Nagata et al. 1981, Taylor et al. 1994). It has been hypothesized that increased recruitment and/or increased firing of fast glycolytic muscle fibers leads to increases in integrated EMG signals. A progressive recruitment in this fiber type would lead to an increased lactate production for a given work rate due to the lower oxidative capacity, greater glycolytic capacity, and reduced mitochondrial density of the fast glycolytic fibers compared with slow oxidative fibers in untrained subjects (Gollnick et al. 1972). Since trained athletes have similar oxidative capacity in both fiber types it is unlikely that a progressive recruitment of Type II fibers leads to increased production of lactate, as prior research has shown Type II fibers are recruited even at low exercise intensities and their selective recruitment does not lead to accumulation of lactate (Ivy et al. 1987). Furthermore, with sufficient stimulus isolated preparations (without fast glycolytic fibers) show a clear lactate threshold (Connett et al. 1986), suggesting the increased iEMG may be a consequence, as opposed to the cause, of lactate threshold. While EMG data can reveal important insights into

the relative intensity of contraction and activation timing, it is unable to determine the amount of muscle used or the physiological stress placed on the working muscle. Therefore, using EMG data in conjunction with markers of physiological stress might allow for a more comprehensive understanding of muscle recruitment and lactate threshold.

Muscle Mass Utilization and Lactate Threshold

Much like the way in which increasing the mitochondria content allows for reduced oxidative work per mitochondria, the distribution of power to a greater percentage of muscle mass should, in theory, increase the number of active mitochondria performing oxidative metabolism. This, in turn, would reduce the oxygen demand per mitochondria, decrease reliance on glycogenolysis, and lower overall lactate production during submaximal exercise (Coyle et al. 1988).

In support of this hypothesis, it's been found that well-trained cyclists with high LT_{VO2} use approximately 1.8kg more muscle mass during cycling compared with low LT_{VO2} cyclists despite having similar VO_{2max}. The muscle mass used (kg) explained approximately 20% of the variance in the LT_{VO2} (Coyle et al. 1988, Coyle 1995). The authors attributed the increased LT_{VO2} to the improved ability to "spread the work" to a greater amount of muscle mass noting the greater number of years spent cycling as a possible contributor (Coyle et al. 1988).

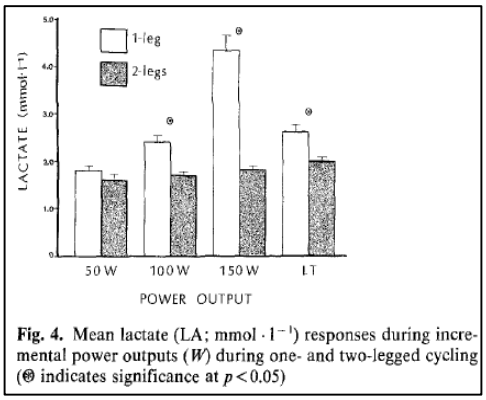

Fig. 4. Mean lactate (LA; mmol · l⁻¹) responses during incremental power outputs (*W*) during one- and two-legged cycling (⊛ indicates significance at $p < 0.05$)

Figure 12. Lactate responses during one- and two-legged cycling (Neary et al. 1986).

Previous research has compared lactate response between exercise involving varying amounts of muscle mass (Davis et al. 1976, Neary et al. 1986, Richter et al. 1988, Savard et al. 1989). Lactate concentration is higher for a given work rate in 1-leg vs 2-leg cycling (Neary et al. 1986) (Figure 12), which is partially explained by the usage of more muscle mass (Richter et al. 1988). However, studies have shown similar $LT_{\%VO2}$ between 1-leg and 2-leg cycling suggesting active muscle mass plays little role in determining lactate threshold (Stamford et al. 1978). Rather, the mode of exercise is likely a more relevant factor in determining LT_{VO2} (Davis et al. 1976). When comparing arm cycling, leg cycling, and treadmill running $LT_{\%VO2}$ has been shown to occur 46%, 63%, and 58% respectively (Davis et al. 1976). In addition, well-trained cyclists exhibit lower LT_{VO2} when running on level ground compared to cycling, suggesting that running is a learned skill (Mazzeo et al. 1989, Bouckaert et al. 1990) and uses different muscles than cycling (Costill et al. 1971, Costill et al. 1974, Costill et al. 1976). However, well-trained cyclists running on a 10% incline demonstrate a comparable LT_{VO2} to that seen while cycling (Coyle et al. 1988) due to the similarities in muscle groups used during uphill running and cycling (Costill et al. 1974).

The distribution of muscle mass contributing to power accomplished should be viewed in a separate context to the electromyographic examination of muscle activity. EMG has been used

to examine the extent of motor unit recruitment during progressive increases in endurance exercise intensity and its relationship to blood lactate threshold; however, EMG fails to quantify the volume of muscle mass activated. Additionally, the increase in EMG associated with lactate threshold could be a result of the decreased pH which reduces force production of individual motor units, signaling increased motor unit recruitment to compensate for the reduced force (Nagata et al. 1981).

Muscle Utilization Patterns During Exercise

Exercise intensity, training status, and fiber type all play a role in determining glycogen utilization of the working muscle. However, due to the invasive nature of muscle glycogen assessment via muscle biopsies few studies have examined differences between muscle groups during exhaustive exercise. Those studies that have examined glycogen usage between muscles have found that glycogen depletion is related to the involvement of the particular muscle in performing work (Costill et al. 1971, Costill et al. 1971, Costill et al. 1974).

Glycogen usage occurs at all exercise intensities and increases progressively as the intensity of exercise increases. Moderate intensity exercise (i.e. ~65% of VO_{2max}) performed to exhaustion (~2-3 hours) can markedly reduce muscle glycogen. However, high intensity exercise (I.e. 90% of VO_{2max}), which can typically only be performed for 30-60 minutes, also shows reductions in glycogen equal to that observed with long duration, moderate intensity exercise. Additionally, in regards to glycogen depletion, fiber types respond differently to exercise intensity and duration. Type I fibers are used throughout all exercise intensities and demonstrate reduced glycogen levels during exercise of moderate and high intensity (Gollnick et al. 1973, Gollnick et al. 1973, Gollnick et al. 1974). However, Type II fibers appear to lose glycogen only during moderate intensity exercise if the exercise is performed until exhaustion (Gollnick et al. 1973,

Gollnick et al. 1973). Additionally, as exercise intensity increases above 90% VO_{2max} Type II will reduce their glycogen content as much as, or more than, Type I fibers (Gollnick et al. 1974); this suggests that Type II are recruited during exhaustive, moderate intensity exercise and short periods of high intensity exercise.

Variations in glycogen depletion are not limited to differences in exercise intensity, exercise duration, or fiber type. Glycogen depletion varies between active muscles which contribute different amounts of work to the exercise task. It was once thought that distance running to exhaustion did not result in complete glycogen depletion, as significant decrements in muscle glycogen levels of the vastus lateralis were not seen with endurance running (Costill et al. 1971, Costill et al. 1971); whereas cycling elicited large decreases in muscle glycogen at the same relative exercise intensity in the vastus lateralis (Coyle et al. 1988). However, the studies examining runners assumed sampling from the vastus lateralis was appropriate due to the large reductions seen in cycling. When directly comparing glycogen use of the vastus lateralis during level and uphill running the glycogen depletion of the vastus lateralis during uphill running is elevated compared to level running suggesting total work performed by the vastus lateralis during level running is reduced (Costill et al. 1974). Therefore, studies using both cycling and running protocols to determine VO_{2max} and LT_{VO2} should have the athletes run uphill to utilize similar muscles recruited during cycling. This uphill protocol has been used as an indicator of cycling skill, as low LT_{VO2} while cycling and high LT_{VO2} while uphill running indicate that those athletes have the capacity for a higher cycling LT, yet something is preventing a high LT_{VO2} while cycling (Coyle et al. 1988). Although direct determination of muscle glycogen via muscle biopsy provides a precise physiological measure of muscle use it is invasive to implement. More recently,

researchers have used a non-invasive technique to determine oxygenation status of working muscle which provides an indication of the physiological stress of the muscle while exercising.

Muscle Oxygenation During Cycling

Near-infrared spectroscopy (NIRS) is a non-invasive method used to assess oxygenation status of working skeletal muscle. The chromophores of O_2Hb and HHb have different optical properties of absorbing near-infrared light and can be detected with NIRS. The initial amplitude of light from the NIRS transmitter is attenuated by the HHb within tissue and the resultant amplitude is influenced by the absorption coefficient, concentration of the tissue, and distance, in accordance with the Beer-Lambert law. After being detected by the receiver, the index of the regional deoxygenation status is converted to Hb, allowing for the determination of the absolute concentration of oxygenated hemoglobin (O_2Hb) and deoxygenated hemoglobin (HHb). Further, percent saturation (%Sat) is calculated by the ratio of O_2Hb and the total Hb concentration (THb)=O_2Hb + HHb.

Concentrations of oxygenated (O_2Hb) and deoxygenated (HHb) hemoglobin reflect the balance between O_2 delivery and O_2 consumption at the muscle level. NIRS has been used previously to determine the relative physiological stress placed (i.e. Δ in HHb) on the muscle in response to changes in work rate (Skovereng et al. 2016) and cadence (Boone et al. 2015, Skovereng et al. 2016) during cycling.

Moreover, NIRS has previously been used to detect the relationship between local physiological stress (i.e. HHb) and muscle activation (i.e. iEMG). Despite a linear increase in iEMG with increasing work rates HHB plateaus at high work rates suggesting changes in HHb are independent of changes in muscle activation, but rather are reflective of O_2 delivery and O_2 uptake

at the local level (Chin et al. 2010). In this context, NIRS can be used as an indicator of 'physiological stress' at the muscle level.

BIOMECHANICS OF CYCLING

Muscle Activity While Cycling

Surface electromyography (EMG) has been used to measures changes in muscle activation during cycling in response to changes in saddle height (Ericson et al. 1985), cadence (Ryan et al. 1992, Marsh et al. 1995, Neptune et al. 1997, MacIntosh et al. 2000, Baum et al. 2003), work rate (Ericson 1986, Ryan et al. 1992, MacIntosh et al. 2000), and cycling skill level (Chapman et al. 2006, Chapman et al. 2008, Chapman et al. 2009).During steady state cycling, muscle activation occurs in a predictable, cyclical fashion reflective of the movement itself, with activation and deactivation of lower extremity muscles occurring in specific regions of the crank cycle. By determining the crank location at top dead center (TDC) and bottom dead center (BDC) EMG profiles can be obtained as a function of time expressed in a percentage of the total duration of the crank cycle. Knee extensors (e.g. vastus lateralis (VL), vastus medialis (VM), and rectus femoris (RF)) are first activated close to top dead center (e.g. ~0°/TDC of crank cycle). Hip joint extensors (e.g. gluteus maximus (Gmax)) activate later in the crank cycle (e.g. 90° of crank cycle) (Hug et al. 2009). A majority of the mechanical energy produced during the down stroke (~86%) is accomplished primarily from the gluteus maximus and the vastii muscle groups (Neptune et al. 1997, Raasch et al. 1997, Neptune et al. 2000). However, only 55% of the energy produced by the gluteus maximus and vastii are directly delivered to the crank, rather 45% of the energy is delivered to the limbs (Raasch et al. 1997, Neptune et al. 2000). A combination of plantar flexor and knee flexor activation occurs towards the bottom of the down stroke (e.g. 180°/BDC of crank cycle) where the plantarflexors help to transfer the energy produced from the gluteus maximus and vastii

towards the acceleration of the crank (Gregor et al. 1991, Neptune et al. 1997, Raasch et al. 1997, Hug et al. 2009).

Activation timing appears to be little influenced by changes in power output (Jorge et al. 1986). However, pedaling rate appears to have a large influence on activation timing of lower extremity muscles. As pedaling rate increases a general shift in peak activity to earlier in the crank cycle occurs (Marsh et al. 1995), with onset and offset timing appearing earlier in the Gmax, BF, RF, and VM (Neptune et al. 1997). Although activation timing provides useful information as to when muscle is active while cycling, EMG is less informative regarding the relative intensity of muscle activation. The intensity of muscle activation during pedaling is typically quantified by taking the root mean square (RMS)(Dorel et al. 2009) or integrated EMG (iEMG) (Ericson et al. 1985, Takaishi et al. 1998) of the raw EMG data and normalizing the values to maximum value to account for between subject differences in raw EMG values (Hug et al. 2008, Hug et al. 2009).

Increases in work rate (i.e. increased resistance by ergometer) elicit dramatic changes in EMG activity of select lower limb muscles while cycling (Ericson 1986, MacIntosh et al. 2000, Hug et al. 2009). Constant-load exercise performed at different intensities while controlling for cadence and fatigue allows for accurate comparison of changes in muscle activity across work rates. Large increases in muscle activity of the primary muscles used while cycling (i.e. gluteus maximus, vastus lateralis, rectus femoris, vastus medialis, and biceps femoris) were found when work rate was progressively increased from 120-240W. Similarly, increasing work rate elicits large increases in gluteus maximus activity as well as increases in total lower extremity joint activity (Figure 13 & 14, respectively) (MacIntosh et al. 2000).

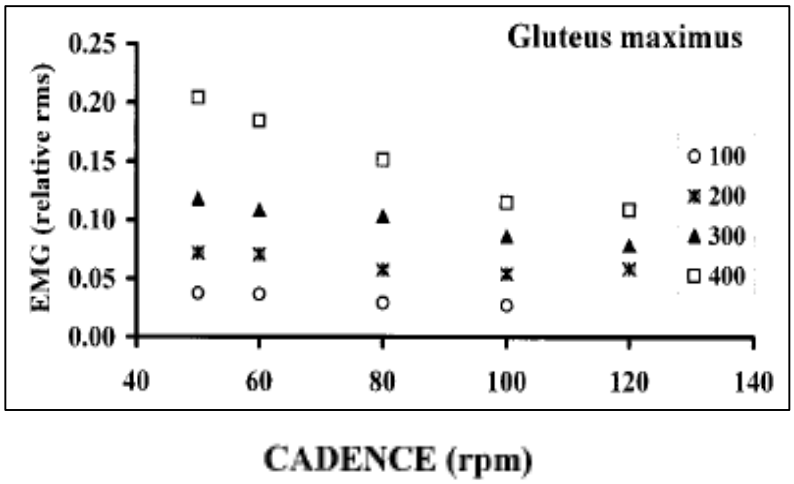

Figure 13. Gluteus maximus EMG activity with changes in cadence and work rate (MacIntosh et al. 2000).

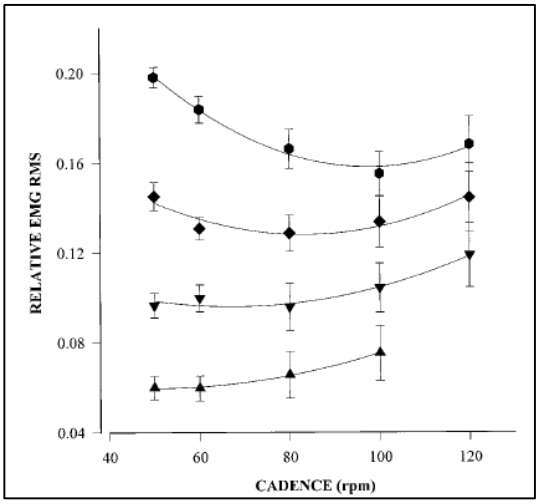

Figure 14. Changes in total lower extremity muscle activity in response to changes in cadence and work rate. Lines represent power outputs of 100,200,300, & 400 watts from bottom to top, respectively (MacIntosh et al. 2000).

Muscle activity is not only influenced by the mechanical load, but the interaction of cadence and work rate while cycling as well (Marsh et al. 1995, MacIntosh et al. 2000). There have been large discrepancies in the literature regarding the EMG response to increased cadence (Ericson et al. 1985, Neptune et al. 1997, MacIntosh et al. 2000, Sarre et al. 2003). Muscle activity

changes while cycling are influenced by the power output at which the subject is cycling, which could help explain the discrepancies in the effects of cadence on muscle activation seen in previous studies (MacIntosh et al. 2000). When cycling at relatively low power outputs, increases in cadence and muscle activation for most lower extremity muscles are linearly related (Ericson et al. 1985); however, at higher work rates (300-400W) increasing cadence decreases activation of the VL and Gmax (Lucia et al. 2004), as well as total lower extremity muscle activation (MacIntosh et al. 2000). Therefore, it has been hypothesized a minimal muscle activation for a given work rate occurs and can be influenced by cadence.

Joint Contribution While Cycling

External work rate increases elicit increases in the absolute joint power of the hip, knee, and ankle (Elmer et al. 2011, Skovereng et al. 2016). However, the relative contribution of the hip, knee, and ankle to total lower extremity power have differing responses to increases in work rate (Elmer et al. 2011, Skovereng et al. 2016, Aasvold 2017). Relative hip joint contribution has been shown to increase (Skovereng et al. 2016, Aasvold 2017) or remain constant (Elmer et al. 2011) with increases in work rate. Relative knee extensor contribution decreases as the external work rate increases (Elmer et al. 2011, Skovereng et al. 2016), and the relative ankle contribution remains constant with changes in work rate despite increases in absolute ankle power with increasing work rates (Elmer et al. 2011, Skovereng et al. 2016, Aasvold 2017). The largest contributors to power generation during cycling comes from the muscles of the hip and knee joints (Raasch et al. 1997, Neptune et al. 2000, Korff et al. 2009, Martin et al. 2009, Skovereng et al. 2016). While the total joint power generated from the hip and knee extensors increases linearly with increases in external work rate the relative contribution of the hip extensors increases and the

relative knee contribution decreases with increasing work rate in trained cyclists (Skovereng et al. 2016).

Cadence has varying effects on relative joint contribution during cycling and is largely dependent on the work rate selected to assess cadence. Prior research has found decreases in the hip joint specific power and increases in knee joint-specific power with increased cadences in recreational cyclists (Skovereng et al. 2016), which is in agreement with research in which both professional and recreational cyclists were compared (Aasvold 2017). However, others have found increased absolute and relative joint contribution from both the hip and knee joints when combining high cadences with maximal work rates (McDaniel et al. 2014) or no difference in absolute or relative hip or knee contribution with increased cadence (Bini et al. 2010). The discrepancies between studies are likely due to the work rates utilized: with high cycling cadence, hip joint specific power is decreased with low work rates and increased with high work rates (McDaniel et al. 2014, Skovereng et al. 2016). The decrease in the hip joint power at low work rates is in line with EMG data of the gluteus maximus: with high cycling cadence, gluteus maximus activity is decreased with low work rates and increased with high work rates (MacIntosh et al. 2000). Therefore, to determine the independent effect of cycling experience or work rate on relative joint contribution during submaximal cycling researchers should control for cadence to minimize the effects on relative joint contribution.

Joint Contribution and Cycling Experience

Determining changes in the relative joint contribution in response to changes in work rate or cadence play an important role in identifying how the body accommodates changes in cycling task demands; however, comparing individuals' relative joint contribution may provide insight in how the body adapts to long-term cycling training. To date studies which have attempted to

determine relative joint contribution between groups of individuals have focused on 'cycling skill' typically defined as an individual who is an experienced or novice cyclists or by comparing cyclists to triathletes (Hoshikawa 2007, Bini et al. 2014). While cycling at a constant work rate, experienced cyclists have a lower relative contribution from the hip and higher relative knee contribution compared with recreationally active individuals (Hoshikawa 2007). However, the work rate chosen by Hosikawa 2007 was 200W, which is a relatively high work rate for the recreationally active subjects and a relatively low work rate for experienced cyclists. Therefore, differences in relative joint contribution could be due to experienced cyclists generating the necessary power by utilizing primarily the knee extensors, while recreational active individuals would rely more on both the hip and knee joints to produced power since it is closer to their maximal intensity and relative hip and knee joint contribution increase with increases in work rate (Skovereng et al. 2016).

Studies which have examined differences in relative joint contribution between cyclists and triathletes have found no difference in hip contribution or knee contribution between groups at higher work rates; closer to what would be experienced in a race (i.e. ~275 Watts) (Bini et al. 2014). Recently, researchers have compared differences in relative joint contribution between professional (i.e. Continental/World Tour) cyclists with recreational riders with prior cycling experience while exercising at similar relative work rates (i.e. 55, 85, and 100% LT_{watts}) (Aasvold 2017). The professional riders had higher relative hip and lower relative knee joint contribution at all work rates compared to the recreational riders, which suggests well-trained riders develop a strategy consistent with increasing hip contribution and decreasing knee contribution as they become more skilled (Aasvold 2017). Due to the large differences in athletic ability between groups the differences in lactate threshold between the professional riders (~315 W) and

recreational cyclists (~275 W) were likely a result of differences in maximal capacities of oxygen delivery and/or consumption rather than differences in a joint contribution (Aasvold 2017).

Future Directions

Prior research comparing subjects based on 'cycling skill' have done so with binary definitions of skill (i.e. cyclists or non-cyclists), years of cycling experience (i.e. competitive/professional vs novice/recreational), or competitive athlete type (i.e. cyclist vs triathlete). When comparing individuals based on 'skill', subject populations can have large differences in VO_{2max} and/or LT_{VO2} capabilities (Aasvold 2017) or researchers have often failed to report VO_{2max}, LT_{VO2}, or both (Hoshikawa 2007, Bini et al. 2014). To date, no study has systemically controlled for both VO_{2max} and varied LT_{VO2} of well-trained cyclists while determining relative joint power contribution. Controlling for these factors would allow for relative joint contribution to be compared at similar absolute and relative work rates, minimizing the confounding effects of work rate on relative joint contribution. This would allow researchers to compare joint power differences in athletes with similar VO_{2max} capabilities but who vary on LT_{VO2} while cycling to determine if higher or lower LT_{VO2} are associated with a specific cycling strategy (i.e. do high LT_{VO2} cyclists have a higher relative hip contribution compared to low LT_{VO2} cyclists, similar to the differences found between elite and recreational cyclists(Aasvold 2017) and in agreement with hypotheses put forward by others (Coyle et al. 1988, Bassett et al. 2000, Millet et al. 2009)?). Although joint power calculated through inverse dynamics has often been used interchangeably to describe biomechanics associated with muscular power, these joint power calculations are independent of the mechanical energy expenditure of working muscles during exercise and quantification of individual muscle contribution (Kautz et al. 1994, Neptune et al.

1998, Kautz et al. 2002). Therefore, electromyography and/or near-infrared spectroscopy can be used in conjunction with joint power calculations to determine the relative joint contribution during submaximal cycling and the effects on muscle/oxygenation and metabolic stress.

Appendices

APPENDIX A.

Methodological Techniques

Oxygen Consumption

During exercise the participants breathed through a two-way non-rebreathing valve (Hans Rudolph, Kansas City, MO). Ventilation was measured via an inspiratory pneumotachometer attached to the two-way valve (Hans Rudolph, Kansas City, MO). Expired gas samples were taken from a mixing chamber which was directly connected via capillary tubing to oxygen and carbon dioxide analyzers (Applied Electrochemistry, Models S-3A/I and CD-3A, respectively). MOXUS metabolic software (Applied Electrochemistry) was then used to continuously analyze VO_2, VCO_2, and RER throughout the exercise trials.

Near-Infrared Spectroscopy

Near-infrared spectroscopy (OxiplexTS, ISS, Champaign, IL) was used to measure oxygenated hemoglobin [O_2Hb] and deoxygenated hemoglobin [HHb] in the vastus lateralis. NIRS uses the feature that the chromophores of O_2Hb and HHb have different optical properties of absorbing near-infrared (wave length: 690 nm, 830 nm). This enables NIRS to measure the absolute concentrations of O_2Hb and HHb in μM at real-time in noninvasive manner (Ryan et al. 2012, Boone et al. 2015, Skovereng et al. 2016).

Before every test, the NIRS was calibrated after 30 minutes of warm-up. Figure 15. shows the description of the probe designed for skeletal muscle measurements and this probe was used for this study. The acquisition frequency of 2 Hz was used for this study. The NIRS data was continuously monitored and averaged for data analysis.

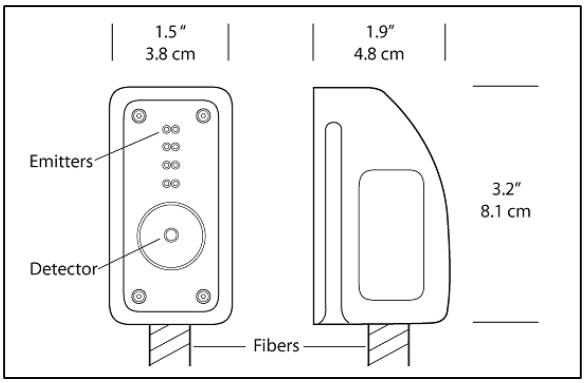

Figure 15. OxiplexTS NIRS Probe.

Blood Lactate Concentration

This protocol requires 30 min of continuous exercise at submaximal intensities (5 min at each stage of approximately 40, 50, 60, 70, 80, 90% of maximal oxygen consumption). The lactate threshold was determined from analysis of a series of blood samples obtained, between min. 4-5 in each stage. Blood samples were immediately deproteinized by placing it in 8% perchloric acid and lactic acid levels were later measured on the supernatant. Enzymatic analysis was used to determine blood lactate concentration based on methods of Farrell et al. (Farrell et al. 1979, Coyle et al. 1988). The lactate threshold was defined as the exercise intensity that elicits a 1mM increase above baseline in blood lactate concentration (Coyle et al. 1988, Coyle et al. 1991).

Blood lactate concentration was determine using the following procedures and enzymatic reactions:

Part 1: Supplies, solutions, etc.

Glassware

1. Polypropylene 12x75mm test tube Qty: 2 (per blood sample)
2. Eppendorf 1.5mL tube Qty: ((X+3)*2)

Solutions and Reagents

1. NAD (Sigma N-7004)
2. LDH (Sigma L-3916)
3. Hydrazine (Sigma H-9507)
4. Glycine (Fisher G-46)
5. Lactate Std (Sigma 826-10)

6. Perchloric Acid (Fisher A-229 70%)

PCA: to get 10%, take 71.42mL of 70% stock, bring up to 500mL with dH_2O

Glycine-Hydrazine Buffer for 1000mL

0.33M glycine25.02g

0.27M hydrazine23.98mL

Mix and bring up to 1000mL with dH_20, pH to 9.2

Page Break

Part 2: Sample preparation

Step 1: Reagent Cocktail Preparation

1. Prepare reagent cocktail- for each sample or tube
   a. 1 mL of glycine-hydrazine buffer
   b. 0.83mg of NAD
   c. 5U of LDH, if use 1000U/ml stock, need 5uL
2. If you have X blood samples:
   a. ((X+3)*2+1)*(above recipe)
   b. Need the samples, one blank, two standards, all in duplicate, plus one extra so you have enough buffer for all of your samples

Step 2: Blood Deproteinization

1. Protective gloves, glasses, and lab coat should be used when handling blood.
2. Exactly 0.5mL of whole blood should be immediately mixed with 1.5mL 8%PCA
3. Vortex the tube to fully deproteinize the sample.
4. Centrifuge at 4°C for at least 15 minutes at 3000 RPM
5. Transfer the clear supernatant to an appropriately labeled tube.
   Lactate is stable in supernatant is stable for at least one week at 2-6°C, longer if frozen.

Step 3: Supernatant/Reagent Mixture

1. Add 1mL of reagent cocktail (see Part 2, Step 1 above)
2. Add 50uL of 10% PCA to the Eppendorf 1.5Ml tubes for blank.
3. Add 50uL of two lactic acid standards to std1 and std2 eppendorf 1.5mL tubes.
4. Add 50uL of sample supernatant to sample 1 to sample N eppendorf 1.5mL tubes.
5. Vortex each eppendorf tube
6. Incubate tubes at 37°C for 45 minutes in shaking water bath at 60 RPM.

Part 3: Sample analysis

Step 1: Spectrophotometer & Calculations

1. Warm the spectrophotometer for 30 minutes, read the sample at 340nM
   a. Instrument: Spectrophotometer Beckman DU-600
   b. Method: A:\LAT
   c. Read average time: 0.5s
   d. Fixed wavelength: 340nM
   e. Factor 10.13
2. Calculations
   a. Lactate standard 40mg/100mL, 400
   b. mg/L, or 4.44mM (Sigma 826-10, now Trinity Biotech 82610)
      i. Low 10mg/100mL (1.11mM)
      ii. High 20mg/100mL(2.22mM)
   b. Abs/E.C.= Abs/6.22

c. 1.05/0.05= cuvette d
d.
e. dilution (0.05mL blood in 1mL reagent cocktail)
d.
e.
f. Standard concentration = Abs/6.22 x cuvette dilution= Abs x 3.38
e. 3/1= blood dilution (0.5mL blood in 1.5mL of 10%PCA)
f. Sample concentration= (Abs/6.22) x 1.05/0.05 x 3/1= Abs x 10.13mM

Blood Lactate Concentration [La]=abs×10.13mM

Electromyography

Bagnoli™ Desktop EMG system (Delsys INC., Natick, MA) will be used to determine muscle activity of the: gluteus maximus, rectus femoris, vastus lateralis, vastus medialis, biceps femoris, gastrocnemius, soleus, and tibialis anterior. Locations were determined based on recommendations of the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) ((SENIAM). 2006). Skin sites were cleaned and shaved with an alcoholic prep wipe and disposable razor. Electrodes were then placed along the skin surface to run in parallel with the skeletal muscle fibers, and secured with double sided tape and athletic wrap. Raw EMG signals were smoothed using a fourth-order, band-pass Butterworth filter with a frequency range set between 20 and 500Hz. Onset and offset of EMG activity were determined when the signal with an amplitude above two standard deviations beyond the mean of the quiescent phase between EMG bursts (Diefenthaeler et al. 2012). All EMG data were visually inspected and data manually selected to determine periods of quiescence before and after EMG bursts. EMG data were normalized as the percentage of the highest value which occurred during testing.

Joint Power Calculations

Pedal Force

Normal and tangential components of force applied to the pedal were collected using a custom-designed force pedal with two piezo electric force transducers (Kistler, model 9251AQ01) at a sampling rate of 2000Hz. Pedal force was filtered by a third-order low-pass Butterworth filter

with a cutoff frequency of 10Hz (Coyle et al. 1991, Diefenthaeler et al. 2012). Data were paired to match the kinematic data obtained through motion analysis. Using the pedal angle obtained in the coordinate data, forces were transformed from the reference frame of the pedal into the inertial reference frame. Data were used for inverse dynamics calculations that will determine the joint moments/power from the hip, knee, and ankle (Hull et al. 1985, Skovereng et al. 2016, Skovereng et al. 2016). Data was processed using a custom MatLab (MathWorks Inc., Natick, MA, USA) program.

Motion Analysis

During this procedure, subjects will be exercising on a stationary cycle ergometer while wearing 3M reflective markers. The markers were secured to the subjects using double-sided tape and placed on 9 sites (acromion process, mid-axillary, greater trochanter, mid-femur, lateral epicondyle of knee, mid-shank, lateral malleolus, toe of shoe, and heel of shoe). The Vicon Nexus motion analysis system uses infrared technology to collect data on the position of the markers in a 3-dimensional space and provides coordinates that were used for data processing and analysis (Vicon Motion Systems Ltd., Lake Forest, CA). Data were used for inverse dynamics calculations that will determine the joint moments/power from the hip, knee, and ankle (Hull et al. 1985, Neptune et al. 1995, Neptune et al. 1996, Skovereng et al. 2016, Skovereng et al. 2016). All data were processed using a custom MatLab (MathWorks Inc., Natick, MA, USA) program.

Joint powers at the hip, knee, and ankle were derived using standard inverse dynamics techniques (Hull et al. 1985). Rigid segment models of the crank, foot, leg, and thigh were generated. Hip position was determined based on the location of the anterior superior iliac spine (Neptune et al. 1995). Linear and angular velocities and accelerations of the limb segments were determined by finite differentiation of position data with respect to time. Segmental mass

proportions, center of mass locations, and radii of gyration will be estimated from anthropometric tables (de Leva 1996). Joint moments of the ankle, knee, and hip were calculated through use of angular accelerations of the segments, normal and tangential pedal forces, and acceleration of segmental center of gravity. Joint powers were then calculated by the product of joint angular velocities and joint moments.

Joint reaction forces:

Sum of Horizontal Forces, $SF_x = m.a_x$:
$R_{xp} = m.a_x - R_{xd}$ ...

(where p = proximal, d = distal joint, a_y = acceleration of segment center of mass, CoM, in y direction; Note that d = force pedal when p = ankle)

from Newton, Sum of Vertical Forces, $SF_y = m.a_y$:
$R_{yp} = m.a_y + mg - R_{yd}$ ...

Joint moment about segment CoM:
Using motion co-ordinates:

$$M_{zp} = I_z a - M_{zd} - R_{xp}.(y_p - y_{CoM}) + R_{yp}.(x_{CoM} - x_p) + R_{xd}.(y_{CoM} - y_d) - R_{yd}.(x_d - x_{CoM}) \ldots$$

where (x_{CoM}, y_{CoM}) are the co-ordinates of the center of mass of the segment, (x_p, y_p) the coordinates of the proximal joint (x_d, y_d) the coordinates of the distal joint.

APPENDIX B.

Study 1: Individual Data Tables

Study 1 Individual Data: Low (LLT) and High (HLT) Lactate Threshold Cyclists Maximal Oxygen Consumption Results While Cycling

LLT	VO_{2max} (mL/kg/min)	VO_{2max} (L/min)	Respiratory Exchange Ratio	Heart Rate (bpm)	Work Rate (Watts)	Rating of Perceived Exertion
1	72.4	4.87	1.10	194	410	20
2	60.0	4.00	1.08	171	335	18
3	62.1	4.90	1.11	170	355	18
4	68.1	4.50	1.11	181	325	17
5	66.1	3.92	1.10	180	290	17
6	62.1	4.21	1.09	188	295	18
7	63.6	4.60	1.10	171	275	18
8	64.9	4.42	1.11	180	325	18
Mean	*64.9*	*4.43*	*1.10*	*179*	*326*	*18*
HLT						
1	68.9	4.90	1.10	186	390	18
2	61.3	5.30	1.11	185	313	18
3	64.5	3.91	1.10	196	425	19
4	65.4	4.61	1.10	190	375	18
5	54.4	4.51	1.11	194	375	19
6	52.9	4.46	1.14	190	360	18
7	58.8	4.10	1.10	180	280	19
8	56.4	4.90	1.09	198	350	18
Mean	*60.3*	*4.58*	*1.11*	*190*	*356*	*18*

Study 1 Individual Data: Low (LLT) and High (HLT) Lactate Threshold Cyclists Maximal Oxygen Consumption Results While Treadmill Running

LLT	VO_{2max} (mL/kg/min)	VO_{2max} (L/min)	Respiratory Exchange Ratio	Heart Rate (bpm)	Rating of Perceived Exertion
1	76.7	5.07	1.09	188	19
2	61.4	4.09	1.09	168	18
3	59.7	4.76	1.13	175	17
4	69.9	4.69	1.10	180	17
5	64.5	3.89	1.02	181	17
6	64.6	4.40	1.09	189	17
7	64.6	4.60	1.00	171	18
8	65.7	4.50	1.09	180	18
Mean	*65.9*	*4.49*	*1.07*	*179*	*18*
HLT					
1	64.1	4.62	1.11	183	18
2	60.2	5.05	1.11	177	18
3	60.3	3.93	1.10	183	17
4	60.0	4.19	1.10	173	19
5	55.0	4.59	1.11	184	18
6	52.9	4.46	1.15	192	19
7	60.3	4.16	1.02	182	18
8	56.4	4.78	0.98	197	19
Mean	*58.7*	*4.47*	*1.09*	*184*	*18*

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' oxygen consumption (L/min) during cycling lactate threshold testing

		LLT Oxygen Consumption (L/min)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*1.77*	2.16	1.69	2.00	1.70	1.46	1.67	1.81	1.70
50	*2.10*	2.54	2.02	2.01	2.21	1.57	2.13	2.20	2.13
60	*2.62*	3.14	2.39	2.57	2.60	2.60	2.46	2.60	2.60
70	*3.08*	3.55	2.71	3.06	3.22	3.01	2.94	3.10	3.06
80	*3.55*	4.04	3.23	3.75	3.60	3.10	3.48	3.60	3.54
90	*4.03*	4.64	3.63	4.45	4.11	3.50	3.93	4.00	4.04
		HLT Oxygen Consumption (L/min)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*1.80*	2.05	1.80	1.62	1.18	1.93	1.71	1.60	2.49
50	*2.22*	2.42	2.22	1.93	1.90	2.31	2.15	1.99	2.86
60	*2.76*	2.96	2.76	2.20	3.19	2.77	2.82	2.22	3.19
70	*3.21*	3.42	3.21	2.70	3.68	3.23	3.35	2.70	3.42
80	*3.66*	3.85	3.66	3.12	3.80	3.74	3.84	3.20	4.10
90	*4.23*	4.39	4.23	3.46	4.90	4.15	4.34	3.90	4.50

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' heart rate (bpm) during cycling lactate threshold testing

		LLT Heart Rate (bpm)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*114*	126	87	123	109	137	110	101	113
50	*121*	136	102	142	118	148	120	110	125
60	*137*	152	119	155	133	158	129	117	138
70	*152*	164	132	164	155	172	153	126	152
80	*164*	176	146	175	169	185	167	135	165
90	*174*	186	156	190	179	191	175	145	175
		HLT Heart Rate (bpm)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*114*	109	111	114	105	118	118	100	138
50	*124*	122	122	131	118	128	128	104	139
60	*138*	137	137	141	130	141	141	130	150
70	*151*	152	147	166	139	154	154	139	160
80	*165*	161	161	178	150	168	168	164	174
90	*176*	176	171	195	163	176	176	179	179

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' respiratory exchange ratio during cycling lactate threshold testing

		LLT Respiratory Exchange Ratio							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*0.89*	0.85	0.82	0.97	0.88	0.91	0.94	0.92	0.90
50	*0.92*	0.90	0.87	0.99	0.90	0.93	0.96	0.92	0.92
60	*0.94*	0.92	0.89	1.01	0.90	0.95	0.98	0.96	0.94
70	*0.95*	0.96	0.92	1.04	0.93	0.93	0.98	0.99	0.96
80	*1.00*	0.98	0.94	1.10	0.98	1.00	1.01	1.00	1.00
90	*1.02*	1.03	0.95	1.02	1.00	1.02	1.10	1.02	1.02
		HLT Respiratory Exchange Ratio							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*0.86*	0.92	0.88	0.88	0.76	0.87	0.87	0.85	0.85
50	*0.90*	0.91	0.91	0.91	0.96	0.90	0.90	0.88	0.86
60	*0.92*	0.90	0.91	0.92	1.06	0.93	0.93	0.89	0.85
70	*0.95*	0.93	0.92	0.94	1.07	0.97	0.97	0.92	0.87
80	*0.98*	0.94	0.93	0.98	1.12	1.00	1.00	0.97	0.93
90	*0.99*	0.98	0.96	1.02	0.99	1.04	1.04	1.02	0.92

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' work rate (Watts) during cycling lactate threshold testing

		LLT Work Rate (Watts)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*117*	135	100	135	120	100	110	115	120
50	*153*	175	135	175	155	130	145	150	155
60	*186*	215	170	215	190	160	175	180	185
70	*221*	255	200	250	230	190	210	215	220
80	*256*	295	230	290	265	220	245	250	255
90	*291*	335	260	330	300	250	275	285	290
		HLT Work Rate (Watts)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*120*	130	145	100	110	120	120	110	120
50	*155*	170	190	135	145	155	155	140	150
60	*191*	210	230	165	175	195	195	170	190
70	*228*	250	275	200	210	230	230	210	220
80	*265*	290	320	230	245	265	265	250	260
90	*302*	330	360	265	280	300	300	290	290

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' rating of perceived exertion (RPE) during cycling lactate threshold testing

		LLT Rating of Perceived Exertion							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*9*	9	9	8	9	11	7	10	9
50	*11*	11	10	10	11	13	11	12	11
60	*13*	13	12	12	13	16	13	14	13
70	*15*	15	14	13	15	17	15	15	15
80	*17*	17	16	17	17	18	17	16	17
90	*18*	18	18	18	18	19	19	18	18
		HLT Rating of Perceived Exertion							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*8*	10	7	9	9	7	7	7	9
50	*10*	12	11	12	10	9	9	9	11
60	*12*	13	13	14	11	11	11	9	12
70	*14*	14	14	16	13	13	13	12	14
80	*16*	17	16	18	14	16	16	13	16
90	*18*	19	18	19	16	18	18	17	17

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' blood lactate concentration (mmoL) during cycling lactate threshold testing

		LLT Blood Lactate Concentration (mmoL)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*1.07*	1.0	0.4	1.1	0.9	1.3	0.7	2.1	1.1
50	*1.28*	1.0	0.4	1.1	1.1	1.7	0.8	2.9	1.3
60	*2.06*	1.2	0.5	2.1	1.1	1.4	2.3	5.8	2.1
70	*2.50*	2.1	0.7	2.7	1.5	2.8	2.5	5.5	2.5
80	*4.40*	3.8	1.5	4.6	2.4	3.8	5.1	9.6	4.4
90	*6.30*	6.6	3.0	7.1	2.9	7.5	5.6	10.5	6.2
		HLT Blood Lactate Concentration (mmoL)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*0.78*	0.5	0.6	1.1	0.7	0.5	0.5	1.2	1.2
50	*0.81*	0.6	0.7	1.3	0.6	0.7	0.7	1.1	0.8
60	*1.01*	0.9	0.7	0.8	0.6	1.6	1.6	0.9	1.0
70	*1.35*	1.5	0.8	0.8	0.7	2.2	2.2	1.7	0.9
80	*2.10*	2.4	1.5	1.1	1.4	3.3	3.3	2.1	1.7
90	*4.31*	4.1	3.2	2.1	3.5	6.6	6.6	4.5	3.9

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' oxygen consumption (L/min) during uphill running lactate threshold testing

		LLT Oxygen Consumption (L/min)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*1.64*	2.02	1.40	1.53	1.58	1.45	1.80	1.64	1.63
50	*2.01*	2.80	1.80	2.03	1.83	1.57	2.10	1.90	2.00
60	*2.80*	3.33	2.70	2.43	2.73	2.50	2.60	2.96	2.75
70	*3.41*	3.94	3.50	3.75	3.40	3.01	3.10	3.17	3.41
80	*3.79*	4.61	3.70	4.15	3.65	3.09	3.70	3.68	3.80
90	*4.12*	4.90	4.10	4.36	3.96	3.50	3.90	4.20	4.13
		HLT Oxygen Consumption (L/min)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*1.63*	1.42	2.00	1.20	1.90	1.64	1.86	1.29	1.70
50	*1.98*	1.65	2.58	1.60	2.50	2.18	1.82	1.42	2.10
60	*2.86*	2.39	4.01	2.70	3.20	3.32	2.23	1.99	3.00
70	*3.43*	3.12	4.61	3.20	3.50	3.61	3.24	2.53	3.60
80	*3.95*	3.79	5.34	3.95	3.80	3.76	3.65	2.99	4.30
90	*4.48*	4.50	5.55	4.66	4.20	4.11	4.21	3.90	4.70

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' heart rate (bpm) during uphill running lactate threshold testing

		LLT Heart Rate (bpm)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*107*	138	82	112	93	137	94	102	108
50	*119*	146	102	135	109	141	102	106	120
60	*142*	164	130	145	136	165	132	127	143
70	*158*	176	150	163	149	176	148	148	159
80	*170*	187	162	168	161	182	170	160	170
90	*177*	193	171	173	169	190	180	173	178
		HLT Heart Rate (bpm)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*103*	88	110	106	92	117	110	97	100
50	*114*	98	121	123	112	126	113	100	115
60	*142*	123	151	176	154	157	130	123	121
70	*161*	145	160	193	169	166	153	147	156
80	*173*	163	176	193	172	176	165	167	170
90	*184*	177	180	193	195	180	177	180	189

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' respiratory exchange ratio (RER) during uphill running lactate threshold testing

		LLT Respiratory Exchange Ratio							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*0.85*	0.90	0.81	0.86	0.85	0.88	0.86	0.85	0.86
50	*0.91*	0.95	0.86	0.95	0.90	0.90	0.91	0.90	0.91
60	*0.94*	0.96	0.87	0.98	0.92	0.98	0.94	0.96	0.94
70	*0.96*	0.98	0.91	1.02	0.93	0.93	0.95	0.97	0.95
80	*0.97*	1.03	0.93	0.99	0.94	0.99	0.98	0.98	0.98
90	*1.03*	1.10	1.01	1.02	0.98	1.04	1.02	1.02	1.02
		HLT Respiratory Exchange Ratio							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*0.82*	0.88	0.93	0.85	0.82	0.75	0.83	0.75	0.78
50	*0.88*	0.88	0.87	0.93	0.88	0.89	0.91	0.80	0.85
60	*0.93*	0.91	0.95	0.97	0.93	0.94	0.92	0.88	0.91
70	*0.93*	0.92	0.94	0.99	0.93	0.96	0.93	0.88	0.89
80	*0.97*	0.95	0.98	0.98	0.97	1.02	0.97	0.90	0.96
90	*0.99*	1.01	0.99	1.02	0.99	0.99	0.97	0.99	0.98

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' rating of perceived exertion (RPE) during uphill running lactate threshold testing

		LLT Rating of Perceived Exertion							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*7*	6	7	7	7	10	7	7	7
50	*9*	9	9	9	9	13	7	9	9
60	*12*	12	11	12	11	15	11	12	12
70	*13*	14	13	14	12	16	12	13	13
80	*15*	16	15	15	13	17	13	15	15
90	*17*	19	17	17	14	18	17	17	17
		HLT Rating of Perceived Exertion							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*8*	7	10	8	9	7	11	8	9
50	*10*	8	12	10	11	12	13	10	11
60	*11*	11	14	15	13	13	13	11	13
70	*13*	12	15	18	15	15	15	13	14
80	*15*	14	17	18	17	18	16	15	18
90	*17*	17	18	18	18	18	17	17	19

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' blood lactate concentration (mmoL) during uphill running lactate threshold testing

		LLT Blood Lactate Concentration (mmoL)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*0.6*	0.4	0.3	0.6	0.6	0.8	0.1	1.5	0.6
50	*0.7*	0.5	0.4	0.7	0.5	0.7	0.2	2.3	0.8
60	*1.1*	0.7	0.5	1.2	0.8	0.9	0.6	3.2	1.1
70	*1.8*	1.5	0.8	3.6	1.0	2.0	0.6	3.4	1.8
80	*2.5*	2.9	1.5	4.4	1.3	2.4	1.2	4.3	2.6
90	*4.1*	4.9	2.6	5.5	1.9	4.7	2.1	7.5	4.2
		HLT Blood Lactate Concentration (mmoL)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
40	*0.6*	0.9	0.5	0.6	0.4	0.2	0.1	1.1	0.6
50	*0.7*	0.9	0.5	0.8	1.2	0.5	0.1	1.2	0.6
60	*1.2*	1.2	0.9	0.7	2.7	1.3	0.4	1.3	1.2
70	*1.9*	1.2	1.4	1.4	4.4	2.3	0.8	1.3	2.6
80	*2.8*	1.7	2.9	2.1	4.6	3.6	1.2	1.4	4.8
90	*3.8*	3.5	4.5	2.3	5.5	3.6	2.3	2.7	6.2

Study 1 Individual Data: Low (LLT) and high (HLT) total hemoglobin concentration (μM) during submaximal biomechanical testing

		LLT Total Hemoglobin Concentration (μM)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Rest	*100.1*	118.5	112.4	36.2	88.9	117.5	132.6	82.6	98.4
60	*104.1*	118.5	120.4	40.1	91.4	117.5	144.8	82.6	102.2
70	*111.8*	116.2	113.5	42.7	97.6	162.4	152.3	96.8	111.6
80	*109.4*	121.3	97.7	43.6	100.7	167.2	144.9	98.9	110.6
90	*106.4*	125.4	97.7	36.7	103.9	168.3	114.5	101	106.8
		HLT Total Hemoglobin Concentration (μM)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Rest	*65.3*	66.9	80.1	98.7	40.0	59.1	47.2	76.72	53.4
60	*69.4*	66.9	78	102.5	40.1	66.8	47.2	94.9	58.4
70	*75.5*	69	85.4	104.9	39.8	76.2	55.6	110.5	62.4
80	*78.1*	77.9	84.5	106.9	43.6	78.0	60.5	109.1	64.3
90	*81.1*	76.5	89	107.6	46.8	82.3	63.2	119	64.7

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' oxygenated hemoglobin concentration (μM) during submaximal biomechanical testing

		LLT Oxygenated Hemoglobin Concentration (μM)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Rest	*72.6*	86.3	87.4	20.4	59.8	85.6	108.1	47.8	70.8
60	*71.5*	86.3	92.7	22.4	61.0	85.6	118.4	47.8	73.5
70	*79.8*	81.8	80.2	25.8	65.5	132.0	126.7	64.4	82.3
80	*78.1*	87.3	61.6	25.6	66.2	137.1	117.6	64.9	80.0
90	*72.7*	90.9	61.6	18.8	69.5	139.3	71.4	65.9	73.9
		HLT Oxygenated Hemoglobin Concentration (μM)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Rest	*41.3*	38.6	54.1	66.4	18.9	38.1	23.6	60.0	30.8
60	*43.5*	38.6	53.3	62.5	18.9	43.6	23.6	72.5	34.7
70	*48.6*	38.4	56.6	64.7	19.2	52.6	33.0	87.7	37.2
80	*50.5*	42.8	56.2	67.0	22.2	54.0	36.6	86.3	39.0
90	*53.3*	42.6	58.4	67.6	25.2	58.1	38.6	95.9	39.7

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' deoxygenated hemoglobin concentration (μM) during submaximal biomechanical testing

		LLT Deoxygenated Hemoglobin Concentration (μM)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Rest	*27.0*	32.2	25.0	15.8	29.1	32.0	24.5	34.8	27.6
60	*28.1*	32.2	27.7	17.7	30.4	32.1	26.4	34.8	28.7
70	*28.8*	34.4	33.3	16.9	32.1	30.4	25.6	32.4	29.3
80	*29.9*	34.0	36.2	18.0	34.5	30.1	27.2	33.9	30.6
90	*31.9*	34.5	36.3	17.9	34.4	29.1	43.1	35.1	32.9
		HLT Deoxygenated Hemoglobin Concentration (μM)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Rest	*23.9*	28.3	26.0	32.3	21.1	20.9	23.5	16.7	22.6
60	*25.9*	28.3	24.8	39.9	21.1	23.2	23.5	22.4	23.7
70	*26.8*	30.5	28.7	40.2	20.6	23.5	22.6	22.8	25.2
80	*27.6*	35.1	28.2	39.9	21.4	24.0	23.9	22.8	25.3
90	*27.9*	33.9	30.7	40.0	21.6	24.2	24.6	23.1	25.0

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' percent saturation during submaximal biomechanical testing

					LLT %Saturation				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Rest	*69.4*	72.8	77.7	56.3	67.2	72.8	81.5	57.9	69.5
60	*69.3*	72.8	76.8	55.8	66.7	72.8	81.7	57.9	69.2
70	*71.2*	70.3	70.6	60.4	67.1	81.2	83.1	66.5	71.3
80	*69.7*	71.8	63.0	58.8	65.7	81.9	81.2	65.7	69.7
90	*65.9*	72.4	63.0	51.2	66.9	82.7	60.3	65.2	66.0
					HLT % Saturation				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Rest	*60.8*	57.7	67.6	67.2	47.3	60.8	50.1	78.2	57.6
60	*60.2*	57.7	69.2	61.0	47.3	60.2	50.1	76.4	59.4
70	*61.5*	55.7	66.3	61.7	48.2	61.5	59.4	79.4	59.6
80	*62.2*	54.9	66.6	62.6	50.9	62.2	60.4	79.1	60.7
90	*63.0*	55.7	65.4	62.8	53.9	63.0	61.1	80.9	61.3

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' vastus lateralis activation (%Activation) during submaximal biomechanical testing

		LLT Vastus Lateralis Activation (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*36.8*	45.4	32.7	30.6	33.6	44.4	33.6	43.1	31.3
70	*35.9*	44.4	35.6	31.7	35.2	33.2	35.2	40.3	32.1
80	*45.7*	52.8	37.6	37.9	36.8	70.7	36.8	59.3	33.8
90	*44.6*	57.0	41.2	31.1	38.5	-----	38.5	76.5	29.5
		HLT Vastus Lateralis Activation (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*37.4*	41.2	26.7	39.9	43.7	43.1	35.6	42.1	27.1
70	*39.5*	42.4	31.1	44.3	42.3	40.9	38.8	46.8	29.7
80	*41.4*	39.8	28.6	48.7	40.1	48.3	41.9	51.6	32.4
90	*47.1*	55.0	36.2	53.2	49.7	43.3	45.1	56.4	37.7

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' vastus medialis activation (%Activation) during submaximal biomechanical testing

		LLT Vastus Medialis Activation (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*50.3*	28.7	24.9	28.8	70.3	75.9	70.3	74.4	29.0
70	*52.0*	31.9	22.8	31.7	62.9	95.3	62.9	81.0	27.5
80	*41.6*	33.6	29.4	33.2	55.5	41.0	55.5	52.9	31.6
90	*40.9*	44.1	35.3	30.3	48.1	39.0	48.1	48.1	34.7
		HLT Vastus Medialis Activation (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*29.6*	29.2	30.8	32.6	26.2	-----	23.2	34.6	30.8
70	*32.5*	32.6	33.5	36.7	26.2	-----	25.3	39.1	33.9
80	*33.3*	28.2	35.1	40.9	20.6	-----	27.4	43.6	37.0
90	*39.8*	36.3	40.6	45.0	30.2	-----	29.5	48.0	41.7

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' rectus femoris activation (%Activation) during submaximal biomechanical testing

		LLT Rectus Femoris Activation (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*24.2*	27.4	22.5	26.6	22.8	24.1	22.8	26.5	21.6
70	*23.0*	28.5	22.4	23.2	21.4	22.2	21.4	25.9	19.2
80	*26.8*	31.1	33.2	38.9	20.0	23.6	20.0	35.6	11.8
90	*32.2*	46.0	34.6	24.9	18.6	-----	18.6	71.5	11.1
		HLT Rectus Femoris Activation (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*29.3*	20.1	17.2	27.7	60.7	37.5	26.7	28.6	15.9
70	*32.7*	22.7	17.8	29.6	66.8	35.3	27.9	30.7	30.7
80	*30.5*	23.3	18.1	31.5	40.3	36.4	29.1	32.8	32.8
90	*35.8*	42.2	27.4	33.5	49.2	34.1	30.3	34.9	34.9

Study 1 Individual Data; Low (LLT) and high (HLT) lactate threshold cyclists' biceps femoris activation (%Activation) during submaximal biomechanical testing

					LLT Biceps Femoris Activation (%)				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*35.2*	26.6	23.2	-----	42.8	39.4	42.8	44.3	27.3
70	*36.7*	30.2	28.1	-----	42.2	39.8	42.2	43.3	30.9
80	*35.9*	34.5	25.0	-----	41.6	36.0	41.6	42.5	30.2
90	*39.2*	45.2	34.9	-----	40.9	33.3	40.9	44.0	35.1
					HLT Biceps Femoris Activation (%)				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*40.9*	27.8	47.4	34.5	46.1	55.8	32.6	34.7	48.5
70	*41.7*	24.7	51.6	34.9	42.7	59.8	34.4	35.1	50.5
80	*41.8*	37.4	51.9	35.2	44.2	41.0	36.3	35.5	52.7
90	*43.9*	48.4	54.0	35.6	47.7	37.2	38.1	35.8	54.5

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' gluteus maximus activation (%Activation) during submaximal biomechanical testing

					LLT Gluteus Maximus Activation (%)				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*49.0*	36.9	47.3	-----	46.9	58.1	-----	56.0	-----
70	*55.9*	42.2	54.2	-----	59.3	61.2	-----	62.8	-----
80	*66.3*	77.7	64.0	-----	50.0	64.3	-----	75.3	-----
90	*69.8*	74.7	69.0	-----	64.8	67.4	-----	73.2	-----
					HLT Gluteus Maximus Activation (%)				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*43.6*	24.0	41.6	23.7	55.5	49.2	55.5	-----	-----
70	*49.2*	39.2	46.9	25.6	53.3	63.4	53.3	-----	-----
80	*53.7*	46.3	50.1	26.9	51.0	75.6	51.0	-----	-----
90	*52.8*	56.0	49.4	24.7	48.7	68.9	48.7	-----	-----

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' tibialis anterior activation (%Activation) during submaximal biomechanical testing

		LLT Tibialis Anterior Activation (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*33.7*	45.3	24.6	68.2	8.9	43.8	8.9	56.7	13.5
70	*23.1*	4.4	21.6	57	11.7	30	11.7	41.8	6.7
80	*34.8*	29.8	17.7	100	14.6	28.1	14.6	67.1	6.3
90	*36.5*	31.9	36.1	62.6	17.4	51.4	17.4	54.3	20.7
		HLT Tibialis Anterior Activation (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*54.6*	68.1	61	40.1	93.6	47.8	25.8	41	59.8
70	*48.4*	46.2	65.3	41.8	51.5	54.1	30.0	42.8	55.1
80	*40.1*	46.2	30.3	43.6	21.7	49.6	34.2	44.7	50.2
90	*48.7*	59.9	57.2	45.3	30.9	48.3	38.4	46.6	62.6

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' gastroc medialis activation (%Activation) during submaximal biomechanical testing

					LLT Gastroc Medialis Activation (%)				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*29.4*	29.2	53.6	33.2	11.7	34.9	11.7	44.9	15.7
70	*26.6*	24.7	42.0	34.0	14.3	29.2	14.3	37.4	17.1
80	*27.6*	28.6	43.0	35.9	16.9	23.1	16.9	37.9	18.5
90	*27.2*	23.8	37.6	33.6	19.5	28.5	19.5	34.6	20.8
					HLT Gastroc Medialis Activation (%)				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*36.7*	28.0	36.0	22.4	55.3	55.2	35.0	23.7	38.3
70	*35.8*	30.6	37.5	25.1	46.3	47.4	36.2	26.6	36.5
80	*36.1*	32.4	41.1	27.8	41.8	43.5	37.5	29.6	35.2
90	*34.2*	39.5	29.2	30.5	38.4	38.2	38.7	32.5	26.8

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' soleus activation (%Activation) during submaximal biomechanical testing

					LLT Soleus Activation (%)				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*40.9*	38.6	51.9	52.8	33.3	34.7	33.3	49.9	33.(
70	*40.4*	36.7	51.9	49.4	35.2	33.1	35.2	48.4	33.4
80	*44.6*	39.3	60.5	58.2	37.2	33.9	37.2	56.1	34.3
90	*42.3*	35.9	49.3	51.1	39.1	39.6	39.1	48.6	37.
					HLT Soleus Activation (5)				
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*52.6*	51.3	90.1	35.3	62.5	56.3	34.6	38.0	----
70	*54.2*	58.6	70.1	40.8	42.8	58.8	39.3	44.0	79.3
80	*51.5*	50.4	57.3	46.4	47.4	58.5	44.0	50.0	57.3
90	*50.5*	60.8	41.5	52.0	50.0	53.2	48.7	56.1	41.3

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' normalized hip joint specific power (W/kg) during submaximal biomechanical testing

		LLT Normalized Hip Joint Specific Power (W/kg)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*1.09*	0.79	1.15	1.64	1.28	1.07	0.89	1.09	0.87
70	*1.15*	0.93	1.34	1.44	1.26	1.15	1.11	1.08	0.86
80	*1.30*	1.31	1.14	1.60	1.59	1.22	1.33	1.12	0.85
90	*1.34*	1.00	1.10	1.76	2.05	1.29	1.55	1.10	0.84
		HLT Normalized Hip Joint Specific Power (W/kg)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*1.16*	1.44	1.05	0.59	1.27	1.07	1.39	1.44	1.03
70	*1.25*	1.47	1.07	1.13	1.28	1.08	1.47	1.44	1.07
80	*1.38*	1.50	1.09	1.66	1.37	1.10	1.56	1.64	1.14
90	*1.46*	1.53	1.10	2.20	1.31	1.12	1.64	1.72	1.04

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' normalized knee joint specific power (W/kg) during submaximal biomechanical testing

		LLT Normalized Knee Joint Specific Power (W/kg)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*1.31*	1.29	1.38	1.64	1.24	1.32	1.39	1.08	1.10
70	*1.38*	1.42	1.42	1.69	1.30	1.48	1.56	1.07	1.07
80	*1.49*	1.58	1.31	1.87	1.57	1.64	1.73	1.15	1.04
90	*1.58*	1.58	1.27	2.05	1.91	1.80	1.90	1.14	1.01
		HLT Normalized Knee Joint Specific Power (W/kg)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*1.42*	1.41	1.21	1.44	1.54	1.23	1.43	1.95	1.13
70	*1.42*	1.37	1.25	1.48	1.60	1.27	1.47	1.71	1.21
80	*1.45*	1.33	1.28	1.36	1.63	1.31	1.52	1.91	1.24
90	*1.43*	1.30	1.32	1.32	1.38	1.35	1.56	1.90	1.27

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' normalized ankle joint specific power (W/kg) during submaximal biomechanical testing

		LLT Normalized Ankle Joint Specific Power (W/kg)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*0.27*	0.30	0.26	0.18	0.06	0.46	0.36	0.37	0.20
70	*0.26*	0.15	0.44	0.12	0.05	0.25	0.38	0.40	0.30
80	*0.27*	0.13	0.42	0.21	0.17	0.05	0.39	0.42	0.40
90	*0.35*	0.33	0.47	0.30	0.09	0.07	0.41	0.61	0.50
		HLT Normalized Ankle Joint Specific Power (W/kg)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*0.16*	0.19	0.08	0.27	0.16	0.09	0.31	0.08	0.07
70	*0.17*	0.08	0.10	0.46	0.14	0.10	0.31	0.08	0.06
80	*0.18*	0.08	0.11	0.44	0.16	0.11	0.31	0.12	0.09
90	*0.19*	0.15	0.12	0.49	0.17	0.13	0.31	0.08	0.07

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' relative hip contribution (% contribution) during submaximal biomechanical testing

		LLT Relative Hip Contribution (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*40.7*	33.1	41.1	47.4	49.6	37.7	33.8	42.8	40.0
70	*41.1*	37.2	41.8	44.4	48.4	39.8	36.5	42.4	38.5
80	*41.6*	43.2	39.7	43.5	47.7	41.9	38.5	41.6	37.0
90	*40.1*	34.6	38.8	42.8	50.6	39.7	40.1	38.5	35.7
		HLT Relative Hip Contribution (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*42.2*	47.5	44.7	25.6	42.9	44.9	44.4	41.6	46.2
70	*44.2*	50.4	44.1	36.7	42.4	44.4	45.3	44.7	45.7
80	*45.9*	51.5	43.6	48.0	43.5	43.8	46.0	44.7	46.2
90	*46.9*	51.4	43.1	54.9	45.7	43.4	46.7	46.4	43.7

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' relative knee contribution (% contribution) during submaximal biomechanical testing

		LLT Relative Knee Contribution (%)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*48.9*	54.2	49.5	47.4	48.0	46.3	52.6	42.7	50.8
70	*49.4*	56.7	44.4	51.9	49.7	51.4	51.2	42.0	48.0
80	*48.8*	52.4	45.5	50.7	47.3	56.4	50.1	42.8	45.4
90	*47.9*	54.2	44.7	49.8	47.2	55.5	49.3	40.0	43.0
		HLT Relative Knee Contribution (%)							
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*52.0*	46.3	51.6	62.5	51.8	51.6	45.7	56.0	50.8
70	*50.2*	46.8	51.8	48.3	52.9	51.7	45.3	53.0	51.7
80	*48.4*	45.7	51.9	39.2	51.5	51.8	44.8	52.2	50.1
90	*47.2*	43.5	52.0	33.0	48.2	51.9	44.4	51.3	53.5

Study 1 Individual Data: Low (LLT) and high (HLT) lactate threshold cyclists' relative ankle contribution (% contribution) during submaximal biomechanical testing

		LLT Relative Ankle Contribution (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*10.9*	12.7	9.4	5.3	2.4	16.0	13.7	14.5	9.2
70	*9.5*	6.1	13.8	3.8	1.9	8.7	12.4	15.6	13.5
80	*9.5*	4.4	14.8	5.8	5.0	1.7	11.4	15.7	17.5
90	*11.6*	11.2	16.4	7.3	2.2	2.2	10.6	21.5	21.3
		HLT Relative Ankle Contribution (%)							
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
60	*5.7*	6.2	3.7	11.9	5.3	3.5	9.8	2.4	2.9
70	*5.6*	2.8	4.1	15.0	4.7	3.9	9.5	2.3	2.5
80	*5.7*	2.7	4.5	12.7	5.0	4.3	9.2	3.2	3.7
90	*5.9*	5.1	4.9	12.1	6.0	4.7	8.9	2.3	2.8

APPENDIX C.

Study 2: Individual Data Tables

Study 2 Individual Data: Control and training group baseline treadmill maximal oxygen consumption testing results

Control	VO_2(L/min)	Respiratory Exchange Ratio	Heart Rate (bpm)	Rating of Perceived Exertion
1	4.29	1.04	194	17
2	3.45	1.00	202	18
3	4.08	1.10	192	17
4	4.26	1.08	181	17
5	3.41	1.09	188	19
6	2.96	1.04	189	16
7	2.17	1.15	199	19
8	2.75	0.98	183	20
9	5.30	1.10	182	18
10	2.49	1.12	204	18
Mean	*3.52*	*1.07*	*191*	*18*
Training				
1	1.95	1.00	181	16
2	3.69	1.07	190	17
3	3.02	1.12	195	19
4	1.76	1.13	182	17
5	4.07	1.11	188	18
6	3.25	1.18	190	17
7	4.46	1.13	210	18
8	4.66	1.20	179	19
9	3.12	1.07	198	17
10	4.44	1.10	167	16
Mean	*3.44*	*1.11*	*188*	*17*

Study 2 Individual Data: Control and training group baseline cycling peak oxygen consumption testing results

Control	VO_2(L/min)	Respiratory Exchange Ratio	Work rate (Watts)	Heart Rate (bpm)	Rating of Perceived Exertion
1	3.55	1.10	260	180	16
2	2.38	0.91	155	174	17
3	4.03	1.04	280	182	17
4	3.69	1.10	235	180	17
5	3.28	1.08	245	193	18
6	2.48	1.08	190	178	17
7	2.04	1.12	145	188	18
8	1.98	1.17	180	181	20
9	4.21	1.10	315	180	16
10	2.11	1.10	160	182	19
Mean	*2.98*	*1.08*	*217*	*189*	*18*
Training					
1	1.82	1.04	130	180	17
2	3.21	1.07	240	184	18
3	2.54	1.00	180	175	17
4	3.47	1.09	250	172	18
5	1.84	1.09	150	179	18
6	3.13	1.11	240	172	20
7	4.32	1.13	285	190	20
8	4.12	1.17	265	175	17
9	3.15	1.01	225	198	18
10	3.97	1.05	310	160	19
Mean	*3.16*	*1.08*	*228*	*179*	*18*

Study 2 Individual Data: Control and training group post-intervention/training cycling peak oxygen consumption testing results

Control	VO_2(L/min)	Respiratory Exchange Ratio	Work Rate (Watts)	Heart Rate (bpm)	Rating of Perceived Exertion
1	3.52	1.06	260	179	17
2	2.36	0.95	175	179	18
3	3.91	0.99	280	183	20
4	3.69	1.10	235	180	17
5	3.28	1.09	245	192	18
6	2.40	1.08	190	183	17
7	2.10	1.06	150	182	17
8	2.07	1.14	185	184	20
9	4.28	1.07	355	182	19
10	2.15	1.12	160	183	19
Mean	*2.98*	*1.07*	*224*	*183*	*18*
Training					
1	1.86	1.08	130	178	17
2	3.41	1.03	270	183	18
3	2.92	1.06	200	183	18
4	3.99	1.08	280	186	19
5	1.90	1.10	160	179	16
6	3.40	1.06	260	176	20
7	4.26	1.04	315	188	19
8	4.13	1.15	300	176	19
9	3.39	1.00	225	198	19
10	3.96	1.04	275	161	19
Mean	*3.32*	*1.06*	*241*	*181*	*18*

Study 2 Individual Data: Baseline control and training group oxygen consumption (L/min) during cycling lactate threshold testing

		Control Group Oxygen Consumption (L/min)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*1.52*	2.12	1.57	1.92	1.79	1.16	1.17	0.93	0.97	2.58	1.01
50	*1.80*	2.41	1.78	2.28	2.32	1.38	1.33	0.99	1.25	3.05	1.21
60	*2.07*	2.76	2.09	2.65	2.64	1.66	1.56	1.08	1.48	3.41	1.40
70	*2.40*	3.10	2.25	3.24	3.32	1.96	1.78	1.25	1.78	3.88	1.48
80	*2.64*	3.42	2.51	3.50	3.75	2.11	1.98	1.17	1.91	4.30	1.73

		Training Group Oxygen Consumption (L/min)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*1.43*	1.15	1.45	1.14	1.62	0.76	1.38	1.83	2.08	1.31	1.59
50	*1.70*	1.27	1.74	1.40	1.87	0.92	1.71	2.09	2.57	1.49	1.91
60	*1.95*	1.33	2.08	1.63	2.18	1.02	2.00	2.37	2.95	1.67	2.22
70	*2.31*	1.50	2.38	1.90	2.62	1.07	2.29	2.80	3.52	2.24	2.74
80	*2.69*	1.64	2.78	2.26	3.37	1.08	2.70	3.24	3.95	2.59	3.33

Study 2 Individual Data: Post-intervention/training control and training group oxygen consumption (L/min) during cycling lactate threshold testing

		Control Group Oxygen Consumption (L/min)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*1.51*	2.12	1.55	1.85	1.79	1.16	1.15	1.01	0.99	2.43	1.02
50	*1.76*	2.41	1.77	2.13	2.32	1.38	1.37	1.07	1.19	2.79	1.19
60	*2.03*	2.76	2.05	2.50	2.64	1.66	1.56	1.16	1.42	3.19	1.37
70	*2.35*	3.10	2.30	2.90	3.32	1.96	1.82	1.21	1.62	3.75	1.54
80	*2.62*	3.42	2.55	3.35	3.75	2.11	2.02	1.32	1.84	3.95	1.88

		Training Group Oxygen Consumption (L/min)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*1.41*	1.01	1.15	1.15	1.45	0.83	1.45	1.67	2.25	1.34	1.81
50	*1.66*	1.11	1.27	1.37	1.78	1.02	1.75	1.94	2.62	1.54	2.15
60	*1.87*	1.23	1.33	1.62	2.09	1.05	1.96	2.27	3.07	1.76	2.33
70	*2.18*	1.37	1.5	1.83	2.55	1.22	2.29	2.65	3.65	1.94	2.84
80	*2.53*	1.6	1.64	2.18	3.26	1.37	2.59	3.11	4.06	2.32	3.19

Study 2 Individual Data: Baseline control and training group respiratory exchange ratio during cycling lactate threshold testing

		Control Respiratory Exchange Ratio									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*0.93*	0.97	0.84	0.93	0.99	0.85	0.89	0.95	0.90	1.03	0.94
50	*0.95*	0.98	0.89	0.97	0.97	0.90	0.92	0.97	0.94	1.00	0.96
60	*0.97*	1.02	0.89	0.97	1.03	0.93	0.96	0.97	0.94	1.03	0.97
70	*0.98*	1.06	0.91	0.96	1.03	0.90	0.97	0.95	0.98	1.07	1.00
80	*1.01*	1.08	0.94	1.03	1.05	0.94	1.01	0.96	0.99	1.07	1.02

		Training Respiratory Exchange Ratio									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*0.96*	0.92	0.95	0.86	0.94	1.01	1.05	0.98	1.02	0.92	0.94
50	*0.97*	0.95	0.96	0.89	0.96	1.02	1.01	1.03	1.00	0.91	0.95
60	*0.98*	0.96	0.97	0.91	0.98	1.03	0.98	1.06	1.06	0.94	0.95
70	*1.00*	0.99	0.98	0.93	1.01	1.04	0.99	1.03	1.12	0.91	1.00
80	*1.04*	1.01	1.01	1.01	1.03	1.04	1.03	1.10	1.14	0.97	1.02

Study 2 Individual Data: Post-intervention/training control and training group respiratory exchange ratio during cycling lactate threshold testing

		Control Group Respiratory Exchange Ratio									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*0.93*	0.97	0.89	0.92	0.99	0.85	0.89	0.94	0.90	0.97	1.01
50	*0.96*	0.98	0.94	0.96	0.97	0.90	0.93	0.95	0.93	0.95	1.06
60	*0.98*	1.02	0.94	0.97	1.03	0.93	0.97	0.95	0.96	1.00	1.05
70	*0.99*	1.06	0.95	0.98	1.03	0.90	0.98	0.95	0.97	1.02	1.05
80	*1.01*	1.08	0.98	1.00	1.05	0.94	1.01	0.95	1.01	1.05	1.02

		Training Group Respiratory Exchange Ratio									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*0.94*	0.91	0.95	0.91	0.98	0.83	0.96	0.95	1.03	0.97	0.92
50	*0.97*	0.94	0.96	0.94	1.00	0.88	0.98	1.00	1.05	0.98	0.95
60	*0.99*	1.00	0.97	0.96	1.03	0.95	0.98	1.04	1.07	1.00	0.95
70	*1.00*	1.04	0.98	0.98	1.05	0.95	0.94	1.01	1.07	1.03	0.98
80	*1.04*	1.05	1.01	1.05	1.08	1.01	0.99	1.04	1.09	1.01	1.03

Study 2 Individual Data: Baseline control and training group heart rate (bpm) during cycling lactate threshold testing

		Control Group Heart Rate (bpm)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*123*	127	139	127	118	100	114	128	118	144	117
50	*137*	152	149	134	134	120	122	131	136	158	138
60	*150*	166	159	142	148	140	142	140	150	171	145
70	*163*	181	173	152	162	158	153	151	165	181	157
80	*175*	200	183	166	177	164	167	155	170	195	169

		Training Group Heart Rate (bpm)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*116*	136	107	107	105	100	108	114	131	146	105
50	*132*	151	136	113	121	113	136	131	144	154	119
60	*145*	157	152	123	145	130	146	150	159	161	125
70	*158*	168	164	142	158	139	160	165	171	174	137
80	*171*	176	179	160	177	149	170	182	184	183	150

Study 2 Individual Data: Post-intervention/training control and training group heart rate (bpm) during cycling lactate threshold testing

		Control Group Heart Rate (bpm)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*119*	128	138	118	118	100	106	123	107	132	120
50	*136*	148	160	130	134	120	129	131	123	149	133
60	*149*	164	168	146	148	140	145	138	139	162	144
70	*163*	179	179	159	162	158	164	143	156	175	157
80	*175*	197	194	173	177	164	184	148	166	180	168

		Training Group Heart Rate (bpm)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*113*	117	117	108	102	93	102	107	130	135	118
50	*127*	125	127	119	121	108	125	128	148	148	125
60	*141*	137	150	134	139	120	139	151	160	155	129
70	*154*	155	158	145	157	132	151	161	170	167	141
80	*168*	168	174	166	177	145	162	177	184	179	151

Study 2 Individual Data: Baseline control and training group rating of perceived exertion during cycling lactate threshold testing

		Control Group Rating of Perceived Exertion									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*10*	9	9	11	11	13	9	11	11	9	9
50	*12*	11	11	13	13	15	12	11	13	10	11
60	*14*	12	14	14	15	17	15	11	15	11	12
70	*16*	15	17	15	18	18	17	13	17	15	14
80	*18*	17	17	17	20	20	20	14	19	16	17

		Training Group Rating of Perceived Exertion									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*9*	11	8	7	13	12	12	9	8	7	7
50	*11*	11	11	9	15	12	13	12	11	11	9
60	*13*	12	13	11	15	13	14	14	14	11	11
70	*15*	13	15	13	16	13	16	16	19	12	12
80	*16*	15	18	15	17	14	17	17	19	13	14

Study 2 Individual Data: Post-intervention/training control and training group rating of perceived exertion during cycling lactate threshold testing

		Control Group Rating of Perceived Exertion									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*10*	7	10	11	11	13	9	10	9	9	8
50	*12*	10	12	12	13	15	11	10	11	11	12
60	*14*	12	14	13	15	17	13	11	13	13	14
70	*16*	15	17	15	18	18	15	12	15	15	16
80	*18*	18	19	17	20	20	17	13	17	19	18

		Training Group Rating of Perceived Exertion									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*10*	11	7	8	13	12	12	11	8	7	7
50	*11*	11	9	11	14	12	13	12	11	8	8
60	*13*	13	12	13	15	12	13	13	14	11	11
70	*15*	14	15	15	17	13	15	15	19	12	12
80	*16*	16	17	16	18	14	17	16	20	13	15

Study 2 Individual Data: Baseline control and training group blood lactate concentration (mmoL) during cycling lactate threshold testing

		Control Group Blood Lactate Concentration (mmoL)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*1.3*	1.4	1.4	0.9	1.6	1.0	0.9	2.2	1.3	0.8	1.2
50	*1.7*	1.8	1.4	1.7	1.7	1.1	2.0	1.9	1.4	1.7	1.8
60	*2.6*	3.9	2.5	2.4	3.6	1.3	2.6	1.9	1.9	2.5	3.5
70	*3.2*	4.8	3.3	3.7	5.0	1.4	3.5	2.3	2.6	2.8	2.4
80	*5.0*	7.9	4.4	4.5	7.4	2.5	6.0	2.1	5.3	3.7	5.7

		Training Group Blood Lactate Concentration (mmoL)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*1.6*	2.9	2.1	1.3	1.4	1.2	1.0	1.4	1.2	1.8	1.3
50	*2.1*	3.4	3.4	1.1	1.8	2.2	2.3	1.3	1.5	2.1	1.6
60	*2.8*	4.9	4.5	1.8	2.0	2.5	3.4	2.8	1.6	2.5	2.2
70	*3.4*	5.6	4.9	2.5	3.6	2.3	3.4	3.8	2.3	3.0	2.8
80	*4.9*	5.4	8.2	3.9	4.7	1.4	5.8	6.3	3.8	5.4	4.1

Study 2 Individual Data: Post-intervention/training control and training group blood lactate concentration (mmoL) during cycling lactate threshold testing

		Control Group Blood Lactate Concentration (mmoL)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*1.1*	0.8	0.7	0.8	1.6	1.0	1.4	1.3	1.3	0.8	1.2
50	*1.4*	1.5	1.0	0.9	1.7	1.0	1.5	1.3	1.4	1.7	1.8
60	*2.3*	3.2	1.5	1.2	3.5	1.3	2.3	1.9	1.9	2.5	3.5
70	*3.1*	5.1	3.1	2.8	5.1	1.6	3.4	2.0	2.6	2.8	2.4
80	*5.2*	10.5	4.5	5.6	7.3	2.8	4.4	2.4	5.3	3.7	5.7

		Training Group Blood Lactate Concentration (mmoL)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*1.7*	2.1	1.1	2.0	1.5	1.0	2.3	1.1	1.3	2.2	2.0
50	*2.0*	2.2	2.4	1.9	2.1	1.0	2.8	1.6	1.2	2.3	2.2
60	*2.4*	3.3	3.2	1.4	2.9	0.9	1.8	2.8	1.9	2.9	2.5
70	*3.7*	4.5	5.5	3.7	3.3	1.5	4.3	3.0	3.3	3.8	4.2
80	*5.4*	4.9	7.0	5.1	6.0	2.1	7.7	5.5	5.7	4.6	4.9

Study 2 Individual Data: Baseline control and training group total hemoglobin concentration (μM) during cycling lactate threshold testing

		Control Group Total Hemoglobin Concentration (μM)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*56.3*	87.5	59.6	93.4	93.6	35.1	52.0	25.2	43.7	43.0	30.0
50	*59.5*	91.7	62.5	96.1	102.7	35.6	54.4	26.2	45.6	47.2	32.6
60	*61.7*	96.0	64.1	100.4	104.9	37.2	56.5	26.4	50.1	46.7	34.2
70	*62.7*	95.4	63.8	108.0	106.5	38.5	57.0	27.2	51.5	45.0	34.5
80	*65.3*	99.6	66.0	111.5	112.2	43.6	59.2	27.7	51.6	46.9	34.3

		Training Group Total Hemoglobin Concentration (μM)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*61.5*	40.7	81.4	36.6	82.1	36.6	81.2	90.6	43.0	17.5	105.1
50	*63.4*	43.9	85.6	37.0	84.9	37.3	82.2	95.8	47.2	19.3	100.5
60	*63.6*	43.9	86.7	38.9	85.7	37.8	84.5	97.1	46.7	19.2	96.1
70	*64.0*	45.7	85.6	37.2	89.7	39.1	84.8	98.1	45.0	20.5	94.6
80	*64.7*	47.2	87.7	39.6	88.4	40.8	85.1	98.6	46.9	19.1	93.5

Study 2 Individual Data: Post-intervention/training control and training group total hemoglobin concentration (μM) during cycling lactate threshold testing

		Control Group Total Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*53.9*	83.6	59.6	84.0	93.6	35.1	40.3	33.4	41.2	38.1	30.8
50	*57.1*	91.2	62.5	88.4	102.7	35.6	43.2	35.6	42.0	37.9	32.1
60	*58.6*	93.2	64.1	93.5	104.9	37.2	43.5	36.0	43.9	37.9	31.5
70	*59.3*	94.5	63.8	96.1	106.5	38.5	41.3	37.5	45.6	39.4	30.2
80	*61.8*	99.3	66.0	99.2	112.2	43.6	42.9	37.9	46.3	40.2	30.1

		Training Group Total Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*57.2*	32.2	60.3	34.8	80.2	40.1	86.0	92.0	61.7	17.0	68.1
50	*59.9*	36.4	63.6	34.9	83.0	44.7	89.1	97.5	62.4	19.6	68.2
60	*61.7*	36.8	66.1	35.2	85.9	44.3	88.4	101.5	66.5	22.0	69.9
70	*62.8*	33.8	68.2	34.7	88.9	44.9	89.3	108.0	67.1	22.8	69.8
80	*62.8*	33.1	68.5	34.3	87.7	44.9	90.9	105.9	69.5	22.4	70.9

Study 2 Individual Data: Baseline control and training group oxygenated hemoglobin concentration (μM) during cycling lactate threshold testing

		Control Group Oxygenated Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*37.4*	58.4	36.3	58.2	62.1	21.6	35.0	18.7	31.5	29.3	22.4
50	*38.9*	59.5	37.9	54.1	67.5	22.3	38.5	19.7	33.5	31.1	24.8
60	*39.2*	59.3	39.2	54.1	62.9	23.5	39.0	19.8	37.1	31.5	25.7
70	*40.8*	56.8	39.2	58.5	75.8	24.8	38.7	20.4	37.9	29.8	26.0
80	*41.6*	57.2	40.7	56.4	76.1	30.3	40.8	20.7	37.5	30.9	25.6

		Training Group Oxygenated Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*39.6*	26.8	53.4	23.1	47.8	25.6	49.4	61.3	29.3	13.0	66.1
50	*39.7*	30.4	53.8	24.8	44.7	27.1	47.4	63.0	31.1	14.6	60.5
60	*39.1*	30.4	55.8	26.9	44.0	27.7	43.0	61.7	31.5	14.7	55.7
70	*38.1*	31.2	53.9	24.6	45.0	28.2	39.3	59.6	29.8	15.6	53.8
80	*38.8*	32.3	56.2	28.1	44.4	29.5	39.2	58.4	30.9	14.3	54.3

Study 2 Individual Data: Post-intervention/training control and training group oxygenated hemoglobin concentration (μM) during cycling lactate threshold testing

		Control Group Oxygenated Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*35.6*	51.9	36.3	52.5	62.1	21.6	29.5	23.5	30.8	26.5	21.2
50	*37.5*	55.4	37.9	53.2	67.6	22.5	30.9	25.2	30.9	28.1	23.6
60	*37.4*	54.5	39.2	54.9	62.9	23.4	31.3	25.5	32.0	27.9	22.4
70	*38.8*	55.2	39.2	53.8	75.9	24.9	29.2	26.9	33.2	29.0	20.7
80	*39.8*	56.5	40.7	53.8	76.2	30.3	30.1	28.0	33.1	29.3	20.0

		Training Group Oxygenated Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*37.7*	21.5	42.6	22.9	48.8	28.5	51.4	61.6	41.3	11.9	46.4
50	*39.3*	25.8	44.8	23.7	49.9	32.6	49.7	66.6	39.9	14.7	45.6
60	*39.6*	26.7	45.4	23.3	51.6	32.6	44.8	66.8	41.8	16.6	46.4
70	*39.7*	24.8	48.7	23.4	49.2	33.6	42.6	72.1	41.0	16.5	45.1
80	*38.8*	23.7	48.8	21.5	47.5	33.3	43.2	67.6	41.2	16.0	44.8

Study 2 Individual Data: Baseline control and training group deoxygenated hemoglobin concentration (μM) during cycling lactate threshold testing

		Control Group Deoxygenated Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mena**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*18.9*	29.1	23.3	35.2	31.4	13.5	17.0	6.5	12.2	13.7	7.7
50	*20.5*	32.2	24.5	42.0	35.1	13.1	15.9	6.5	12.1	16.1	7.8
60	*22.5*	36.7	24.9	46.3	42.0	13.8	17.5	6.6	13.1	15.2	8.5
70	*21.9*	38.6	24.5	49.6	30.7	13.6	18.2	6.7	13.6	15.2	8.4
80	*23.7*	42.5	25.3	55.1	36.0	13.3	18.5	7.0	14.1	16.0	8.7

		Training Group Deoxygenated Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*21.9*	13.8	28.0	13.5	34.3	11.0	31.8	29.3	13.7	4.5	39.0
50	*23.6*	13.5	31.8	12.2	40.3	10.2	34.8	32.8	16.1	4.7	40.0
60	*24.5*	13.5	31.0	12.0	41.7	10.0	41.5	35.4	15.2	4.5	40.4
70	*25.9*	14.5	31.7	12.6	44.7	10.9	45.5	38.5	15.2	4.8	40.8
80	*25.9*	14.8	31.4	11.5	43.9	11.2	45.9	40.1	16.0	4.8	39.2

Study 2 Individual Data: Post-intervention/training control and training group deoxygenated hemoglobin concentration (μM) during cycling lactate threshold testing

		Control Group Deoxygenated Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*18.4*	31.7	23.3	31.5	31.4	13.5	10.8	9.9	10.4	11.5	9.6
50	*19.6*	35.7	24.5	35.2	35.1	13.1	12.3	10.4	11.1	9.8	8.5
60	*21.2*	38.7	24.9	38.5	42.0	13.8	12.1	10.5	11.9	10.0	9.1
70	*20.6*	39.3	24.5	42.3	30.7	13.6	12.2	10.5	12.4	10.4	9.6
80	*21.9*	42.6	25.3	45.5	36.0	13.3	12.5	10.0	13.2	10.9	10.0

		Training Group Deoxygenated Hemoglobin Concentration (μM)									
Work Rate (%VO_{2max})	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*19.6*	10.7	17.7	11.9	31.4	11.6	34.6	30.4	20.4	5.1	21.7
50	*20.6*	10.6	18.7	11.2	33.1	12.2	39.4	30.9	22.5	4.9	22.6
60	*22.1*	10.1	20.7	11.9	34.3	11.7	43.6	34.7	24.6	5.4	23.6
70	*23.0*	8.9	19.5	11.4	39.6	11.3	46.7	35.9	26.1	6.2	24.7
80	*24.1*	9.4	19.7	12.8	40.2	11.6	47.7	38.3	28.2	6.5	26.1

Study 2 Individual Data: Baseline control and training group percent saturation (%) during cycling lactate threshold testing

		Control Group Percent Saturation (%)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*67.4*	66.7	60.9	62.3	66.4	61.6	67.3	74.1	72.1	68.2	74.5
50	*67.2*	64.8	60.7	56.3	65.7	63.3	70.8	75.2	73.4	65.9	76.1
60	*66.0*	61.7	61.2	53.9	59.9	63.0	69.0	75.0	73.9	67.4	75.1
70	*66.9*	59.5	61.5	54.1	71.1	64.7	68.0	75.2	73.5	66.1	75.5
80	*66.5*	59.5	61.7	49.9	67.9	69.5	68.9	74.7	72.7	65.7	74.7

		Training Group Percent Saturation (%)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*65.6*	65.1	65.6	63.2	58.2	69.9	60.8	67.6	68.2	74.2	62.8
50	*64.9*	69.5	62.8	66.9	52.6	72.6	57.6	65.7	65.9	75.4	60.1
60	*64.4*	69.3	64.3	69.2	51.2	73.5	50.8	63.4	67.4	76.7	57.9
70	*62.6*	68.2	62.9	66.0	50.1	72.2	46.3	60.6	66.1	76.5	56.8
80	*63.0*	68.5	64.1	71.0	50.3	72.5	46.0	59.2	65.8	75.0	58.0

Study 2 Individual Data: Post-intervention/training control and training group percent saturation (%) during cycling lactate threshold testing

		Control Group Percent Saturation (%)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*67.0*	62.1	60.9	62.5	66.4	61.6	73.2	70.3	74.7	69.7	68.7
50	*67.4*	60.8	60.7	60.2	65.7	63.3	71.5	70.8	73.6	74.1	73.4
60	*66.2*	58.4	61.2	58.7	59.9	63.0	72.1	70.8	72.8	73.6	71.1
70	*66.9*	58.4	61.5	56.0	71.1	64.7	70.6	71.9	72.8	73.6	68.3
80	*66.4*	56.5	61.7	54.1	67.8	69.5	70.0	73.7	71.5	72.9	66.6

		Training Group Percent Saturation (%)									
Work Rate ($\%VO_{2max}$)	**Mean**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
40	*66.7*	66.8	70.6	65.9	60.8	71.1	59.7	66.9	66.9	69.9	68.1
50	*67.2*	70.9	70.5	67.9	60.1	72.8	55.7	68.2	63.9	74.9	66.8
60	*66.2*	72.5	68.7	66.3	60.0	73.5	50.6	65.7	63.0	75.5	66.3
70	*65.5*	73.6	71.4	67.2	55.3	74.9	47.7	66.6	61.0	72.7	64.5
80	*63.8*	71.6	71.2	62.6	54.1	74.2	47.5	63.7	59.0	71.1	63.2

Study 2 Individual Data: Training group mean peak power (W) across the five training days

Subject	Day 1	Day 2	Day 3	Day 4	Day 5
1	512	518	497	484	501
2	787	881	911	848	900
3	820	807	759	761	750
4	1251	1216	1273	1337	1281
5	565	596	562	627	650
6	1213	1344	1212	1168	1158
7	1115	1173	1114	1160	1116
8	1802	1697	1823	1788	1799
9	779	809	771	752	768
10	993	1003	950	1009	1018
Mean	*984*	*1004*	*987*	*993*	*994*

Study 2 Individual Data: Training group mean revolutions per minute at peak power across the five training days

Subject	Day 1	Day 2	Day 3	Day 4	Day 5
1	120	109	121	109	113
2	127	127	128	128	126
3	130	131	131	124	126
4	140	140	141	134	142
5	108	112	121	110	127
6	140	149	134	137	145
7	127	140	134	132	133
8	130	138	133	133	134
9	103	110	102	100	100
10	127	120	129	134	120
Mean	*125*	*128*	*127*	*124*	*127*

Study 2 Individual Data: Training group mean relative peak power (W/kg) across the five training days

Subject	Day 1	Day 2	Day 3	Day 4	Day 5
1	8.8	8.9	8.5	8.3	8.6
2	12.1	13.3	14.2	13.0	13.8
3	12.6	12.4	11.7	11.7	11.4
4	16.7	16.2	16.9	18.0	17.3
5	9.9	9.6	9.0	10.1	10.5
6	16.9	18.8	16.7	16.2	16.1
7	15.1	15.9	15.1	15.7	15.1
8	15.2	14.4	15.4	15.1	15.2
9	10.9	11.3	10.8	10.5	10.8
10	15.5	15.6	14.8	15.7	15.8
Mean	*13.4*	*13.6*	*13.3*	*13.4*	*13.5*

Study 2 Individual Data: Training group average heart rate (bpm) during the training session across the five training days

Subject	Day 1	Day 2	Day 3	Day 4	Day 5
1	103	111	103	106	106
2	80	93	94	95	96
3	87	83	83	97	102
4	100	91	111	101	101
5	94	95	107	100	99
6	99	110	103	98	103
7	88	88	82	95	96
8	98	95	93	93	95
9	101	101	100	106	98
10	83	91	77	81	83
Mean	*93*	*96*	*95*	*97*	*98*

Study 2 Individual Data: Training group average peak heart rate (bpm) during the training session across the five training days

Subject	Day 1	Day 2	Day 3	Day 4	Day 5
1	130	136	129	131	131
2	111	123	135	117	117
3	124	112	107	125	128
4	119	119	138	110	120
5	120	119	121	127	127
6	125	128	129	118	123
7	115	116	111	121	129
8	128	126	121	121	120
9	128	128	128	132	128
10	106	105	104	107	106
Mean	*121*	*121*	*122*	*121*	*123*

Study 2 Individual Data: Training group average resting heart rate (bpm) during the training session across the five training days

Subject	Day 1	Day 2	Day 3	Day 4	Day 5
1	94	93	89	91	91
2	65	75	84	78	83
3	58	67	67	75	81
4	84	75	97	85	85
5	78	72	93	78	91
6	78	93	78	80	80
7	61	59	54	60	62
8	80	76	74	74	72
9	74	74	72	81	69
10	63	63	60	65	64
Mean	*74*	*75*	*77*	*77*	*78*

APPENDIX D.

Research Consent Forms.

Consent for Participation in Research

Title: *Maximal Oxygen Consumption When Cycling or Running*

Introduction

The purpose of this form is to provide you information that may affect your decision as to whether or not to participate in this research study. The person performing the research will answer any of your questions. Read the information below and ask any questions you might have before deciding whether or not to take part. If you decide to be involved in this study, this form will be used to record your consent.

Purpose of the Study

You have been asked to participate in a research study about the relationship between your aerobic fitness capability and endurance performance. The purpose of this study is measure your level of physical fitness for running or bicycling. These measures can be used to compare your individual attributes to those of previous endurance athletes who have been studied in the Human Performance Lab (HPL). We also want to determine if athletes who have been performing endurance training for a greater number of years have a higher maximal oxygen consumption (VO_{2max}) than the athletes with fewer years of training.

What will you be asked to do?

If you agree to participate in this you will be asked to:

1. Complete a health history questionnaire.
2. Have your height and weight measured.
3. Apply a heart rate monitor strap to your chest.
4. Have your maximal oxygen consumption measured during cycling or running.
5. Cool down for about 10 minutes before leaving the laboratory.

This study will take **one visit** of **approximately 45 minutes** and will include approximately **50** study participants.

Overview of Procedures:

Health History (~15min): Before you can be admitted to the study, you will be given a brief examination. This examination will include filling out a brief Health History Questionnaire, and taking measurements of your height and weight.

Maximal Oxygen Consumption (~30 min.): You will be asked to perform a maximal oxygen consumption test (VO_{2max}), which will take between 6 – 12 minutes. The intensity of exercise will be increased every 1-2 min. until you are at your maximal effort level and cannot maintain the exercise speed. The sensation of effort and fatigue during the last 1-2 min will be comparable to a race. During the test, you will breathe into a mouthpiece, while wearing a nose clip that will collect and analyze the O_2 and CO_2 content of expired air. In addition, a heart rate monitor will be worn around the chest that will be used to monitor heart rate throughout the course of the study. From this data we can determine your VO_{2max}.

The maximal oxygen consumption protocol can be done either while running on a treadmill or while cycling on a stationary cycle ergometer. Both protocols will require similar incremental increases in exercise intensity, leading to fatigue in 6-12 min.

Please initial below next to which test you will be performing today.

_____ I have chosen to perform the maximal oxygen consumption test **while riding a stationary cycle ergometer** while the resistance increases progressively.

_____ I have chosen to perform the maximal oxygen consumption test **while running on a treadmill**
while progressively increasing treadmill speed and incline.

Your participation **may** be **photographed or videotaped** during the course of this experiment. Any photographs and/or videotapes of your performance (without your name or likeness revealed) may be shown to educational audiences, such as conferences.

What are the risks involved in this study?

This maximal oxygen consumption test may involve risks that are currently unforeseeable. Possible risks associated with this study are:

The fatigue test to measure VO_{2max} will feel like a very short race or a single bout of intense interval training. There is a very small risk that you could experience a muscular injury, such as a muscle strain. It is possible, although very rare, that intense exercise such as performed in this study might cause a heart attack. During the test you will have the sensation of fatigue, this fatigue will be similar to what is felt during a

short race or training session. During the tests, you may stop performing the task at any time for any reason if you feel you need to do so.

What are the possible benefits of this study?

You will receive no direct benefit from participating in this study; however, each subject completing the study will be provided with information about his or her VO2max, which is useful to running and bicycling training and performance.

Do you have to participate?

No, your participation is voluntary. You may decide not to participate at all or, if you start the study, you may withdraw at any time. Withdrawal or refusing to participate will not affect your relationship with The University of Texas at Austin (University) in anyway.

If you would like to participate please fully read, sign, and return this form to the principal investigator of this study (Brian Leary). You will receive a copy of this form for your personal records.

What are the potential Conflicts of Interest?

Dr. Coyle holds equity in Sports Texas: Fitness, Training and Nutrition, Inc., a company that consults on the topics of physical training and monitoring athletic progress. The business interests of Sports Texas: Fitness, Training and Nutrition, Inc. overlap with this area of research.

Will there be any compensation?

You will not receive any type of payment participating in this study.

What if you are injured because of the study?

1. The University has no program or plan to provide treatment for research related injury or payment in the event of a medical problem. In the event of a research related injury, please contact the principal investigator.
2. The University has no program or plan for continuing medical care and/or hospitalization for research-related injuries or for financial compensation.
3. If injuries occur as a result of study activity, eligible University students may be treated at the usual level of care with the usual cost for services at the Student Health Center, but the University has no program or plan to provide

payment in the event of a medical problem.

How will your privacy and confidentiality be protected if you participate in this research study?

Each subject will be assigned a unique Subject ID code. This informed consent form and the Health History Questionnaire are the only places where any personal identifying information will be recorded. These forms will be stored in a locked file cabinet. In all other cases, your data will only be identifiable by your unique code. Only the director of the laboratory (Dr. Coyle) will have access to a master list that will link your identity to your code.

Because you will be participating in this study and may do so along with other subjects in a small group, we will ask that you do not disclose names of participants in your group or any information that was discussed with other group members outside of the experimental session.

If it becomes necessary for the Institutional Review Board to review the study records, information that can be linked to you will be protected to the extent permitted by law. Your research records will not be released without your consent unless required by law or a court order. The data resulting from your participation may be made available to other researchers in the future for research purposes not detailed within this consent form. In these cases, the data will contain no identifying information that could associate it with you, or with your participation in any study.

If you choose to participate in this study, you may be photographed or video recorded. Any photographs or video recordings will be stored securely and only the research team will have access to the recordings. Recordings will be kept for 3 years after the research experiment has been completed and then erased.

Whom to contact with questions about the study?

Prior, during or after your participation you can contact the researcher **Brian Leary** at **(512)471-8598** or send an email to **briankleary@austin.utexas.edu** for any questions or if you feel that you have been harmed.

This study has been reviewed and approved by The University Institutional Review Board and the study number is **2014-08-0082.**

Whom to contact with questions concerning your rights as a research participant?

For questions about your rights or any dissatisfaction with any part of this study,

you can contact, anonymously if you wish, the Institutional Review Board by phone at (512) 471-8871 or email at orsc@uts.cc.utexas.edu.

Participation

If you agree to participate please sign and return this form to a member of the research team.

Signature

You have been informed about this study's purpose, procedures, possible benefits and risks, and you have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time. You voluntarily agree to participate in this study. By signing this form, you are not waiving any of your legal rights.

NOTE: Include the following if recording is optional:

_____ I agree to be **photographed/video** recorded.

_____ I do not want to be **photographed/video** recorded.

_____

Printed Name

_____ _____

Signature Date

As a representative of this study, I have explained the purpose, procedures, benefits, and the risks involved in this research study.

_____

Print Name of Person obtaining consent

_____ _____

Signature of Person obtaining consent Date

Consent for Participation in Research

Title: *Lactate Threshold While Cycling or Running*

Introduction

The purpose of this form is to provide you information that may affect your decision as to whether or not to participate in this research study. The person performing the research will answer any of your questions. Read the information below and ask any questions you might have before deciding whether or not to take part. If you decide to be involved in this study, this form will be used to record your consent.

Purpose of the Study

You have been asked to participate in a research study about your level of physical fitness as measured by your blood lactic acid concentration during cycling or running. These measures can be used to compare your individual attributes to those of previous endurance athletes who have been studied in the HPL.

What will you be asked to do?

You will be asked to report to the Human Performance Laboratory at the University of Texas at Austin, located in Bellmont Hall, Room 820. You should wear a shirt, shorts, and tennis shoes. Gentlemen may be asked to perform shirtless. This visit will take about 45-60min. The experimental procedures are as follows:

1. Complete a health history questionnaire
2. Have your height and weight measured
3. Allow the researcher to insert a Teflon catheter in your arm for blood draws
4. Apply a heart rate monitor strap to your chest
5. Wear a mouth piece to measure your volume of oxygen consumed
6. Permit 7 blood draws to measure your blood lactate concentration
7. Cycle or run on for 30 minutes as your intensity of exercise increases every 5 minutes
8. After 30 minutes, a cool down of easy exercise will continue for 10 minutes

This study will take **one visit** of **approximately 75 minutes** and will include approximately **50** study participants.

Health History Questionnaire (~15mins): Before you can be admitted to the study, you will be given a brief examination. This examination will include filling out a brief

Health History Questionnaire, and taking measurements of your height and weight. Only if you are apparently healthy with no prior history of disease or medical condition will you be permitted to participate in the study. Specific exclusion criteria include the following: history of heart disease or coronary artery disease, hypertension, lung or respiratory problems, persistent chest pain during and/or after exercise, fainting or loss of consciousness during exercise, and palpitations/arrhythmias during exercise.

Blood Measurements: At the beginning of the lactate threshold testing session, a 1-1/2 inch flexible Teflon catheter will be inserted, under sterile conditions, into a forearm vein and taped into place. Blood will be drawn from this catheter 7 times for each testing session (1 resting value and 1 for each of the 6 submaximal stages.
These blood samples will be used to determine blood lactic acid concentration and then your lactate threshold.

Lactate Threshold (~60 min): Once you are familiarized with the exercise equipment you will be asked to exercise for 30 min. During this time, the intensity of exercise will be increased every 5 min. The intensity of exercise during the 6 stages will be approximately 40, 50, 60, 70, 80 and 90% of your maximal oxygen consumption. This progressive exercise will feel like a 'warm-up' and should elicit only moderate fatigue during the last 5-10 min.

Before you start exercise and at the end of each of the 6 stages of exercise, a small sample of blood (1 milliliter or
< 0.1 tablespoons) will be obtained from the venous catheter. Lactic acid levels in your blood and 'lactate threshold' will be determined, which is another predictor of performance ability. Your blood sample will be frozen, stored, and will be kept frozen for less than one month after your visit to the laboratory.

During the test, you will breathe into a mouthpiece, while wearing a nose-clip, that will collect and analyze the O_2 and CO_2 content of expired air. From this we can determine your oxygen consumption and precisely determine how hard you are working. In addition, a heart rate monitor will be worn as a strap around your chest so we can also determine your lactate threshold and heart rate relationship.

The lactate threshold protocol can be done either while running on a treadmill or while cycling on a stationary cycle ergometer. Both protocols will require the same level of exercise intensity. ***Please initial below next to which test you will be performing today.***

___ I have chosen to perform the lactate threshold test **while riding a stationary cycle ergometer.** I understand that intensity will be determined based by increasing the resistance on the cycle ergometer based on my individual level of fitness.

___ I have chosen to perform the lactate threshold test **while running on a treadmill.** I understand that intensity will be adjusted by increasing treadmill speed and incline based on my individual level of fitness.

Your participation **may** be **photographed/video** recorded.

What are the risks involved in this study?

None of the above procedures are expected to be unduly painful or uncomfortable in a healthy individual. There is a very small risk that you could experience a muscular injury, such as a muscle strain. It is possible, although very rare, that intense exercise such as performed in this study might cause a heart attack. A minimal amount of fatigue may occur during the test, the amount of fatigue typically experienced is similar to what you would encounter during a short race or training session. You will be asked to use a mouthpiece to measure oxygen consumption, this may provide slight discomfort but feels similar to a mouth guard commonly used in sporting activities. During the tests, you may stop performing the task at any time for any reason if you feel you need to do so.

Approximately one tablespoon of blood will be obtained via draws from the venous catheter. You may experience some mild pain and irritation during the catheter insertion procedure and the catheter sometimes is uncomfortable, although rarely, during exercise. There is also a very slight risk of infection, bruising, excessive bleeding, pain, fainting/light headedness, and blood clotting. To minimize this risk, only sterile and/or disposable equipment will be used and the skin where the catheter is inserted will be swabbed with rubbing alcohol both before and immediately after the procedure. Additionally, you will be asked to be well hydrated and eat a light meal before you arrive to perform the lactate threshold test to minimize risk of fainting.

What are the possible benefits of this study?

You will receive no direct benefits from participating in this research study. However, after completing the study you will be provided with information useful to training and performance, including your lactate threshold.

Do you have to participate?

No, your participation is voluntary. You may decide not to participate at all or, if you start the study, you may withdraw at any time. Withdrawal or refusing to participate will not affect your relationship with The University of Texas at Austin (University) in any way.

If you would like to participate please read and sign the consent form and return to the study investigator. You will receive a copy of this form for your personal records.

Will there be any compensation?

You will not receive any type of payment participating in this study.

What if you are injured because of the study?

1. The University has no program or plan to provide treatment for research related injury or payment in the event of a medical problem. In the event of a research related injury, please contact the principal investigator.
2. The University has no program or plan for continuing medical care and/or hospitalization for research- related injuries or for financial compensation.
3. If injuries occur as a result of study activity, eligible University students may be treated at the usual level of care with the usual cost for services at the Student Health Center, but the University has no program or plan to provide payment in the event of a medical problem.

How will your privacy and confidentiality be protected if you participate in this research study?

Each subject will be assigned a unique Subject ID code. This informed consent form and the Health History Questionnaire are the only places where any personal identifying information will be recorded. These forms will be stored in a locked file cabinet. In all other cases, your data will only be identifiable by your unique code. Only the director of the laboratory (Dr. Coyle) will have access to a master list that will link your identity to your code. The identifiable information collected will be deleted one year after the completion of the research study.

Because you will be participating in this study and may do so along with other subjects in a small group, we will ask that you do not disclose names of participants in your group or any information that was discussed with other group members outside of the experimental session.

Authorized persons from The University of Texas at Austin and the Institutional Review Board have the legal right to review your research records and will protect the confidentiality of those records to the extent permitted by law. Otherwise, your research records will not be released without your consent unless required by law or a court order. If the results of this research are

published or presented at scientific meetings, your identity will not be disclosed.

If it becomes necessary for the Institutional Review Board to review the study records, information that can be linked to you will be protected to the extent permitted by law. Your research records will not be released without your consent unless required by law or a court order. The data resulting from your participation may be made available to other researchers in the future for research purposes not detailed within this consent form. In these cases, the data will contain no identifying information that could associate it with you, or with your participation in any study.

If you choose to participate in this study, you **may be photographed/video** recorded. Any **photographs or video** recordings will be stored securely and only the research team will have access to the recordings. These photographs and video recordings will be used for instructional purposes with permission from the subject. Recordings will be kept for **3 yrs. past the date of study completion** and then erased.

Whom to contact with questions about the study?

Prior, during or after your participation you can contact the researcher **Brian Leary** at **(512)471-8598** or send an email to **briankleary@austin.utexas.edu** for any questions or if you feel that you have been harmed.

This study has been reviewed and approved by The University Institutional Review Board and the study number is
2014-08-0083.

Whom to contact with questions concerning your rights as a research participant?

For questions about your rights or any dissatisfaction with any part of this study, you can contact, anonymously if you wish, the Institutional Review Board by phone at (512) 471-8871 or email at orsc@uts.cc.utexas.edu.

Participation

If you agree to participate **please sign below and return to the principal investigator.**

Signature

You have been informed about this study's purpose, procedures, possible benefits and risks, and you have received a copy of this form. You have been given the

opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time. You voluntarily agree to participate in this study. By signing this form, you are not waiving any of your legal rights.

____ I agree to be **photographed or video** recorded for instructional purposes.

____ I do not want to be **photographed or video** recorded for instructional purposes.

_____

Printed Name

_____ _____

Signature Date

As a representative of this study, I have explained the purpose, procedures, benefits, and the risks involved in this research study.

_____

Print Name of Person obtaining consent

_____ _____

Signature of Person obtaining consent Date

Consent for Participation in Research

Title: *Biomechanical Differences in Cycling*.

Introduction

The purpose of this form is to provide you information that may affect your decision as to whether or not to participate in this research study. The person performing the research will answer any of your questions. Read the information below and ask any questions you might have before deciding whether or not to take part. If you decide to be involved in this study, this form will be used to record your consent.

Purpose of the Study

You have been asked to participate in a research study about the biomechanical differences in cycling that occur between various type of endurance athletes. The purpose of this study is to measure your aerobic fitness and cycling biomechanical strategy. These measures can be used to compare your individual attributes to those of other endurance athletes who have been studied in the Human Performance Lab (HPL).

What will you be asked to do?

If you agree to participate in this study, you will be asked to:

- Complete a health history questionnaire.
- Have your height and weight measured.
- Have your maximal oxygen consumption measured while cycling.
- Have your cycling strategy analyzed while cycling.

This study will take **three visits** of **approximately 60 minutes** and will include approximately **50** study participants.

Overview of Procedures:

Visit # 1: Health History and Maximal Oxygen Consumption

- Health History Questionnaire (~15minutes): Before you can be admitted to the study, you will be given a brief examination. This examination will include filling out a brief Health History Questionnaire, and taking measurements of your height and weight.

- Maximal Oxygen Consumption (~30 min.): You will be asked to perform a maximal oxygen consumption test (VO_{2max}), which will take between 6 – 12 minutes. The intensity of exercise will be increased every 1-2 min. until you are at your maximal effort level and cannot maintain the exercise speed. The sensation of effort and fatigue during the last 1-2 min will be comparable to a race. During the test, you will breathe into a mouthpiece, while wearing a nose clip that will collect and analyze the O_2 and CO_2 content of expired air. In addition, a heart rate monitor will be worn around the chest that will be used to monitor heart rate throughout the course of the study. From this data, we can determine your VO_{2max}.

Visit #2&3: Biomechanical Assessment and Near Infrared Spectroscopy (Visit 2 will serve as a familiarization for the biomechanical protocol. Visit 3 will be a repeat of the procedures followed during Visit 2).

- Subject Preparation (~30minutes):
    - Before beginning the exercise protocol, you will need to be prepped for the biomechanical assessment. During this time, we will mark your skin site at the appropriate location for electrode placement that will be used to determine how hard your muscles are working. Your skin will be cleaned with an alcohol pad and shaved with a razor to provide a more accurate measurement of muscle activity. Electrodes will be placed on your skin over your muscles of the lower extremity and secured with double sided tape and athletic wrap. Reflective markers will be secured to your upper and lower extremity so the motion analysis software can detect where you are at in the lab. Shoes will be provided for you that care custom designed for our pedals, and you will wear these for the duration of the test.
    - A probe will be placed over your thigh muscle emits near-infrared light and calculate the oxygen concentration within the muscle in a non-invasive manner. The probe will be secured to the skin with double sided tape and wrapped with athletic wrap.

- Biomechanical Exercise Protocol (~30minutes):
    - You will be asked to ride at a submaximal effort at 3 different workloads for 5 minutes. A warm-up period of 5 minutes will be given so you can become accustom with the bicycle. After the warm-up period, you will exercise at either 200 watts, 300 watts, and 80% of your VO2max for 5 minutes at each workload; the order of testing will be randomized. After you have completed the testing session a 10-minute cool-down will have provided. From this data, we can determine your bicycling biomechanics.

Your participation **may** be **video** recorded. Any photographs and/or videotapes of your performance (without your name or likeness revealed) may be shown to educational audiences, such as conferences.

What are the risks involved in this study?

This maximal oxygen consumption test may involve risks that are currently unforeseeable. Possible risks associated with this study are:

The fatigue test to measure VO_{2max} will feel like a very short race or a single bout of intense interval training. There is a very small risk that you could experience a muscular injury, such as a muscle strain. It is possible, although very rare, that intense exercise such as performed in this study might cause a heart attack. During the test, you will have the sensation of fatigue, this fatigue will be similar to what is felt during a short race or training session. During

the tests, you may stop performing the task at any time for any reason if you feel you need to do so.

What are the possible benefits of this study?

You will receive no direct benefit from participating in this study; however, each subject completing the study will be provided with information about his or her VO_{2max}, which is useful to running and bicycling training and performance.

Do you have to participate?

No, your participation is voluntary. You may decide not to participate at all or, if you start the study, you may withdraw at any time. Withdrawal or refusing to participate will not affect your relationship with The University of Texas at Austin (University) in anyway.

If you would like to participate please fully read, sign, and return this form to the principal investigator of this study (Brian Leary). You will receive a copy of this form for your personal records.

Will there be any compensation?

You will not receive any type of payment participating in this study.

What if you are injured because of the study?

1. The University has no program or plan to provide treatment for research related injury or payment in the event of a medical problem. In the event of a research related injury, please contact the principal investigator.
2. The University has no program or plan for continuing medical care and/or hospitalization for research-related injuries or for financial compensation.
3. If injuries occur as a result of study activity, eligible University students may be treated at the usual level of care with the usual cost for services at the Student Health Center, but the University has no program or plan to provide payment in the event of a medical problem.

How will your privacy and confidentiality be protected if you participate in this research study?

Each subject will be assigned a unique Subject ID code. This informed consent form and the Health History Questionnaire are the only places where any personal identifying information will be recorded. These forms will be stored in a locked file cabinet. In all other cases, your data will only be identifiable by your unique code. Only the director of the laboratory (Dr. Coyle) will have access to a master list that will link your identity to your code.

Because you will be participating in this study and may do so along with other subjects in a small group, we will ask that you do not disclose names of participants in your group or any information that was discussed with other group members outside of the experimental session.

If it becomes necessary for the Institutional Review Board to review the study records, information that can be linked to you will be protected to the extent permitted by law. Your research records will not be released without your consent unless required by law or a court order. The data resulting from your participation may be made available to other researchers in the future for research purposes not detailed within this consent form. In these cases, the data will contain no identifying information that could associate it with you, or with your participation in any study.

If you choose to participate in this study, you may be photographed or video recorded. Any photographs or video recordings will be stored securely and only the research team will have access to the recordings. Recordings will be kept for 3 years after the research experiment has been completed and then erased.

What are the potential Conflicts of Interest?

Dr. Coyle holds equity in Sports Texas: Fitness, Training and Nutrition, Inc., a company that consults on the topics of physical training and monitoring athletic progress. The business interests of Sports Texas: Fitness, Training and Nutrition, Inc. overlap with this area of research.

Whom to contact with questions about the study?

Prior, during or after your participation you can contact the researcher **Brian Leary** at **(512)471-8598** or send an email to **briankleary@austin.utexas.edu** for any questions or if you feel that you have been harmed.

This study has been reviewed and approved by The University Institutional Review Board and the study number is **[STUDY NUMBER].**

Whom to contact with questions concerning your rights as a research participant?

For questions about your rights or any dissatisfaction with any part of this study, you can contact, anonymously if you wish, the Institutional Review Board by phone at (512) 471-8871 or email at orsc@uts.cc.utexas.edu.

Participation

If you agree to participate to a member of the research team.

Signature

You have been informed about this study's purpose, procedures, possible benefits and risks, and you have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time. You voluntarily agree to participate in this study. By signing this form, you are not waiving any of your legal rights.

_____ I agree to **video** recorded.
_____ I do not want to be **video** recorded.

_____
Printed Name

_____ _____
Signature Date

As a representative of this study, I have explained the purpose, procedures, benefits, and the risks involved in this research study.

_____
Print Name of Person obtaining consent

_____ _____
Signature of Person obtaining consent Date

Consent for Participation in Research

Title: *Lactate Threshold Following Short-Term Training*

Introduction

The purpose of this form is to provide you information that may affect your decision as to whether or not to participate in this research study. The person performing the research will answer any of your questions. Read the information below and ask any questions you might have before deciding whether or not to take part. If you decide to be involved in this study, this form will be used to record your consent.

Purpose of the Study

You have been asked to participate in a research study about change in lactate threshold following a short-term cycling training program. These measures can be used to compare your individual attributes to those of other endurance athletes who have been studied in the Human Performance Lab (HPL).

What will you be asked to do?

You will be asked to report to the Human Performance Laboratory at the University of Texas at Austin, located in Bellmont Hall, Room 820. You should wear a shirt, shorts, and tennis shoes. Gentlemen may be asked to perform shirtless. The testing sessions will span over 10 days, each visit will take between 30 to 120 minutes (details below). The experimental procedures are as follows (see Figure 1 below for graphical overview):

Overview of Procedures:

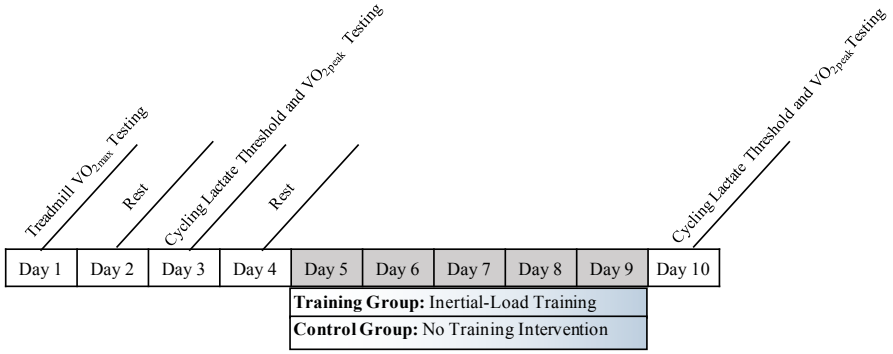

Figure 1. Study Overview

Day # 1: Health History and Treadmill Maximal Oxygen Consumption (~60 minutes)

- Health History Questionnaire (~15minutes): Before you can be admitted to the study, you will be given a brief examination. This examination will include filling

out a brief Health History Questionnaire, and taking measurements of your height and weight.

- Treadmill Maximal Oxygen Consumption (~30 min.): You will be asked to perform a maximal oxygen consumption test (VO_{2max}), which will take between 8 – 12 minutes. The intensity of exercise will be increased every 1-2 min. until you are at your maximal effort level and cannot maintain the exercise speed. The sensation of effort and fatigue during the last 1-2 min will be comparable to a race. During the test, you will breathe into a mouthpiece, while wearing a nose clip that will collect and analyze the O_2 and CO_2 content of expired air. In addition, a heart rate monitor will be worn around the chest that will be used to monitor heart rate throughout the course of the study. From this data, we can determine your treadmill VO_{2max}.

Day #2: Rest Day: This day will serve as a rest day, you will be asked to refrain from vigorous exercise on this day and maintain your normal diet pattern.

Day #3 & 10: Lactate Threshold, Near Infrared Spectroscopy (NIRS), and Cycling Peak Oxygen Consumption (~120 minutes total)

- Subject Preparation (~30minutes):
  - Before you start a venous catheter will be inserted into your arm, and will be used to collect a resting blood measurement as well as exercising blood measurements.
  - Before beginning the exercise protocol, you will need to be prepped for near-infrared spectroscopy assessment. We will mark and clean a skin site over one of the muscle of your thigh, and then shave with a razor and rub with an alcohol wipe. Shoes will be provided for you that are custom designed for our pedals, and you will wear these for the duration of the test.
  - A probe will be placed over your thigh muscle emits near-infrared light and calculate the oxygen concentration within the muscle in a non-invasive manner. The probe will be secured to the skin with double sided tape and wrapped with athletic wrap.
  - After completion of the submaximal protocol subjects will have the venous catheter removed and the NIRS device taken off before the rest period starts.

- Submaximal Exercise Protocol (~30minutes):
  - You will be asked to exercise for 30 minutes. During this time, the intensity of exercise will be increased every 5 min. The intensity of exercise during the 6 stages will be approximately 40,50,60,70,80& 90% of your maximal oxygen consumption. This progressive exercise

test will feel like a 'warm-up' and should elicit only moderate fatigue during the last 5-10min.
    - During the final minute of each stage a small amount (1 milliliter or <0.1 tablespoons) of blood will be obtained from the venous catheter. Lactic acid levels in your blood and 'lactate threshold' will be determined.
    - During this test, you will breathe into a mouthpiece, while wearing a nose-clip, that will collect and analyze oxygen and carbon dioxide content of expired air.
    - Following the submaximal protocol you will be give 30 minutes to rest. After completion of the submaximal test the venous catheter and NIRS probe will be removed.

- Cycling Peak Exercise Protocol (~8-12 minutes):
    - You will be asked to perform a maximal oxygen consumption test (VO_{2max}), which will take between 8 – 12 minutes. The intensity of exercise will be increased every 1-2 min. until you are at your maximal effort level and cannot maintain the exercise speed. The sensation of effort and fatigue during the last 1-2 min will be comparable to a race. During the test, you will breathe into a mouthpiece, while wearing a noseclip that will collect and analyze the O_2 and CO_2 content of expired air. In addition, a heart rate monitor will be worn around the chest that will be used to monitor heart rate throughout the course of the study. From this data, we can determine your cycling VO_{2peak}.

Days #5-9: Control or Training Intervention (~30minutes):
If you are assigned to the Control group you will be asked to maintain your normal diet and exercise regimen throughout Days 5-9. If you are assigned to the Training group you will be asked to come to the laboratory on Days 5-9 to perform the training protocol below.

- Warm-Up (15 minutes): You will perform 5 minutes of active warm-up (i.e. squats and stretching). Immediately following the 5 minute of active warm-up you will ride a cycle ergometer for 5 minutes at 100 watts with a cadence of 100-120rpms. Five minutes of rest will be given after the warm-up and after you will perform the training protocol.
- Inertial-Load Training: You will perform 10 'maximal' sprints lasting ~4 seconds on the cycle ergometer. You will be asked to pedal as hard and fast as possible for the 4 seconds. After each repetition, you will be give 2 minutes of rest.

Health History Questionnaire: Before you can be admitted to the study, you will be given a brief examination. This examination will include filling out a brief Health

History Questionnaire, and taking measurements of your height and weight. Only if you are apparently healthy with no prior history of disease or medical condition will you be permitted to participate in the study. Specific exclusion criteria include the following: history of heart disease or coronary artery disease, hypertension, lung or respiratory problems, persistent chest pain during and/or after exercise, fainting or loss of consciousness during exercise, and palpitations/arrhythmias during exercise.

Blood Measurements: At the beginning of the lactate threshold testing session, a 1-1/2 inch flexible Teflon catheter will be inserted, under sterile conditions, into a forearm vein and taped into place. Blood will be drawn from this catheter 7 times for each testing session (1 resting value and 1 for each of the 6 submaximal stages. These blood samples will be used to determine blood lactic acid concentration and then your lactate threshold.

Lactate Threshold: Once you are familiarized with the exercise equipment you will be asked to exercise for 30 min. During this time, the intensity of exercise will be increased every 5 min. The intensity of exercise during the 6 stages will be approximately 40, 50, 60, 70, 80 and 90% of your maximal oxygen consumption. This progressive exercise will feel like a 'warm-up' and should elicit only moderate fatigue during the last 5-10 min.

Before you start exercise and at the end of each of the 6 stages of exercise, a small sample of blood (1 milliliter or < 0.1 tablespoons) will be obtained from the venous catheter. Lactic acid levels in your blood and 'lactate threshold' will be determined, which is another predictor of performance ability. Your blood sample will be frozen, stored, and will be kept frozen for less than one month after your visit to the laboratory.

During the test, you will breathe into a mouthpiece, while wearing a nose-clip, that will collect and analyze the O_2 and CO_2 content of expired air. From this we can determine your oxygen consumption and precisely determine how hard you are working. In addition, a heart rate monitor will be worn as a strap around your chest so we can also determine your lactate threshold and heart rate relationship.

Your participation **may** be **photographed/video** recorded.

What are the risks involved in this study?

None of the above procedures are expected to be unduly painful or uncomfortable in a healthy individual. There is a very small risk that you could experience a muscular injury, such as a muscle strain. It is possible, although very rare, that intense exercise such as performed in this study might cause a heart attack. A minimal amount of fatigue may occur during the test, the amount of fatigue typically experienced is similar to what you would encounter during a short race or training session. You will be asked to use a mouthpiece to measure oxygen consumption, this may provide slight discomfort but feels similar to a mouth guard commonly used in sporting activities. During the tests, you may stop performing the task at any time for any reason if you feel you need to do so.

Approximately one tablespoon of blood will be obtained via draws from the venous catheter. You may experience some mild pain and irritation during the catheter

insertion procedure and the catheter sometimes is uncomfortable, although rarely, during exercise. There is also a very slight risk of infection, bruising, excessive bleeding, pain, fainting/light headedness, and blood clotting. To minimize this risk, only sterile and/or disposable equipment will be used and the skin where the catheter is inserted will be swabbed with rubbing alcohol both before and immediately after the procedure. Additionally, you will be asked to be well hydrated and eat a light meal before you arrive to perform the lactate threshold test to minimize risk of fainting. To further minimize the risk of fainting you will be asked to lie in a supine position while the venous catheter is inserted.

What are the possible benefits of this study?

You will receive no direct benefits from participating in this research study. However, after completing the study you will be provided with information useful to training and performance, including your VO_{2max} and lactate threshold.

Do you have to participate?

No, your participation is voluntary. You may decide not to participate at all or, if you start the study, you may withdraw at any time. Withdrawal or refusing to participate will not affect your relationship with The University of Texas at Austin (University) in anyway.

If you would like to participate please read and sign the consent form and return to the study investigator. You will receive a copy of this form for your personal records.

Will there be any compensation?

You will not receive any type of payment participating in this study.

What if you are injured because of the study?

4. The University has no program or plan to provide treatment for research related injury or payment in the event of a medical problem. In the event of a research related injury, please contact the principal investigator.
5. The University has no program or plan for continuing medical care and/or hospitalization for research-related injuries or for financial compensation.
6. If injuries occur as a result of study activity, eligible University students may be treated at the usual level of care with the usual cost for services at the Student Health Center, but the University has no program or plan to provide payment in the event of a medical problem.

How will your privacy and confidentiality be protected if you participate in this research study?

Each subject will be assigned a unique Subject ID code. This informed consent form and the Health History Questionnaire are the only places where any personal identifying information will be recorded. These forms will be stored in a locked file cabinet. In all other cases, your data will only be identifiable by your unique code. Only the director of the laboratory (Dr. Coyle) will have access to a master list that will link your identity to your code. The identifiable information collected will be deleted one year after the completion of the research study.

Because you will be participating in this study and may do so along with other subjects in a small group, we will ask that you do not disclose names of participants in your group or any information that was discussed with other group members outside of the experimental session.

Authorized persons from The University of Texas at Austin and the Institutional Review Board have the legal right to review your research records and will protect the confidentiality of those records to the extent permitted by law. Otherwise, your research records will not be released without your consent unless required by law or a court order. If the results of this research are published or presented at scientific meetings, your identity will not be disclosed.

If it becomes necessary for the Institutional Review Board to review the study records, information that can be linked to you will be protected to the extent permitted by law. Your research records will not be released without your consent unless required by law or a court order. The data resulting from your participation may be made available to other researchers in the future for research purposes not detailed within this consent form. In these cases, the data will contain no identifying information that could associate it with you, or with your participation in any study.

If you choose to participate in this study, you **may be photographed/video** recorded. Any **photographs or video** recordings will be stored securely and only the research team will have access to the recordings. These photographs and video recordings will be used for instructional purposes with permission from the subject. Recordings will be kept for **3 yrs. past the date of study completion** and then erased.

Whom to contact with questions about the study?

Prior, during or after your participation you can contact the researcher **Brian Leary** at **(512)471-8598** or send an email to **briankleary@austin.utexas.edu** for any questions or if you feel that you have been harmed.

This study has been reviewed and approved by The University Institutional Review Board and the study number is **2014-08-0083.**

Whom to contact with questions concerning your rights as a research participant?

For questions about your rights or any dissatisfaction with any part of this study, you can contact, anonymously if you wish, the Institutional Review Board by phone at (512) 471-8871 or email at orsc@uts.cc.utexas.edu.

Financial Conflict of Interest Disclosure

The faculty advisor of the student researcher who is leading this study, Dr. Edward F. Coyle, holds equity in and consults on physical training and monitoring athletic progress for a company called Sports Texas: Fitness, Training, and Nutrition, Inc. The business interests of this company overlap with the topic of this study.

Participation

If you agree to participate **please sign below and return to the principal investigator.**

Signature

You have been informed about this study's purpose, procedures, possible benefits and risks, and you have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time. You voluntarily agree to participate in this study. By signing this form, you are not waiving any of your legal rights.

_____ I agree to be **photographed or video** recorded for instructional purposes.

_____ I do not want to be **photographed or video** recorded for instructional purposes.

_____
Printed Name

_____ _____
Signature Date

As a representative of this study, I have explained the purpose, procedures, benefits, and the risks involved in this research study.

_____
Print Name of Person obtaining consent

_____ _____
Signature of Person obtaining consent Date

APPENDIX E.

Health History Questionnaire

HUMAN PERFORMANCE LABORATORY – THE UNIVERSITY OF TEXAS

IRB #: Subject ID: _____

Date of Birth (mm/dd/yy) _____ Age:

_____

MALE _____ FEMALE ____

Height _____ Weight _____

GENERAL HEALTH QUESTIONS

1. Are you taking any of the following medications on a regular basis? Y / N

(Psychotropics, Antihistamines, Asthma Meds, Aldomet, Clonidine,

Anti-Depressants, Anti-Anxiety Meds)

2. Are you taking any cardiovascular acting drugs? Y/N

3. Any over-the-counter meds? Y / N

If yes, explain:

4. Do you have any disability or impairment that affects physical performance? Y / N

5. Have you ever had any broken bones, surgery or injury to your lower extremities? Y / N

If yes, explain:

6. Have you had any significant medical problems within the last 10 years? Y / N

If yes, explain:

7. Do you have any drug and/or alcohol dependence? Y / N

If yes, explain:

8. Do you have any heart problems or coronary artery disease? Y / N

If yes, explain.

9. Do you have hypertension (high blood pressure)? Y / N

If yes, explain.

10. Do you have any lung or respiratory problems? Y / N

If yes, explain.

11. Do you smoke? Y / N

If yes, pattern.

12. Do you use alcohol? Y / N

If yes, pattern.

13. Do you use caffeine (cola, coffee, etc…)? Y / N

If yes, pattern.

14. Do you have any allergies that require medication? Y / N

If yes, explain.

15. Do you experience difficulty swallowing medications or vitamins? Y / N

If yes, explain.

16. Do you take any dietary supplements aimed at increasing your exercise performance? Y / N

If yes, what supplements so you normally take?

17. Have you been diagnosed with an obstructive disease of the gastrointestinal tract including but not limited to esophageal stricture, diverticulous, inflammatory bowel disease (IBD), peptic ulcer disease, Crohn's disease, ulcerative colitis, and previous gastro-esophageal surgery. Y / N

HAVE YOU EVER HAD ANY SIGNIFICANT SYMPTOMS ASSOCIATED WITH EXERCISE?

1. Easy fatigability or prolonged fatigue after exercise? Y / N

If yes, explain.

2. Persistent chest pain during and/or after exercise? Y / N

If yes, explain.

3. Fainting or loss of consciousness during exercise? Y / N

If yes, explain.

4. Palpitations (rapid, irregular, or skipped heartbeats) during exercise? Y / N

5. Have you ever been told to give up sports because of a health problem? Y / N

PHYSICAL TRAINING HISTORY

How many years have you been training?

_____

What type of physical training do you participate in?

_____

Describe in general, the type of training you have performed for each of your years of training.

1st

2nd

3rd

4th

5th

6th

7th

8th

others

What is your personal best race time (if more than one please list distance, time and type):

_____

_____

_____

_____

_____

_____

PLEASE GENERALLY DESCRIBE YOUR TRAINING PROGRAM DURING THE LAST 6 MONTHS

Type of training:

_____

_____

_____

_____

_____

Average time spent or work done (i.e.; distance):

_____

_____

_____

_____

_____

General Intensity:

_____

_____

_____

_____

_____

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