EFFECT OF SEvere-HEAVY exercise transitions ON measures of oxygen uptake AND blood lactate accumulation in moderate-wELL trained males

Miles Bartlett

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Approved By:
Anthony C. Hackney, PhD, DSc
Claudio L. Battaglini, PhD
J. Troy Blackburn, PhD, ATC
ABSTRACT

MILES BARTLETT: Effect of severe-heavy exercise transitions on measures of oxygen uptake and blood lactate accumulation in moderate-well trained males
(Under the direction of Anthony C. Hackney, Ph.D., D.Sc.)

Ten males completed an incremental maximal exercise test and three experimental trials (ET) to investigate whether or not sprinting at the start of competitive endurance events has a negative impact on physiological measures of performance. The control ET began with a square-wave transition to a heavy workload, whereas the sprint ETs began with a supramaximal sprint (110% VO_{2max}), lasting 15 or 60 seconds, before the workload was reduced to a heavy intensity. Oxygen uptake and blood lactate accumulation were significantly elevated (p<0.05) during the first few minutes of the sprint ETs; however, no significant differences were found between any of the ETs after 10 minutes of exercise (p<0.05). Thus, sprints lasting up to 60 seconds do not appear to have prolonged negative effects on physiological measures of performance during subsequent endurance exercise, and may benefit athletes by allowing them to gain positive tactical positioning.
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CHAPTER I

BASIS OF STUDY

Introduction

Although modern textbooks continue to describe the oxygen uptake-workload (VO₂-wkld) relationship as being linear (1, 2), numerous investigations have shown that the relationship becomes non-linear during exercise above the lactate threshold (3). Specifically, exercise above the lactate threshold (LT) requires more oxygen than would be predicted from moderate VO₂-wkld relationships. This increase in the VO₂-wkld relationship appears to be characterized by a delay in the attainment of steady state oxygen uptake. That is, during exercise above the LT, VO₂ continues to slowly rise beyond the traditional 2-3 minute window in which VO₂ normally plateaus, failing to reach steady state until 5-6 minutes into the exercise bout. Together, these findings have become known as the ‘slow component’ of oxygen uptake kinetics (VO₂SC).

While hyperventilation and oxidation of lactate by nonworking tissue certainly play a part in the VO₂SC, Poole et al. (4) calculated that approximately 86% of the VO₂SC measured from respiratory oxygen uptake can be attributed to working muscle. Despite the numerous hypotheses that have been presented to explain the VO₂SC intramuscularly, an exact mechanism that explains both the slow rise in VO₂ and the increased oxygen consumption for a given workload remains elusive. Among the most popular hypotheses are increased lactate and hydrogen ion (H⁺) production and increased motor unit recruitment.
Increases in blood lactate and $\text{H}^+$ have been shown to correlate well with the VO$_{2\text{SC}}$ both temporally and in magnitude (5, 6). Lactate as a causal factor of the VO$_{2\text{SC}}$, however, appears unlikely as exogenous lactate infusion does not appear to influence the VO$_{2\text{SC}}$ (7). Furthermore, changes in VO$_2$ and blood lactate do not always coincide with one another (3, 8, 9). For example, glycogen depletion has been shown to significantly reduce blood lactate accumulation during exercise despite VO$_2$ being significantly increased (9). Acidosis, on the other hand, may have a causal effect, as pre-exercise reduction of pH by ingestion of ammonium chloride (NH$_4$Cl) has been reported to significantly increase the magnitude of the VO$_{2\text{SC}}$ during heavy exercise (10). End exercise VO$_2$, however, does not appear to be altered following ingestion of NH$_4$Cl (10, 11), suggesting that VO$_2$ kinetics are altered by pre-exercise acidosis rather than the VO$_{2\text{SC}}$ magnitude. Furthermore, glycogen depletion protocols reduce exercise induced metabolic acidosis despite significantly increasing VO$_2$ (Osborne and Schneider, 2006); suggesting that even if decreased intramuscular pH has an effect on the VO$_{2\text{SC}}$, it is not solely responsible.

The VO$_{2\text{SC}}$ has also been postulated to be caused by the recruitment of Type-II muscle fibers, which, compared to type-I fibers, appear to be less aerobically efficient (12, 13). In line with this finding, studies measuring surface electromyographic (EMG) activity following glycogen depletion in type-I fibers have reported significantly increased surface EMG activity and enhancement of the VO$_{2\text{SC}}$ (9, 14). However, it is unknown whether the recruitment of type-II fibers is solely responsible for the VO$_{2\text{SC}}$, as recruitment of non-fatigue muscle fibers, regardless of their aerobic efficiency, would lead to an increase in whole body oxygen uptake (15, 16).
Recently, repeated bouts of exercise have become a popular method for studying the kinetics of oxygen uptake as well as the \( VO_{2SC} \). Overall, prior heavy exercise speeds up the \( VO_2 \) on-kinetics (fast and slow components) of a subsequent bout of heavy exercise (8, 17, 18), and the effects last up to approximately 45 minutes (19). Sahlin et al. (8) demonstrated heavy exercise, followed by short bouts of supra-maximal exercise, increased intramuscular lactate and \( H^+ \) content as well as the magnitude of the \( VO_{2SC} \) (i.e. overconsumption of oxygen) during a subsequent bout of heavy exercise. Conversely, the kinetic component of the \( VO_{2SC} \) (i.e. the delay in attainment of steady state oxygen uptake) was abolished, though this appeared to be due to an elevated baseline \( VO_2 \) at the onset of the second bout of heavy exercise.

While the study of oxygen uptake kinetics through repeated bouts of exercise may be relevant to sports such as soccer, basketball, or football, it is less applicable to endurance events in which a single continuous bout of heavy or severe exercise is performed. Also, although heavy “warm-up” exercise has been shown to improve the \( VO_2 \) response and time to exhaustion during subsequent severe exercise (20), many long distance endurance events, such as cross country races, involve severe exercise (sprints) at the start of the race in order to establish position for the remaining distance to be covered. Sprints may only last 15-20 seconds in smaller races (50-100 athletes), but can last 60 seconds or longer at the start of larger races (300+ athletes). Once position has been established, competitors are then challenged to pace themselves as closely as possible to their individual maximal lactate steady state (MLSS) in order to cover the distance as fast as possible without prematurely fatiguing.
Such sprints at the beginning of a race are conducted at workloads well exceeding maximal oxygen uptake, which significantly increase blood lactate and H+ accumulation as well as type-II fiber recruitment; all of which are correlated with augmenting the VO$_{2SC}$. If sprinting at the start of a race does induce a change in VO$_{2SC}$ magnitude, runners may end up racing at a higher percentage of their VO$_{2max}$ than expected; eventually leading to premature fatigue and poor performance. Many long distance athletes/coaches refer to this as “going out to fast”, and purposely may choose an alternate strategy of racing from behind in order to pick off fatiguing runners as the race progresses. This, however, may result in the athlete getting ‘stuck’ in the back, as the trail or pathway becomes clogged with other competitors. Therefore, from a practical or performance based standpoint, the purpose of this study was to determine if sprinting at the onset of a long distance race compromises mid-race (steady state) performance and/or perception of fatigue. From a strictly physiological perspective, the purpose of this study was to investigate the effect severe exercise has on measures of oxygen uptake and blood lactate accumulation during a subsequent bout of continuous heavy exercise.

**Significance of the Study**

The study was significant in that it allowed for the development and understanding of oxygen uptake kinetics to various exercise interventions. Previous studies investigating the role of varying work-work transitions on oxygen uptake kinetics have typically employed a passive resting protocol of some sort between exercise workloads. Though step-wise work-work transitions have been used before (21, 22), it appears this was the first study to attempt investigating VO$_2$ kinetics during a severe-heavy exercise transitions. The study introduced
a novel approach to investigating the VO$_{2SC}$ and VO$_2$ kinetics, and is directly applicable to athletes competing in endurance events that require appropriate pacing strategies to be successful.

**Research Questions**

1. Does a preceding bout of supra-maximal exercise affect steady state oxygen uptake during subsequent submaximal (heavy) exercise?
2. Does the duration of a preceding bout of supra-maximal exercise have an effect on steady state oxygen uptake during subsequent submaximal (heavy) exercise?
3. Does a preceding bout of supra-maximal exercise increase blood [lactate] during subsequent submaximal (heavy) exercise?
4. Does the duration of a preceding bout of supra-maximal exercise have an effect on the blood [lactate] during subsequent submaximal (heavy) exercise?

**Hypotheses**

1. The magnitude of the VO$_{2SC}$ during submaximal steady state exercise will be significantly higher during the sprint trials (S15 and S60) than during the CON trial.
2. The magnitude of the VO$_{2SC}$ during submaximal steady state exercise will be significantly higher during the S60 trial than during the S15 trial.
3. The blood lactate response will be significantly higher during the sprint trials (S15 and S60) than during the CON trial.
4. The blood lactate response will be significantly higher during the S60 trial than during the S15 trial.
5. There will be a significant positive correlation between the increase in blood [lactate] and the magnitude of the VO$_{2sc}$ when combining data from the CON, S15, and S60 trials together.

**Delimitations**

Lactate production during cycle ergometer exercise can be affected by several physiological variables including increases in core body temperature, high blood glucose levels and cadence speed (23, 24). To control for these factors, the study was performed in a thermo-neutral environment and subjects refrained from eating 2 hours pre-exercise. Although the subjects were not required to maintain a specific pre-determined cadence, they were instructed to maintain a consistent cadence between experimental trials and to remain seated throughout all testing sessions.

Intramuscular glycogen can also have a profound effect on the blood lactate response to exercise (23, 25). Therefore, subjects were asked to maintain a consistent diet rich in carbohydrates and to refrain from vigorous exercise in the 24 hours preceding any exercise session. A handout of carbohydrate rich food sources (i.e. pasta, rice, Gatorade etc.) was also provided to subjects, and they were encouraged to eat items on the list.

Lastly, hormonal fluctuations over the course of the menstrual cycle can effect lactate production during exercise in females (26, 27). Specifically, lactate production is reduced during the luteal phase. Therefore, women were excluded from the study to avoid confounding the results in blood measurements.
Limitations

The current study had several limitations which may have impacted the results and conclusions. The moderate VO₂-wkld relationships were calculated from one incremental exercise test per subject. Because oxygen uptake during exercise can fluctuate from day to day, the individual moderate VO₂-wkld relationship and calculated VO₂sc magnitude may have been affected. Subjects were not be directly monitored in the 24 hours prior to any session, and therefore were trusted to have adhered to pre-experimental dieting and exercise protocols. Additionally, the results are only generalizable to fit young adult males because females and unfit individuals were excluded.
CHAPTER II

REVIEW OF LITERATURE

Introduction

Oxygen uptake kinetics have been studied under a wide range of experimental conditions. Although current exercise physiology textbooks continue to describe the VO$_2$-wkld relationship as being linear up to VO$_{2\text{max}}$ (1, 2), it is well known that during exercise above the lactate threshold (LT), oxygen uptake (VO$_2$) rises to values greater than would be predicted from moderate VO$_2$-wkld relationships (3). The following review will 1) describe the basic fundamentals of oxygen uptake kinetics and how they are measured; 2) introduce the VO$_{2\text{SC}}$ and review previously done studies; and 3) discuss the major hypotheses explaining the VO$_{2\text{SC}}$.

Oxygen Uptake Kinetics

Oxygen is transported from the atmosphere to skeletal muscle through coordination of the respiratory (diaphragm and lungs) and cardiovascular (heart, blood vessels and blood) systems. Starting with contraction of the diaphragm, the lungs expand and pulmonary pressure declines. Consequently, air is drawn from an area of high pressure, the atmosphere, towards an area of low pressure, the alveoli of the lungs, resulting in inhalation. Upon reaching the alveoli, oxygen moves down its concentration gradient, crossing the alveolar membrane into the capillary space, and binds as a ligand to a hemoglobin complex located on
a red blood cell. At sea level, the barometric pressure is ~760 mmHg and the partial pressure of oxygen (PO₂) is ~159 mmHg; resulting in approximately 97.5% of hemoglobin becoming saturated with oxygen (2). The newly oxygenated red blood cells then return to the heart where they are pumped into the systemic circuit towards peripheral tissue, in this case, skeletal muscle. Upon reaching a capillary within skeletal muscle, oxygen diffusion once again moves from an area of high concentration, the blood or capillary space, to an area of low concentration within the muscle. At rest, oxygen demand by skeletal muscle is low, and venous blood hemoglobin remains approximately 75% saturated with oxygen. During exercise, however, oxygen demand by skeletal muscle increases dramatically as mitochondrial metabolism increases to maintain cellular ATP levels. Thus, oxygen diffusion into the skeletal muscle increases dramatically and venous oxy-hemoglobin saturation drops to approximately 5% (28).

Using the principles of the Fick equation (VO₂ = CO * a-v O₂diff), working muscle VO₂ can be calculated by measuring blood flow and arterial-venous O₂ difference (4, 28-31). However, this process is difficult and requires precise placement of catheters. Instead, most researchers measure VO₂ during exercise through pulmonary gas exchange, and subsequently describe VO₂ kinetics in a 3-phase progression (32), as depicted in Figure 1.

Phase one, also known as the time delay, is defined as the time between the start of exercise and the sudden abrupt increase in VO₂ which occurs due to the delayed return of deoxygenated blood from working limbs back to the lungs for gas exchange. Phase two describes the rapid increase in VO₂ and is defined as the time between the end of phase one and the attainment of an oxygen uptake steady state (OUSS). Phase three is defined as the attainment of an OUSS which is usually determined within the first 2-3 minutes of exercise.
It should be noted that gradual rises in VO$_2$ during prolonged exercise (i.e. the O$_2$-drift) due to increases in core temperature or decreases in blood plasma volume do not indicate that phase three has not been achieved.

**VO$_2$ kinetics during moderate, heavy and severe exercise**

Understanding what constitutes moderate, heavy or severe exercise is as crucial to understanding VO$_2$ kinetics as understanding the three phases. In short, exercise intensity is based off of blood lactate responses (3). Specifically, *moderate* exercise is defined as exercise below the lactate threshold (LT), or workloads in which there is no blood lactate accumulation. *Heavy* exercise is defined as workloads equal to the LT (first workload in which blood lactate reaches 2.0 mmol/L) up to the individual’s maximal lactate steady state (MLSS – greatest workload that can be maintained in which blood lactate plateaus). *Severe* exercise is defined as workloads above the MLSS. Keep in mind, the exercise intensities are defined based on physiological thresholds. As will be discussed later, endurance training can improve the workload corresponding to either the LT or the MLSS and thereby influence VO$_2$ kinetics and the VO$_{2sc}$ for a given workload.
The previously described three phase model of oxygen uptake kinetics is based off the VO$_2$ response to moderate intensity exercise and therefore follows the pattern described above. In summary, for moderate intensity exercise, the delay in VO$_2$ response at the onset of exercise (phase one) lasts 10-15 seconds and is immediately followed by a 2-3 minute period (phase two) in which VO$_2$ rapidly rises to a steady state value (phase three).

During heavy exercise, phase one is unaltered. However, instead of reaching an OUSS within 3 minutes of exercise onset, a “slow component” (VO$_{2SC}$) develops during phase two in which VO$_2$ continues to rise until plateauing around 6 minutes into exercise. In addition to being delayed, the OUSS observed during heavy exercise is greater than that which would be predicted from moderate VO$_2$-wkld relationships. In summary, phase two is broken into two components; a fast component in which VO$_2$ rises rapidly in the first 2-3 minutes of exercise, and a slow component in which VO$_2$ continues to rise for another 2-3 minutes. As depicted in Figure 2, phase three is eventually attained but is delayed until 5-6 minutes into exercise.

Figure 2 – Graphical depiction of the three phase model of VO$_2$ on-kinetics for the moderate, heavy, and severe exercise intensities. Redrawn from Xu and Rhodes (32). The development of a VO$_{2SC}$ during heavy and severe exercise is depicted by the shaded areas underneath each respective curve. Notice that during heavy exercise VO$_2$ eventually plateaus, but steady state is delayed 5-6 minutes into exercise.
During severe exercise, once again, phase one is typically unaltered and phase two follows the same pattern described for heavy exercise. There is a 2-3 minute fast component in which VO$_2$ rapidly rises followed by a slow component in which VO$_2$ continues to rise. During severe exercise, however, phase three is typically not attained. Instead, VO$_2$ continuously rises towards VO$_{2\text{max}}$ until the subject becomes exhausted. Highly trained endurance athletes that can run at severe workloads for extended periods of time, however, have been shown to attain a ‘plateau’ in their severe exercise VO$_2$; corresponding to either their individual VO$_{2\text{max}}$ or an approaching asymptote (33).

**Measuring VO$_2$ kinetics and the VO$_2$ slow component**

The kinetics of fast and slow components of oxygen uptake are often calculated and measured to quantify the effect of a given experimental condition. Figure 3 illustrates the variables used to measure VO$_2$ kinetics including time delay (TD), amplitude (Amp), and time constant (34).

![Figure 3](image)

Time delay, which is synonymous with phase one, represents the time between the start of exercise and the abrupt increase in VO$_2$, indicating a return of deoxygenated blood to
the lungs. Amplitude is used to describe the slope of the increase in VO$_2$ during phase two. Although Amp can be calculated linearly, the increase in VO$_2$ becomes non-linear as VO$_2$ approaches steady state. The time constant ($\tau$) represents the time required to attain 63% of the total fast component VO$_2$ response (slow component is measured separately), and allows the linear and non-linear components of the VO$_2$ fast component to be separated. Thus, VO$_2$ at time ($t$) is typically calculated as:

$$VO_2(t) = VO_{2\text{baseline}} + \text{Amp} \times [1 - e^{-(t-TD)/\tau}]$$

where $e$ represents the natural log, and Amp, TD, and $\tau$ represent the amplitude, time delay, and time constant respectively (34, 35).

The kinetics of the slow component are measured using either minute two or three for the TD and calculating a new Amp and $\tau$ for the VO$_{2\text{SC}}$ from that point forward according to the equation:

$$VO_2(t) = VO_{2\text{baseline}} + \text{Amp}_f \times [1 - e^{-(t-TD_f)/\tau_f}] + \text{Amp}_s \times [1 - e^{-(t-TD_s)/\tau_s}]$$

where subscripts f and s denote the aforementioned variables as constituents of the fast and slow components respectively (22, 34).

Not to be confused with the *kinetics* of the slow component, the *magnitude* of the VO$_{2\text{SC}}$ describes the portion of a VO$_2$ response during heavy and severe exercise that is greater than the VO$_2$ that would be ‘predicted’ by moderate VO$_2$-wld relationships (3). This is denoted in Figure 2 by the grey area beneath the VO$_2$ curves of the heavy and severe
exercise intensities. Although the only true method for measuring the magnitude of the $\text{VO}_2\text{SC}$ is to compare a $\text{VO}_2$ response during heavy or severe exercise to the expected $\text{VO}_2$ response from a moderate $\text{VO}_2$-wkld relationship, several shortcuts have been developed. As described previously, $\text{VO}_2$ during moderate exercise typically plateaus within 2-3 minutes, whereas $\text{VO}_2$ during heavy exercise plateaus around 5-6 minutes. Thus, the magnitude of the $\text{VO}_2\text{SC}$ has been quantified as the $\text{VO}_2$ measured at minute 6 the end of exercise minus the $\text{VO}_2$ measured at minute 3 (10, 36, 37). In studies where subjects exercise for longer periods of time, the magnitude of the $\text{VO}_2\text{SC}$ has also be calculated as the $\text{VO}_2$ at the end of exercise minus the $\text{VO}_2$ at minute 3 (8, 9, 38). As will be demonstrated later, however, the use of shortcuts to measure or quantify the magnitude of the $\text{VO}_2\text{SC}$ can have drastic consequences on calculations and consequently the interpretation of experimental results.

II. Investigations of $\text{VO}_2$ kinetics and the $\text{VO}_2\text{SC}$

Oxygen uptake kinetics and the $\text{VO}_2\text{SC}$ have been investigated under several experimental conditions. The effects of prior bouts of exercise, muscle glycogen depletion, and training interventions are among the most popular. Variances in exercise protocols and methods of determining $\text{VO}_2\text{SC}$, however, often make comparing results difficult.

Effects of prior or ‘priming’ exercise

Several studies have examined the effects of prior heavy exercise on the $\text{VO}_2$ kinetics during a subsequent bout of heavy exercise. While protocols vary slightly from study to study, exercise is usually conducted at approximately 70% $\text{VO}_2\text{max}$ and bouts of exercise are typically separated by 2-10 minutes. In general, prior heavy exercise has been shown to
improve VO\textsubscript{2} on-kinetics during a subsequent bout of exercise by increasing/decreasing the Amp and \( \tau \) of the fast component respectively, and eliminating the kinetic response of the VO\textsubscript{2SC} (17, 18).

Burnley et al. (19) investigated the time required for the restoration of normal VO\textsubscript{2} on-kinetics following heavy exercise. Nine healthy male subjects, consisting of competitive cyclists and students familiar with the testing procedures, participated in the study. Subjects completed several trials of two 6-minute bouts of heavy exercise corresponding to 70\% of the difference between gas exchange threshold and VO\textsubscript{2peak}. The duration of the rest between bouts of exercise were altered from trial to trial and lasted either 10, 20, 30, 45, or 60 minutes. Oxygen uptake was measured breath by breath and the kinetics of both the fast and slow components of VO\textsubscript{2} were measured and calculated. Results of the study showed that the Amp of the slow and fast components took 45 and 60 minutes to return to normal values respectively, whereas the \( \tau \) and end-exercise VO\textsubscript{2} (VO\textsubscript{2SC} magnitude) did not appear to be affected at all. The authors also reported a strong relationship between the recovery time of blood [lactate] and the decline in fast component Amp enhancement. They did note, however, that the relationship was obscure and concluded that blood [lactate] was more of a proxy rather than causative variable.

Sahlin et al. (8) used repeated bouts of heavy exercise to investigate the role of intramuscular [lactate] and [H\textsuperscript{+}] on the kinetics of the VO\textsubscript{2SC}. Nine moderately well-trained male subjects performed two 10 minute bouts of heavy exercise corresponding to 75\% VO\textsubscript{2max} on a cycle ergometer. In order to assess the effect of pH and lactate on the VO\textsubscript{2SC}, subjects completed supra-maximal bouts of exercise between periods of heavy exercise. Two minutes after completion of the first 10 minute period of heavy exercise, subjects
performed three bouts of exercise at 110% VO$_{2\text{max}}$ separated by 2 minutes of rest. The first two supra-maximal repeats were performed for two minutes, the third to volitional fatigue. Upon termination of the third repeat, subjects rested passively for 3 minutes followed by 2 minutes of light cycling at 40W and the second 10 minute period of heavy exercise. Gas exchange was measured breath by breath (later averaged in 5-second intervals), muscle biopsies were obtained from the vastus lateralis muscle, and capillary blood samples were taken from a pre-warmed finger. The VO$_2$ during the first 10 minute period of heavy exercise tended to rise throughout the exercise bout and was significantly higher at minute 10 than at minute 3. Both blood/muscle [lactate] and [H$^+$] were significantly increased 3 minutes into exercise but then stabilized. Conversely, the VO$_2$ during the second bout of heavy exercise was not significantly different between the 3$^{\text{rd}}$ and 10$^{\text{th}}$ minute but blood/muscle [lactate] and [H$^+$] were significantly lower after 10 minutes of exercise versus 3. The significant change in VO$_2$ without the significant change in blood/muscle [H$^+$] during the first bout of heavy exercise, followed by the lack of a change in VO$_2$ despite decreases in blood/muscle [H$^+$] in the second bout of exercise, led the authors to conclude that metabolic acidosis is not the sole cause of the VO$_{2\text{SC}}$.

Jones et al. (20) investigated the role of prior heavy exercise on the VO$_2$ on-kinetics of subsequent severe exercise to exhaustion. Seven recreationally active male subjects completed supra-maximal cycle ergometer exercise corresponding to 100%, 110%, or 120% VO$_{2\text{peak}}$ to exhaustion. The exercise intensities were repeated twice each, once with and once without prior heavy exercise. The results of the study indicated that prior heavy exercise positively impacted the VO$_2$ on-kinetics during subsequent severe exercise at 110% and
120% VO$_{2peak}$. Furthermore, time to exhaustion was significantly greater following prior heavy exercise across all severe intensities.

Draper et al. (39) followed this study up by comparing the effect of prior moderate and heavy exercise on the VO$_2$ on-kinetics of a subsequent bout of severe exercise. Results indicated that other than TD (significantly faster following heavy exercise) prior heavy or moderate exercise had a similar influence on the VO$_2$ on-kinetics during subsequent severe exercise. This study, however, failed to use a control trial (single square wave transition to severe intensity exercise) to compare the oxygen uptake kinetics of a severe bout of exercise following prior moderate or heavy exercise. Therefore, while heavy exercise certainly appears to improve subsequent severe exercise performance (20) the effect of moderate intensity exercise on subsequent bouts of severe exercise remains inconclusive.

In summary, prior exercise appears to enhance VO$_2$ on-kinetics during a subsequent bout of exercise mostly by improving or reducing the $\tau$ or Amp of the fast and slow components respectively. Furthermore, prior exercise appears to improve subsequent exercise performance, though further research is necessary before accurate warm-up protocols to enhance exercise performance can be recommended to coaches/athletes.

**Effect of muscle glycogen depletion**

Increased type-II fiber recruitment remains a strong candidate as the cause of the VO$_{2SC}$. As exercise intensity increases, or as type-I fibers begin to fatigue, the increased recruitment of type-II fibers to maintain power output is thought to increase oxygen demand due to their inefficient use of oxygen. To simulate this experimentally, several studies have
used type-I muscle fiber glycogen reduction to simulate muscle fatigue and increase type-II fiber recruitment (9, 40).

Krustrup et al. (14) investigated how selectively depleting glycogen stores within slow twitch fibers effects steady state and end exercise VO$_2$ during moderate exercise in twelve recreationally trained individuals. Subjects cycled for 20 minutes at approximately 50% of their VO$_{2\text{max}}$ on two separate occasions, once following a glycogen reduction protocol (3 hours of cycling at approximately 40% VO$_{2\text{max}}$ followed by an overnight fast) and once under normal glycogen storage conditions. Muscle biopsies showed that the glycogen reduction protocol was successful in depleting glycogen stores within slow twitch fibers while leaving fast twitch fibers relatively unaffected. Blood lactate accumulation and respiratory exchange ratio were significantly reduced during the glycogen reduction trial as compared with the control. Furthermore, VO$_2$ plateaued within 3 minutes during the control trial but was significantly elevated and steadily rose throughout the glycogen reduction trial. Although EMG measurements were not collected during this study, the authors concluded that slow-twitch fiber glycogen reduction altered muscle recruitment patterns and increased pulmonary oxygen uptake due to an increased recruitment of less aerobically efficient fast twitch muscle fibers.

In a similar study, Carter et al. (40) investigated the effect of both type-I and type-II fiber glycogen depletion on oxygen uptake kinetics during moderate and heavy exercise in recreationally active individuals. The glycogen depletion protocols consisted of 3 hours of cycle ergometer exercise at 30% VO$_{2\text{max}}$ and 10 one minute bouts of exercise at 120% VO$_{2\text{max}}$ with five minutes of rest for type-I and type-II fibers respectively. Muscle biopsies taken before and after the two protocols showed the exercises were effective in
independently depleting type-I and type-II fibers. Following glycogen reduction protocols, subjects rested passively for an hour to allow body temperature, blood [lactate], and heart rate to return to resting values. The experimental protocol consisted of a series of 6 minute square wave exercise transitions, 2 moderate intensity bouts and one heavy bout, separated by 6 minutes of rest. Oxygen uptake was measured breath by breath and the experimental protocol was completed alone (control) and following the two glycogen reduction protocols for a total of three trials. Results of the study showed that for moderate intensity exercise, glycogen reduction, regardless of muscle fiber type, did not elicit a significant change in VO$_2$ kinetics. During heavy exercise, however, glycogen reduction within type-II fibers did significantly increase the amplitude of the fast component; delay the onset of the VO$_{2SC}$, and reduce blood lactate accumulation. Glycogen reduction in type-I fibers, on the other hand, did not improve VO$_2$ on-kinetics during heavy exercise, and in contrast to the study of Krustrup et al. (14) did not alter end exercise VO$_2$ during heavy or moderate exercise. Carter et al. (40) concluded that increased type-II fiber recruitment plays a role in the kinetic response of the VO$_{2SC}$, but not the magnitude of the VO$_{2SC}$; as end exercise VO$_2$ was not significantly different between any of the trials.

To take a more direct look at muscle recruitment patterns during exercise, Osborne and Schneider (9) measured surface EMG activity in the vastus lateralis and vastus medialis during cycle ergometer exercise in eight recreational cyclists. Subjects performed two 8 minute bouts of heavy cycle ergometer exercise, once under normal conditions and once following glycogen reduction. Results showed VO$_2$ was significantly elevated throughout exercise during the glycogen reduction trial compared to the control trial. Furthermore, consistent with previous studies, glycogen reduction decreased blood lactate and H$^+$
accumulation (14, 40), suggesting the relationship between the VO$_{2SC}$ and lactate or H$^+$ is coincidental rather than cause and effect. As was expected, surface EMG activity in the vastus lateralis and vastus medialis significantly increased both throughout a given exercise bout (minute 3 to minute 8) as well as between trials (control and glycogen reduction). Interestingly, despite end exercise VO$_2$ being significantly elevated following glycogen reduction, the magnitude of the VO$_{2SC}$ was not calculated to be significantly different between the control and glycogen reduction trials. As was mentioned earlier, shortcuts have been developed to make measuring the VO$_{2SC}$ easier. Osborne and Schneider calculated the VO$_{2SC}$ as the VO$_2$ at minute 8 minus the VO$_2$ at minute 3. The VO$_2$ at minute 3 during the glycogen reduction trial, however, was already significantly elevated compared to the control trial. Thus, by not taking into account the faster kinetic response of VO$_2$ during the glycogen reduction trial, Osborne and Schneider mistakenly concluded that glycogen reduction does not influence the magnitude of the VO$_{2SC}$. Put another way, if end exercise VO$_2$ is elevated, then it stands reason to believe that the magnitude of the VO$_{2SC}$ will be elevated as well, exemplifying the pitfalls of the shortcuts that have been developed to study the VO$_{2SC}$.

In summary, glycogen reduction alters muscle recruitment patterns during exercise (9) and appears to enhance the magnitude of the VO$_{2SC}$ (9, 14), though not always (40). Glycogen reduction, however, does consistently reduce blood lactate accumulation and exercise induced acidosis; suggesting lactate/H$^+$ accumulation may be, at best, only partially responsible for the magnitude of the VO$_{2SC}$. 
Effect of endurance training

The effects of endurance training on submaximal exercise performance have been well documented. While the effects of various endurance training methods differ slightly from program to program, the general effect is an improvement in B-oxidative capacity, a reduction in lactate and H+ accumulation for a given workload, and increased maximal oxygen uptake (41-47). Likewise, endurance training has been shown to improve VO2 kinetics and reduce the magnitude of the VO2SC for a given absolute workload (38, 48-50).

Casaburi et al. (38) investigated the effect of 8 weeks of endurance training on changes in blood lactate accumulation, circulating catecholamine levels, and ventilatory drift in 10 untrained college students. Consistent with previous studies, blood lactate, circulating catecholamine levels, and ventilatory drift were all significantly reduced for a given absolute workload (51, 52). Oxygen uptake for a given workload above the pre-training lactate threshold was also reduced, indicating a reduction in the VO2SC magnitude. Moreover, Casaburi et al. (38) reported the reductions in blood lactate and ventilatory drift for a given workload were significantly correlated with the reduction in the magnitude of the VO2SC. Changes in circulating catecholamine levels, however, were not significantly correlated with the reduction in VO2SC magnitude. Consequently, the authors concluded that attenuation of the VO2SC following endurance training is attributable to factors that reduce blood lactate accumulation and ventilatory drift.

Womack et al. (48) investigated the reduction of the VO2SC throughout the course of a 6 week endurance training program. Seven untrained male subjects completed eight 20 minute trials of heavy cycle ergometer exercise; one pre-training trial and one trial at the end of each week of training. Adaptations in exercise blood lactate and minute ventilation were
measured during each of the exercise trails and related to changes in the magnitude of the VO\textsubscript{2SC}. While the initial time course of adaptation of the VO\textsubscript{2SC} magnitude was similar to that of blood lactate and minute ventilation; the adaptation of the VO\textsubscript{2SC} magnitude was essentially complete by week 2, whereas blood lactate and minute ventilation continued to improve throughout the study. A correlation analysis demonstrated neither improvements in blood lactate or minute ventilation were significantly correlated with the magnitude of the VO\textsubscript{2SC} temporally. Furthermore, an additional trial was completed at the end of the study following epinephrine infusion. Epinephrine infusion resulted in significant increases in minute ventilation and blood lactate, but no change in the magnitude of the VO\textsubscript{2SC}. In contrast to the study of Casaburi et al. (38), the results of Womack et al. (48) suggested the training induced adaptations to blood lactate and minute ventilation were coincidental, rather than causally, linked to improvements observed for the magnitude of the VO\textsubscript{2SC}.

Early studies examining the effect of endurance training on measures of the VO\textsubscript{2SC} employed continuous exercise training at a heavy intensity for prolonged periods of time (38, 48). Recently however, high intensity interval training has gained popularity as an endurance training protocol for both its quick efficiency in eliciting enzymatic adaptations as well as its ability to improve central determinants of oxygen delivery (53, 54). For example, Duffield et al. (49) demonstrated high intensity interval training can improve VO\textsubscript{2peak}, end exercise VO\textsubscript{2} and accumulated oxygen deficit during severe exercise conducted at the same absolute workload.

Berger et al. (55) examined the effects of 6 weeks of continuous or high intensity interval training on VO\textsubscript{2} kinetics during moderate and severe exercise in previously untrained males and females. Subjects partaking in the continuous training protocol exercised at 60%
VO$_{2peak}$ for 30 minutes, whereas subjects in the high intensity interval training group completed 20 1-minute bouts of exercise at 90% VO$_{2peak}$ separated by one minute of rest. Subjects trained three days per week for the first two weeks, and then four days per week for the remainder of the study. Results indicated that VO$_2$ kinetics were significantly improved during both moderate and severe exercise for both training groups, including a significant reduction of the Amp of the VO$_{2SC}$. No significant differences, however, were found when comparing the improvements made between groups. Similar results were obtained when increasing the duration of continuous training to 90-120 minutes per session and intensity of the intervals to 120% VO$_{2max}$ (50). Collectively, these results suggest that continuous and high intensity interval training programs elicit equal improvements in VO$_2$ kinetics during exercise.

III. Hypothetical mechanisms explaining the VO$_2$ slow component

To date, no mechanism has been able to fully explain the dynamics of the VO$_{2SC}$. Early hypotheses revolved around the increased oxygen cost of processes away from working muscle such as hyperventilation and hepatic lactate metabolism. While these factors undeniably account for at least a part of the VO$_{2SC}$, calculations of their maximal contribution to the VO$_{2SC}$ magnitude fall far short of the total excess oxygen uptake response. Furthermore, Poole et al. (4) demonstrated that approximately 86% of the VO$_{2SC}$ can be attributed to the working muscle fibers themselves. More recent hypotheses put forth to explain the VO$_{2SC}$ intramuscularly have revolved metabolic acidosis and/or changes in motor unit recruitment.
Several studies examining the relationship between the surface electromyographical activity of working muscle and the development of the VO$_{2SC}$ during exercise have reported a significant relationship between the two (9, 14, 56, 57). Consequently, changes in motor unit recruitment have been postulated to induce the VO$_{2SC}$ in one of two ways. First, the increased recruitment of type-II fibers may play a role due to their less efficient use of oxygen, or lower P/O ratio. In this way, the VO$_{2SC}$ would be characterized by a decline in mean P/O ratio of the working muscle as a whole. As exercise intensity increases, a higher percentage of type-II fibers become recruited to generate the required force, causing the VO$_2$-workload relationship of working muscle as a whole to increase; leading inversely to an increase in the magnitude of the VO$_{2SC}$. Consistent with this hypothesis, Coyle et al. (12) reported a significant correlation between the gross cycling efficiency and percentage of type-I fibers in the vastus lateralis of trained male cyclists. In a more direct investigation, Jackman and Willis (13) demonstrated that mitochondria isolated from type-I fibers had twice the cytochrome-c reductase capacity of type-II fiber mitochondria, despite similar pyruvate and malate oxidase profiles.

Second, as muscles controlled by lower-order motor units begin to fatigue, the brain increases motor unit recruitment in order to maintain force production and power output. In a study by Zoladz et al. (15), a pseudo-VO$_{2SC}$ was demonstrated in isolated dog gastrocnemius muscle. Electrical stimulation was set to recruit all fibers within the innervated muscle and frequency was set to elicit a workload corresponding to 60-70% of the muscle’s VO$_2$peak. During the contraction period, force declined while oxygen uptake was maintained, indicating an increase in the VO$_2$-workload relationship. The authors concluded that because the VO$_2$-workload relationship increased without an increase in recruitment
(maximal recruitment was achieved throughout the experiment), the VO<sub>2SC</sub> can occur in the absence of increased motor unit recruitment. This point was countered by Borrani et al. (16), who pointed out that the “real” slow component was an increase in oxygen uptake despite constant power output, not the reverse. If the gastrocnemius muscle could have been further recruited, power output could have been maintained and VO<sub>2</sub> would have increased due to the increased oxygen demand of the newly recruited muscles. By this mechanism, the recruitment of type-II fibers is not mandatory for the manifestation of the VO<sub>2SC</sub> per se. Rather, the increased recruitment of non-fatigued muscle fibers, regardless of fiber type, acts as the source of the extra oxygen consumption, which slowly develops at the onset of exercise as lower-order muscles fatigue (16).

Taking an alternative view, some researchers have postulated increased intramuscular [H<sup>+</sup>] causes the VO<sub>2SC</sub>. Though it is now generally accepted that lactate and H<sup>+</sup> production can occur in the presence of adequate oxygen supply, some researchers maintain that metabolic acidosis indicates intramuscular hypoxia (58, 59). Stringer et al. (60) suggested the VO<sub>2SC</sub> may be caused by the increased dissociation of oxygen from hemoglobin due to metabolic acidosis. Synonymous with breathing a hyperoxic gas mixture, the augmented oxyhemoglobin dissociation would increase mitochondrial oxygen availability and ATP turnover, ultimately attenuating the intramuscular hypoxia. While this hypothesis provides an explanation for the slow rise in VO<sub>2</sub> beyond the traditional three minute plateau, it fails to explain the increase, or non-linearity, of the VO<sub>2</sub>-workload relationship for workloads above the LT (61). Furthermore, if intramuscular hypoxia is attenuated following increased oxyhemoglobin dissociation, then H<sup>+</sup> production should decline, eliminating the source of the
acidosis that was originally necessary to increase oxyhemoglobin dissociation in the first place.

The rate of working muscle oxygen uptake is inversely proportional to the decline in intramuscular creatine phosphate (PCr). Capelli et al. (62) suggested metabolic acidosis causes the VO$_{2SC}$ by shifting the creatine kinase reaction to the right, enhancing the decline in intramuscular [PCr] and leading to increased mitochondrial respiration. Others have argued that increased breakdown of PCr would increase the ATP/ADP ratio, which inhibits oxidative phosphorylation (61). It should be noted, however, that as long as ATP turnover is being maintained, the relative contribution of the increased PCr breakdown to the ATP/ADP ratio should be minimal. Interestingly, in line with the hypothesis of Capelli et al. (62), Rossiter et al. (63) demonstrated that when the VO$_{2SC}$ is elicited, PCr depletion remains inversely proportional and continues to decline until oxygen uptake plateaus; almost like a PCr-slow component. Whether this is due to a cause and effect relationship or simply coincidence, however, requires further investigation.

Increased [H$^+$] has also been postulated to affect mitochondrial ATP production by reducing the proton motive force necessary to synthesize ATP via oxidative phosphorylation. The proton leak hypothesis predicts that as intramuscular acidosis develops the flow of protons into the mitochondrial matrix through mechanisms other than ATP Synthase of the decreases the proton motive force, thereby increasing the volume of oxygen required to form ATP through oxidative phosphorylation. While this hypothesis provides an explanation for both the magnitude and kinetic response of the VO$_{2SC}$, the hypothesis is generally not supported by studies measuring state 3 respiration in isolated mitochondria suspended in low pH solutions (64-66). Conversely, Gollnick et al. (67) reported a progressive reduction of
intramuscular pH and respiratory capacity of mitochondria isolated from equine skeletal muscle following repeated bouts of heavy exercise. Although a cause and effect relationship could not be determined between mitochondrial function and intramuscular pH, the results suggest there may be an underlying relationship that is elicited in vivo but not in vitro. That is, while manipulating the pH of the solution mitochondria are suspended in is logical, it potentially eliminates other factors within the muscle fiber that can influence oxidative phosphorylation and mitochondrial functioning. Therefore, intramuscular acidosis may have an impact on factors that regulate oxidative phosphorylation in vivo that are removed or diminished when studying mitochondria in vitro.

One such factor that could diminish the proton motive force during exercise in vivo is uncoupling protein 3 (UCP-3). The UCP family reduces the P/O ratio by allowing H⁺s to flow back into the mitochondria without passing through ATP synthase, thereby reducing the proton motive force and compromising oxidative phosphorylation. While it was once thought the uncoupling proteins were responsible for non-shivering thermogenesis, recent evidence has suggested UCP-3 plays a role in metabolism (68) and may play a role in protecting skeletal muscle from oxidative damage by superoxide anions (69-71).

Superoxide, a product of molecular oxygen interacting with the electron transport chain of mitochondria, can cause considerable damage to cellular membranes and directly stimulates UCP-3 activity (72). In general, the rate of superoxide production is directly proportional to the pO2 of the tissue or solution being studied, and inversely proportional to the activity of the electron transport chain (73, 74). Thus, superoxide production is greater at rest than during exercise when intramuscular pO2 is at its highest and ETC is low. However, this does not preclude the possibility that superoxide anions may be generated during
exercise as a result of exercise induced metabolic acidosis. As described by Mitchell’s Chemiosmotic Theory (75), electron shuttling by the ETC is coupled to H+ pumping across the inner mitochondrial membrane. When the [H+] outside the mitochondrial matrix is too high, H+ pumping, and therefore electron shuttling, is inhibited; increasing the potential for superoxide production. Therefore, superoxide anions may be formed during exercise when intramuscular pH declines, such as during an intense sprint at the start of a race, thereby activating UCP-3 and reducing the efficiency of oxidative phosphorylation and aerobic metabolism.

The mechanism behind an UCP-3 induced VO$_{2SC}$ would mimic the VO$_{2SC}$ mechanism of proton leak. As intramuscular pH declines ETC activity decreased, causing an increase in the generation of superoxide anions. UCP-3 then becomes activated, allowing H*’s to flow back into the mitochondria without passing through ATP synthase, thus allowing the ETC to be active again and reducing superoxide generation. This comes at the expense of the proton motive force, however, which reduces the proton motive force and compromises both mitochondrial ATP production and power output by the working muscle fiber. Although oxygen uptake by the fatiguing muscle fiber would be maintained, the decline in force production would necessitate the recruitment of additional muscle fibers to maintain a given power output. The newly recruited muscle fibers would increase their oxygen consumption to maintain ATP turnover, leading to an increase in total VO$_2$ by the working muscle as a whole, and thus an increase in the pulmonary VO$_{2SC}$ (16). Therefore, the source of the VO$_{2SC}$, that is the source of the uncoupling of the mitochondria P/O ratio, is fatigued muscle fibers, whereas the cause of the VO$_{2SC}$ is the increased recruitment of additional motor units (15, 16).
From an evolutionary perspective, however, the decline in the proton motive force following UCP-3 activation would seem counter-intuitive survival. Thus, cells have developed a second mechanism by which they can protect against superoxide production. Superoxide dismutase (SOD), an enzyme that can be found in both the mitochondrial matrix and cytosol, reduces superoxide anions to the less potent H$_2$O$_2$; which is then further reduced to O$_2$ and H$_2$O by glutathione peroxidase (Nelson and Cox, Principle of Biochem). Although SOD eliminates superoxide anions without compromising the proton motive force, glutathione peroxidase (GP) relies on NADPH as a co-factor. Sustained elimination of superoxide by SOD and GP is therefore exhaustible. When the rate of superoxide production exceeds the rate NADPH regeneration by the nicotinamide nucleotide transhydrogenase (mitochondrial) or pentose phosphate (cytosol) pathways, SOD and GP cannot sufficiently protect the cell from superoxide anions and UCP-3 becomes activated. Thus, UCP-3 activation appears to be a last resort for cells experiencing high rates or volumes of oxidative stress, and favors the immediate survival of the cell or organism over long term energy storage/preservation.

Within humans, studies measuring UCP-3 content and/or mRNA following exercise in humans have been correlational in nature. That is, a cause and effect relationship between the VO$_{2\text{SC}}$ and UCP-3 activity has not yet been made. It is likely that this will persist until pharmacological innovations allow researchers to safely manipulate intramuscular UCP-3 or SOD activities within exercising human muscles. None the less, the correlational studies reported thus far have suggested a positive relationship. Russell et al. (77) demonstrated a strong correlation between the increase in VO$_{2\text{SC}}$ magnitude and UCP-3 mRNA following heavy exercise in trained and untrained males. Although total UCP-3 content and mRNA
expression following heavy exercise only appear to be moderately reduced as a result of endurance training, their expression as a function of mitochondrial volume are significantly decreased (78-81), suggesting tighter coupling despite increased potential for superoxide production.

The most direct evidence suggesting UCP-3 may play a role in the VO2Sc has been demonstrated by studies on rats or gene-knockout (KO) mice. Vidal-Puig (82) reported significantly increased superoxide production in UCP-3 knockout (KO) mice. However, exercise capacity and VO2 measured during exercise were not significantly different between normal and UCP-3 KO mice, suggesting no relationship at all between UCP-3 and the VO2Sc. This may, however, be explained by the exercise protocol utilized (2-hour treadmill run to exhaustion). As was described earlier, the VO2Sc is not synonymous the gradual rise in VO2 observed during prolonged exercise, otherwise known as the O2-drift (3). Therefore, the similarity in exercise VO2 between the control and UCP-3 KO mice may have due to the exercise intensity not being heavy enough to elicit a true VO2Sc response. Specifically, superoxide production within the control rats may have been matched by superoxide clearance through SOD and GP; such that UCP-3 activation was not necessary. Consistent with this notion, SOD KO mice do display significantly lower exercise capacities/VO2-wkld relationships compared to controls (83), supporting the concept that UCP-3 is activated only when superoxide clearance by SOD is insufficient; thereby acting as a last resort against severe oxidative damage. Unfortunately, Kinugawa et al. (83) did not measure UCP-3 activity in the SOD-KO mice, so conjecture about UCP-3 having a direct effect on exercise capacity and VO2 remains speculative. None the less, in contrast to SOD-KO mice, mice and rats display increased skeletal muscle SOD contents following endurance training (69, 84-
an adaptation that appears to directly improve mitochondrial coupling during exposure to oxidative stress (86). And unlike human studies, endurance training does significantly reduce UCP-3 content in rat skeletal muscle (69).

In conclusion, the mechanism behind the VO\textsubscript{2SC} appears to revolve around metabolic acidosis, increased motor unit recruitment, or a combination of the two. Although metabolic acidosis should theoretically increase the electrical potential of the proton motive force, thereby increasing superoxide production and activating UCP-3, \textit{in vitro} studies have reported that cytosolic pH does effect mitochondrial respiration until pH is dropped to levels below those typically seen during exercise. None the less, it is possible mitochondrial isolation alters or removes physiological dynamics that facilitate or increase mitochondrial uncoupling, may therefore occur more readily \textit{in vivo} than \textit{in vitro}. That is, perhaps the electrical potential of the proton motive force needs to be viewed from a physiochemical perspective, rather than the classical acid-base point of view. In this way, changes in metabolic regulation could lead to the accumulation of metabolites that alter the electrochemistry of the cytosol and mitochondrial matrix and lead to mitochondrial uncoupling. It is likely that novel hypotheses will be necessary to stimulate new research and further our understanding of oxygen uptake kinetics and the VO\textsubscript{2SC} during exercise.
CHAPTER III
METHODOLOGY

Subjects

Ten healthy young adult male subjects were recruited through flyers posted throughout the college campus. On the first visit, recruited subjects met with the principal investigator to review the experimental protocol and discuss the risks and benefits associated with participation. After signing an informed consent, each subject underwent a physical and ECG screening to check for congenital heart defects that could endanger the subject’s health and to ensure the subject was physically healthy enough to participate in the study.

Experimental Protocol

Subjects reported to the laboratory on four separate occasions separated by no less than 36 hours. Upon arriving, subjects sat quietly for 15 minutes in an inclined position. Near the end of the 15 minute rest, a resting blood lactate sample was collected using a handheld lactate analyzer. Height and weight were measured and subjects were fitted with a heart rate monitor. Next, subjects completed a standardized warm-up protocol consisting of 3 minutes of cycling at 30W followed by 3 minutes of self-directed stretching.

On the first visit, subjects performed an incremental exercise test to volitional fatigue on an electronic cycle ergometer in order to determine VO\(_{2\text{peak}}\) and appropriate experimental workloads. The test began at 50 watts and increased 50 watts every 2 minutes. Pulmonary oxygen uptake (VO\(_2\)) was measured breath by breath throughout the test and later averaged
in 30 second intervals. At the end of every stage heart rate (HR) and rating of perceived exertion (RPE) were obtained and blood lactate was measured. The test ended when the subject voluntarily stopped. Upon completion, a final HR and RPE were obtained.

During the remaining three visits to the laboratory, subjects completed one of three experimental trials designed to investigate the effect severe exercise has on the oxygen uptake response to subsequent heavy exercise. Subjects completed either 1) a 10 minute square-wave transition to a heavy workload (25%Δ – CON); 2) a 15 second sprint at 110% VO2peak followed by 9 minutes 45 seconds at 25%Δ (S15); or 3) a 60 second sprint at 110% VO2peak followed by 9 minutes at 25%Δ (S60). Subjects completed the CON trial first, followed by the S15 and S60 trials in a randomized order. Before the two severe-heavy trials, subjects were informed of both the workload and duration of the initial ‘sprint’.

Upon reporting to the lab, subjects were fitted with a heart rate monitor and rested in an inclined position for 15 minutes. Near the end of the 15 minute rest, blood lactate was measured using a hand-held lactate analyzer. The subjects then repeated the standardized warm-up protocol of 3 minutes cycling at 30W and 3 minutes self-directed stretching. Appropriate experimental workloads were entered into the computer before exercise began. To begin, subjects were instructed to increase their cadence, at which time the protocol was initiated and the computer automatically set the appropriate workload(s) against the pedals. Severe-heavy transitions were also pre-programmed into the computer making the transitions themselves almost instant. Pulmonary VO2 was measured breath by breath and smoothed out using a 1-min rolling and 5-point averaging. Blood lactate, HR, and RPE were measured and recorded at the end of minutes 1, 2, 4, 6 and 10.
**Blood Analysis Protocol**

Blood lactate measurements during all visits to the laboratory were conducted using a hand-held lactate analyzer. Before exercise began, subjects sat in an inclined position for 15 minutes. Near the end of the rest period, one of the subject’s index fingers was placed in a cup of warm water to increase blood flow. The finger was then cleaned and dried off with an alcohol wipe and sterile gauze pad respectively. To draw a small amount of blood, a lancet was used to apply a small puncture to the fingertip of the warmed index finger. The finger was then gently massaged until a small bead of blood formed. Blood lactate analysis was then conducted using the hand-held lactate analyzer. The fingertip was wiped clean using a sterile gauze pad, and the subject was asked to apply a small amount of pressure to the fingertip until bleeding stopped.

During all exercise trials, fingertips were not pre-warmed as blood flow was already increased. Blood sampling during exercise began by using an alcohol wipe and sterile gauze pad to clean the fingertip, followed by the use of lancet to apply a small puncture. The fingertip was massaged gently, if necessary, until a small bead of blood formed. Blood lactate analysis was then conducted using the hand-held lactate analyzer. Following analysis, the fingertip was wiped clean using a sterile gauze pad and the subject was asked to apply a small amount of pressure to the fingertip. No finger was punctured more than four times in a single session to avoid finger irritation.

**Instrumentation**

Height and weight were measured using a portable stadiometer (Perspective Enterprises, Portage, MI) and mechanical scale (Detecto, Webb City, MO) respectively.
Exercise was performed on a Lode – Corival (20040817, Groningen, Netherlands) cycle ergometer. This model had been used previously and has an accuracy of ≤3%. Pulmonary gas exchange was measured using a PARVO Medics True Max 2400 (Salt Lake City, Utah) gas exchange analyzer (accuracy – 0.1%), and recorded using the accompanying software (ParvoMedics OUSW Version 3.4). Blood lactate was measured using a hand-held lactate analyzer (Accutrend Lactate Plus, Waltham, MA). Heart rate was measured using a Cardiosport First heart rate monitor and RPE was assessed using the Borg 20 point scale.

Calculations

Oxygen uptake during submaximal exercise can fluctuate on a day to day basis, and although the fluctuations are not statistically significant (87), small differences may have a much greater impact when calculating a moderate VO$_2$-wkld relationship. To minimize potential fluctuations from confounding the moderate VO$_2$-wkld relationship within our study, we averaged the VO$_2$ (L/min) responses of all subjects collected at the end of stages 1, 2, and 3 (50, 100, and 150 Watts) during the incremental test. These workloads were all below the subject’s individual lactate thresholds with the exception of 1, who’s calculated LT was 144W. Using the least squares method, the average VO$_2$ responses were then plotted against workload to derive a line of best fit. Because the subjects cycled at different absolute workloads during the experimental trials, the magnitude of the VO$_2$SC was standardized between subjects according to the equation:

\[ VO_{2SC}\text{magnitude} = \frac{(VO_{2\text{experimental}} - VO_{2\text{expected}})}{VO_{2\text{expected}}} \times 100 \]
Severe exercise intensities were calculated by multiplying the heaviest workload (in Watts) that was completed during the incremental test by 1.1, according to the equation:

$$\text{Workload}_{\text{Severe}} = \text{VO}_{2\text{peak}} \times 1.1$$

Heavy exercise intensities were calculated by taking the workload associated with an individual’s LT (in watts) and adding 25% of the difference between the greatest workload (in Watts) completed during the incremental test (VO_{2\text{peak}}) and the individual’s LT (in Watts), according to the equation:

$$\text{Workload}_{\text{Heavy}} = \text{LT} + 0.25(\text{VO}_{2\text{peak}} - \text{LT})$$

**Statistical Analyses**

All statistical analyses were conducted using computerized software (Statistica, version 9.0, StatSoft Inc., Tulsa, OK) with an alpha level of 0.05 or less being considered significant. An independent 3x3 (3 transitions x 3 time points [minutes 4, 6 and 10]) mixed model ANOVA was conducted to assess the effect exercise transition had magnitude of the VO_{2\text{SC}}. Additionally, an independent 3x6 (3 transitions x 6 times points [rest, minutes 1, 2, 4, 6 and 10]) mixed model ANOVA was conducted to assess the effect exercise transition had on oxygen uptake. An independent 3x6 (3 transitions x 6 times points [rest, minutes 1, 2, 4, 6 and 10]) mixed model ANOVA was conducted to assess the effect exercise transition had on blood lactate accumulation. Rating of perceived exertion was assessed using an independent 3x5 (3 transitions x 5 time points [minutes 1, 2, 4, 6, and 10]) mixed model ANOVA. In the event a significant interaction effect was found, Bonferroni adjustment was used for post hoc analysis. A Pearson correlation was conducted using all blood lactate and
VO$_{2SC}$ magnitude data collected at minutes 4, 6 and 10 to assess the relationship between blood lactate accumulation and VO$_{2SC}$ magnitude.
CHAPTER IV
RESULTS

Subjects

Descriptive characteristics of the 10 subjects who completed the study are listed below in Table 1. Seven of the subjects had cycling experience through either the university club cycling team or competitive triathlons with the three remaining subjects consisting of two long distance runners and one water-polo player.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Body Fat %</th>
</tr>
</thead>
<tbody>
<tr>
<td>22.5 ± 3.6</td>
<td>178.1 ± 4.0</td>
<td>72.6 ± 10.0</td>
<td>10.4 ± 4.8</td>
</tr>
</tbody>
</table>

Table 1 – Physical characteristic data: All data are reported as Mean ± SD.

Table 2 presents data from the Incremental Test. The subjects were successfully exercised to their maximum. The average RER was above 1.1, HR was within 10 beats per minute (bpm) of their expected HR max, and RPE was above 18. Collectively, the subjects ranged from moderate-well trained ($\text{VO}_{2\text{max}}$ 55.0 – 80.5 ml/kg/min). The average heavy workload subjects cycled against during the experimental trials was 232.2 ($\pm$ 26.4) Watts, while the average severe workload subjects cycled against during the sprint trials was 352.0 ($\pm$ 38.5) Watts.

<table>
<thead>
<tr>
<th>$\text{VO}_{2\text{max}}$ (ml/kg/min)</th>
<th>RER</th>
<th>RPE</th>
<th>HR (bpm)</th>
<th>LT (Watts)</th>
</tr>
</thead>
<tbody>
<tr>
<td>66.3 ± 8.0</td>
<td>1.19 ± 0.04</td>
<td>18.9 ± 1.2</td>
<td>187.5 ± 6.4</td>
<td>206 ± 29.1</td>
</tr>
</tbody>
</table>

Table 2 – Incremental test data: All data are reported as Mean ± SD. RER = respiratory exchange ratio; RPE = rating of perceived exertion; HR = heart rate; LT = lactate threshold.
Moderate VO₂-wkld relationship

Figure 4 presents the mean oxygen uptake responses (L/min) to each stage of the incremental exercise test. The average VO₂ responses below the LT, observed at the end of stages 1, 2 and 3, were 1.105, 1.665, and 2.204 L/min respectively. The VO₂-wkld relationship calculated via linear estimation was:

\[ \text{VO}_2 \text{(L/min)} = 0.01099 \times \text{Workload (Watts)} + 0.559 \]

A second linear estimation was performed to determine the VO₂-wkld relationship observed during exercise above the LT (stages 5, 6, and 7). The resulting equation was:

\[ \text{VO}_2 \text{(L/min)} = 0.01705 \times \text{Workload (Watts)} - 0.698 \]

![Graph showing oxygen uptake responses during the incremental test](image)

**Figure 4 – Average oxygen uptake responses during the incremental test:** Individual oxygen uptake responses were included only if the subject fully completed the 2 minute stage. Average oxygen uptake responses at 300 and 350 Watts are based off n=9 and n=5 data points respectively. All other mean oxygen uptake responses are based off n=10 data points.

Oxygen uptake and the VO₂SC magnitude

Figure 5 displays the oxygen uptake responses to the three experimental trials. A significant interaction effect was found (p < 0.01), with oxygen uptake being significantly
increased at the end of minutes 1 and 2 during the S60 trial compared to both the S15 and CON trials (p < 0.01), and at the end of the first minute during the S15 trial compared to the CON (p < 0.01). No other significant differences were observed between trials for oxygen uptake at rest or at the end of minutes 4, 6 and 10. Accordingly, there was not a significant interaction effect between trials over time for the magnitude of the VO2SC. However, a significant main effect (p < 0.05), shown in Figure 6, was observed for the magnitude of the VO2SC, with the S60 trial being significantly increased compared to both the S15 and CON trials (p < 0.01). The p-value obtained between the S15 and CON trials approached, but did not reach, statistical significance (p < 0.052). Additionally,

Figure 5 – Mean oxygen uptake responses to the experimental trials: Error bars have been omitted for clarity. Circles = CON trial; squares = S15 trial; diamonds = S60 trial
Blood lactate accumulation

Figure 3 depicts the blood lactate responses to the experimental trials. A significant interaction effect was found (p < 0.01). No significant differences in blood lactate accumulation were observed between the CON and S15 trials at any time points. Blood lactate accumulation during the S60 trial, however, was significantly increased compared to the CON trial at the end of minutes 2, 4, and 6 (p < 0.01); and at the end of minute 4 when compared to the S15 trial (p < 0.05). A significant correlation was also observed between blood lactate accumulation and the magnitude of the VO2SC (r = 0.55; n = 89).

Figure 7 – Blood lactate responses to the 3 experimental trials: Error bars have been omitted for clarity. Circles = CON trial; squares = S15 trial; diamonds = S60 trial.
Rating of perceived exertion

A significant main effect was found for rating of perceived exertion between the three trials with the cumulative RPE being significantly elevated during the S60 compared to the S15 and CON trials (p < 0.01), and the S15 being significantly elevated compared to the CON (p < 0.02). However, there was not a significant interaction effect between trials over time.

Figure 8 – Main effect of exercise transition on rating of perceived exertion: The S60 trial was significantly greater than S15 and CON trials (p < 0.01). The S15 trial was also significantly greater than the CON trial (p < 0.02).
CHAPTER V
DISCUSSION

Introduction

The main goal of the current study was to investigate how sprinting at the onset of endurance exercise affects measures of oxygen uptake and blood lactate accumulation in well-trained males with the intent of judging if sprints lasting 15-60 seconds would affect mid-race performance. Given the novel nature of the exercise protocol used during the experimental trials, the secondary goal of the current study was to investigate the feasibility and practicality of utilizing severe-heavy step-wise exercise transitions within a laboratory setting. The following chapter will briefly describe the physiological implications of the results presented in Chapter IV, relate the findings to previously published literature, and provide possible explanations for the outcomes reported.

Main finding

The main finding of the current investigation is that severe sprints lasting 15-60 seconds do affect measures of oxygen uptake, blood lactate accumulation and RPE during the first few minutes of subsequent endurance exercise in well-trained males, but the responses normalize within about 10 minutes. Consistent with previous research, exercise above the lactate threshold caused a significant increase in blood lactate and produced a VO₂SC (5, 6, 88-90), both of which were at least temporarily increased further by supra-maximal exercise
(91-92). However, despite several significant between trial main effects, oxygen uptake, blood lactate, and rating of perceived exertion were not significantly different at the end of any experimental trial. Rather than indicating a prolonged effect, the significant main effects found for the VO$_{2sc}$ magnitude and RPE, described in Figures 6 and 8 respectively, appear to coincide with the significantly increased VO$_2$ and blood lactate responses (Figures 5 and 7) observed during the first 1-4 minutes of the S60 trial. Collectively, these results suggest that although sprinting at the onset of an endurance competition will influence the kinetic responses of VO$_2$, blood lactate accumulation, and RPE there will not be a sustained negative impact on ensuing race performance as described by the lack of interaction effects displaying significant differences 10 minutes into exercise. Rather, the sprints may actually benefit the athlete by providing the opportunity to establish positive tactical positioning. Put another way, sprinting at the start of a race does not appear to influence the phenomenon coaches refer to as “going out too fast”. Although these findings are contrary to the hypotheses, several possibilities exist to explain the results.

**Methodological explanations**

Given the novel nature of the exercise protocol, perhaps methodological factors had an influence on our findings. The “expected” VO$_2$ values for minutes 4, 6, and 10 of the experimental trials are not likely a source of error, as the incremental test yielded VO$_2$-wkld results that were consistent with previous investigations (3, 5, 93). As depicted in Figure 1, VO$_2$ rose linearly a rate of ~11ml/Watt for workloads below the LT (stages 1-3) and then broke sharply after 200 watts increasing at a rate of ~17ml/Watt. Additionally, the results for the magnitude of the VO$_{2sc}$ were not influenced when reanalyzed with a 3x3 repeated
measures ANOVA in the more common ‘absolute’ (L/min) format; yielding a non-significant interaction effect between time and magnitude, but a significant main effect for Trial, with the S60 trial producing a significantly greater VO$_{2SC}$ magnitude than the S15 and CON trials (Figure 6).

Analytical methods aside, perhaps 10 minutes was not a large enough window to allow VO$_2$ and the VO$_{2SC}$ magnitude to dissociate between trials. However, this does not appear likely either, as the mean VO$_2$ and blood lactate responses were normalizing as time progressed, rather than being driven further apart. Additionally, blood lactate actually declined between minutes 6 and 10 within several of the more aerobically fit subjects; suggesting they were recovering rather than fatiguing. In a similar sense, perhaps the sprints were not long enough to elicit the expected responses. While this may be true, the sprints utilized during this investigation were both realistic to highly competitive endurance races and appropriate to answer the questions. With that being said, this study did not investigate the effect of a prolonged severe exercise bout (i.e. 4-5 minutes at 85% VO$_{2max}$). That is, rather than simulating an athlete who sprints to establish race position, a longer severe exercise bout may simulate an athlete who simply employs poor pacing strategy. Given that the experimental protocol was only 10 minutes long, a 5 minute severe workload did not seem practical. Thus, although a more prolonged experimental protocol does not appear necessary to further investigate whether or not shorter sprints affect endurance performance, it may make investigating a longer sprint more practical.

Lastly, well-trained endurance athletes typically display impeccable pacing abilities and can intuitively exercise just below their MLSS during races. Although it is common to prescribe experimental workloads based upon a % difference between VO$_{2max}$ and LT (17,
one aspect of the study that was not considered beforehand was purposely exercising subjects just below their maximal lactate steady state (MLSS), the optimal race pace for endurance races lasting 30-60 minutes (94), for the “heavy” (Δ25%) intensity exercise. Although detection of the MLSS within the laboratory can be difficult, this may have allowed a better assessment of whether or not sprints affect race performance from a threshold perspective (i.e., does sprinting affect the workload associated with the MLSS). During the current investigation, the intensity of the Δ25% workload appeared to fall into the severe intensity domain for some of the subjects as portrayed by a continued rise in VO$_2$ from minute 6 to 10 during the CON trial. When responses between subjects cycling at heavy (n=5) or severe (n=5) workloads were compared using independent 1-way ANOVAs, the magnitude of the VO$_{25C}$ was significantly greater in the severe group during the CON and S15 trials, but not the S60 trial. Contrariwise to the original analysis, these results would suggest that 60 second sprints do shift well-trained males from a previously heavy intensity, into the severe intensity domain (i.e. shift the MLSS to a lower workload), and that the non-significant finding within the current investigation may have been due to some of the subjects cycling at a Δ25% workload that was too intense for them during the CON trial.

**Physiological explanations**

One plausible explanation not related to our methodology is that mid-race performance in well-trained endurance athletes is simply not affected by a single 15-60 second sprint. Duffield et al. (49) reported that endurance training improved VO$_2$-kinetics (fast component amplitude) and reduced accumulated oxygen deficit during severe intensity exercise. Furthermore, endurance training has been shown to reduce both blood lactate
accumulation and the magnitude of the VO$_{2SC}$ during exercise (38, 50, 95). Interestingly, our two least fit subjects (mean VO$_{2max}$ ~59.4 ml/kg/min) did not appear to tolerate the sprint trials well; displaying a mean end-exercise difference of 0.36 L/min between CON-VO$_2$ and Sprint-VO$_2$ (S15 and S60 trial responses averaged together). In contrast, the well-trained group (mean VO$_{2max}$ ~68.1 ml/kg/min) displayed a 0.09 L/min difference. Furthermore, when the results were reanalyzed with these two subjects excluded, no significant differences in blood lactate accumulation were found between any trials except S60 and CON at minutes 2 and 4 (p < 0.05). Thus, the less trained subjects appeared to account for the non-significant but noticeable differences in VO$_2$ (Figure 5) and blood lactate accumulation (Figure 7) observed between the CON and sprint trials at 10 minutes, indicating training status and/or fitness level may play an important role in an athlete’s ability to tolerate sprints at the start of endurance exercise.

On a similar note, perhaps the variety of endurance activities our subjects engaged in was too heterogeneous. That is, perhaps the lack of cycling experience within three of our subjects had an influence on their physiological responses and therefore the results. With that being said, the physiological responses of the non-cycling subjects appeared to be normal (i.e., comparable to the cycle trained subjects), and when the data was reanalyzed after these subjects had been removed, the results were very similar and no different conclusions could be drawn.

Another factor to consider is the sprints utilized within the current investigation may not have induced fatigue within muscle fibers that were recruited during the Δ25% workload. Some studies have suggested the VO$_{2SC}$ may be caused by the recruitment of type-II fibers (96-98), as faster pedaling frequencies appears to augment the magnitude of the VO$_{2SC}$ and
surface EMG activity in a proportional manor (96-99). Our results would suggest a similar phenomenon, as subjects increased their cadence rate to complete the sprints, potentially contributing to the significantly elevated VO$_2$ and blood lactate responses observed during the first few minutes of the S60 trial. Additionally, once the sprints ended, subjects’ pedaling frequency returned to a cadence similar to that seen during the CON trial, and VO$_2$/VO$_{2sc}$ magnitude gradually normalized between trials. No statistical procedures were conducted on cadence rates between trials, however, so this conjecture remains speculative. None the less, perhaps the sprints fatigued higher order (fast twitch) motor units that were not recruited once the sprint ended, and were thus allowed to slowly recover during the first few minutes of Δ25% cycling. This would also potentially help explain why blood lactate increased greatly during the first few minutes of the S60 trial before declining in the more fit subjects.

If not mid-race performance, perhaps end-race performance is compromised. That is, perhaps sprinting at the start of the race affects an athlete’s ability to sprint at the finish. This too appears unlikely, however, as once again, several of the fitter subjects appeared to be recovering from minutes 6-10. Unfortunately, surface EMG measurements were not collected during the present investigation, so inferences about alterations in motor unit recruitment patterns remain inconclusive.

**Practical implications**

First, it should be acknowledged that one key limitation to the current investigation is that the results pertain to exercise conducted on an electronic cycle ergometer. Although it was our intention to apply the findings to racing tactics for competitive endurance runners, the use of an electronic cycle ergometer made data collection more feasible and drastically
improved the subject’s safety; as stepping onto a treadmill at fast speeds can be dangerous. Therefore, caution should be employed when applying the results of this study to running strategy at the start of a foot race.

With that being said, from a performance perspective, the results of the current investigation suggest that short sprints lasting approximately 15 seconds do not have a negative impact on subsequent performance during endurance races in well-trained males, and thus may be advantageous if they allow the athlete to gain positive tactical positioning. More specifically, 15 second sprints do not appear to affect prolonged measures of oxygen uptake, blood lactate accumulation, or ratings of perceived exertion. On the other hand, the results imply less well-trained individuals may be affected; however, further research will be necessary to provide a more accurate answer.

Similarly, the results suggest well-trained males can tolerate and recover from sprints lasting 60 seconds as well, such that mid-race performance will not be compromised or negatively affected. Once again, however, further research will be necessary before a more accurate recommendation can be made regarding the complete effect 60 second sprints have on endurance race performance. As previously mentioned, when the results were reanalyzed to compare subjects who exercised in the heavy domain during Δ25% to those who appeared to be exercising in a severe domain, only the S60 trial resulted in a non-significant difference between VO$_{25C}$ magnitudes at 10 minutes, suggesting that the 60 second sprint may have indeed shifted the Δ25% workload to a more severe intensity. Additionally, there was a significant main effect for RPE. Specifically, the S60 trial induced a greater overall rating of perceived fatigue than the S15 and CON trials, suggesting the subjects may reach a critical fatigue threshold quicker during the S60 trial if the exercise duration were extended.
Another factor to keep in mind is fitness level. As was discussed, the sprints did not appear to affect the well-trained athletes, whereas the less well-trained athletes did appear to be susceptible to premature fatigue induced by the sprints. Thus, this would suggest the results are only applicable to well-trained males and should not be used to advise untrained individuals until more data can be collected.

Lastly, from a research/methodological perspective, the current investigation demonstrates that severe-heavy exercise transitions can be performed under laboratory conditions in well-trained males using a cycle ergometer. However, based upon the experiences within this study there are several factors that should be considered when utilizing similar methods in the future; these include 1) careful attention to the intensity of CON exercise, 2) smaller workload increments during incremental tests to allow for detection of subjects’ MLSS, and 3) sprint workloads may need to be adjusted (reduced) to allow subjects to complete extended protocols lasting 20 minutes or longer.
CHAPTER VI

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

Ten moderate-well trained males reported to the laboratory on four separate occasions, each separated by at least 48 hours. On the first visit, subjects completed an incremental exercise test to volitional fatigue in order to determine $\text{VO}_{2\text{max}}$, LT, and appropriate experimental workloads. One the remaining three visits, subjects completed one of three experimental trials on a cycle ergometer to determine the effect sprints had on measures of oxygen uptake and blood lactate accumulation during subsequent heavy exercise. The first trial served as a control and consisted of a 10 minute exercise bout at workload calculated to be 25% of the difference between the subjects $\text{VO}_{2\text{max}}$ and LT ($\Delta25\%$). The final two trials were conducted in random order and began with a sprint lasting either 15 or 60 seconds before the workload was immediately reduced to $\Delta25\%$, where it remained for the remainder of the 10 minutes. No significant differences were found between any of the trials at the end of 10 minutes. However, oxygen uptake significantly increased during the S60 trial as compared to the CON at the end of minutes 1 and 2 and blood lactate was significantly increased at the end of minutes 2, 4, and 6. A significant interaction effect between time and $\text{VO}_{2\text{SC}}$ magnitude was not found between trials, however, a significant main effect was found, with the S60 trial being significantly greater than both the S15 and CON trials. The findings suggest that sprints lasting 15 seconds affect oxygen uptake kinetics, but not steady state oxygen uptake, during subsequent heavy exercise.
Similarly, sprints lasting 60 seconds significantly increase oxygen uptake and blood lactate accumulation during the first 4-6 minutes of subsequent heavy exercise, but the effects appear to normalize within 10 minutes. Thus, sprinting at the start of a long distance endurance event does not appear to have prolonged negative consequences on endurance performance, and may be advantageous if it allows athletes to gain positive tactical positioning without compromising mid-race performance.

**Hypotheses (accept or reject)**

1. The magnitude of the VO$_{2SC}$ during submaximal steady state exercise will be significantly higher during the sprint trials (S15 and S60) than during the CON trial.

   (Reject)

2. The magnitude of the VO$_{2SC}$ during submaximal steady state exercise will be significantly higher during the S60 trial during the S15 trial. (Accept)

3. The blood lactate response will be significantly higher during the sprint trials (S15 and S60) than during the CON trial. (Reject)

4. The blood lactate response will be significantly higher during the S60 trial than during the S15 trial. (Accept)

5. There will be a significant positive correlation between the increase in blood [lactate] and the magnitude of the VO$_{2SC}$ when combining data from the CON, S15, and S60 trials together. (Accept)
Recommendations

Methodologically, direct determination of both the LT and the MLSS during incremental tests is recommended as it would provide a more complete description of individual exercise intensity and make appropriate exercise intensity prescription more feasible. That is, it is recommended researchers move away from the “% difference between LT and VO$_{2\text{max}}$” method of prescribing experimental workloads, as it does not uniformly place subjects within the “heavy” or “severe” domains. It is also recommended that smaller stage-stage workload increments be used during incremental tests in order to make detection of the MLSS more feasible.

With regards to severe-heavy exercise transitions, the effect 60 second sprints have on subsequent performance during heavy exercise needs to be reinvestigated; paying special attention to the true intensity of the “heavy workload. According to our results, a more prolonged protocol does not appear necessary to investigate this type of exercise transition; however, it would be advantageous to extend the experimental protocol in order to make investigating a longer sprint (i.e. 5 minutes at ~85% VO$_{2\text{max}}$) more feasible. Thus, it is recommended that our study be repeated by replacing the S15 trial with the more prolonged sprint and extending the duration of the experimental rides out to 20-25 minutes.

On a similar note, adding a “sprint” to the end of an experimental trial would help investigate the effect severe-heavy exercise transitions have on end-race performance. Although pilot testing will likely be necessary to further investigate the feasibility of such a protocol change, it is recommended that time to volitional fatigue be investigated at an intensity approximately equal to 85% VO$_{2\text{max}}$.
It is also recommended that surface EMG data be collected during the experimental trials, as this would contribute valuable information regarding muscle fatigue and recruitment patterns during severe-heavy exercise transitions. Additionally, although faster cadences appear to have a greater influence on the VO\textsubscript{2SC} magnitude than slower cadences, it would be interesting to see if a slower cadence, carried out during the initial severe “sprint”, could fatigue lower order (i.e. slow twitch) muscle fibers and subsequently influence motor unit recruitment patterns and the VO\textsubscript{2SC} magnitude during subsequent heavy exercise.

Lastly, it is recommended this protocol be repeated to investigate the effect severe-heavy exercise transitions have on untrained subjects; as the results suggested less trained individuals may behave differently than well-trained counterparts.
References


