THE EFFECT OF WARM UP INTENSITY ON
TIME TO EXHAUSTION AT VO₂ MAX IN RUNNERS

A Thesis

Presented to the faculty of the Department of Kinesiology
California State University, Sacramento

Submitted in partial satisfaction of
the requirements for the degree of

MASTER OF SCIENCE

in

Kinesiology
(Exercise Science)

by

Kevin Tafalla Dinglasan

SUMMER
2012
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Abstract

of

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Introduction

Substantial research exists detailing the oxygen kinetics, blood lactate responses, and the effect of warm ups on subsequent exercise performance but very few studies attempt to combine these variables. Recent research has demonstrated relationships among these variables in cyclists, eliciting significant performance gains. However the majority of research in this area is often performed on cyclists, not runners. Therefore, the purpose of the current investigation was to examine the effect of a moderate and high intensity warm ups on the physiological responses related to oxygen use, blood lactate and perceptions of exertion in trained runners during time to exhaustion tests at VO₂ max.

Methods

Ten (one female) healthy, well trained subjects volunteered to participate in this study. Each subject completed five visits with at least 48 hours between. The first two visits were baseline VO₂ max testing and submaximal testing to determine warm up intensities and theoretical oxygen costs. The third through fifth visits were a randomization of three different 6 minute warm up conditions: a control condition with no warm up, a moderate intensity warm up at GET, and a high intensity warm up at
70%Δ followed by a 20 minute recovery and a time to exhaustion trial (TTE) conducted at VO₂ max. All subjects kept a diet and exercise log for the three days leading up to each visit. Expired gases were collected continuously, while HR and RPE were collected every minute during TTE. Blood lactate was collected immediately post warm up, and pre and post TTE.

Results

Mean TTE was significantly increased by 21% (287.7 ± 56.5 s v. 236.7 ± 64.9 s, P < 0.05) in the moderate intensity group over the control, but the change in TTE in the high intensity group was not significant (270.5 ± 46.3 s). The intervention had no significant effect on oxygen uptake, accumulated oxygen deficit or ratings of perceived exertion.

Conclusions

A moderate intensity warm up was effective in significantly increasing TTE at VO₂ max in well trained competitive runners. Despite the improved exercise tolerance, there was no significant change in the measured physiological variables regardless of the condition. More research focusing on neurological aspects such as perception and motor drive is warranted to better understand the beneficial aspects of warming up.

_____________________________, Committee Chair
Roberto Quintana, PhD

_____________________________
Date
ACKNOWLEDGEMENTS

First and foremost, I would like to thank God. The faith I have in Him is the lens through which I view the world and my life—it governs my thoughts, my motives and my actions. It is why I am where I am today.

I am forever thankful for my family, the giant upon whose shoulders I stand. My parents, my sisters and my close extended family have all made my academic endeavors possible. Whether it is through a quiet confidence in me, or emotional and financial support, their investment in my life is integral not only to my achievements and well-being, but also to who I am.

I owe a great debt to my cohort, the exercise science students with whom I have spent countless hours in lecture and lab. My most heartfelt congratulations go out to those who have already graduated, and my most sincere encouragement to those who are yet to finish. I would specifically like to thank Rachel Aldous, Kelsey Hammerel and Emily O’Shaughnessy—long time group members in almost all of our classes—for their help, diligence and encouragement. I also want to thank Rick Bradley and Max Polin, both of whom gave me advice on the logistics of running the actual experiment in terms of preparation, scheduling and equipment. Also, I am very grateful to Mike Wortman for his help getting my research started and fine tuning the protocol and procedures.

I want to acknowledge Scott Abbott for his interest in the study and his help recruiting local runners. I also want to thank Ericka Violett. The flyers she designed helped advertise my research, getting the word out to local running stores and clubs and
broadening my recruitment area. Without either of them, this research would still be at a standstill with no subjects.

I want to thank each one of my subjects for committing to the research, following through and completing the study. The protocol was unusual and difficult, but they all finished and I literally would not have been able to this without any of them.

Undoubtedly, I would not have been able to complete this research and degree without each one of my professors that selflessly imparted of themselves for the advancement of my education. So a most heartfelt thank you goes to Dr. William Edwards, Dr. Michael Wright, Dr. Jennifer Lundmark, Dr. Rodney Imamura, Dr. David Mandeville, Dr. Laura Barger, Dr. Daryl Parker and Dr. Roberto Quintana. It is the quality of not only their instruction but also their character that inspired me to pursue my master’s degree at California State University, Sacramento in Exercise Science.

And finally Dr. Roberto Quintana, my thesis advisor. Although I probably spent more time talking to him about track and field, running, training and basically everything but my thesis during his office hours, he was integral to this research. It was his idea that formed the basis of my thesis and his encouragement, auspices and guidance that helped me see it through to completion. His patience and constant pestering when I was too lazy to write kept me on track. And although he was constantly meddling in my running, it kept me in shape and prevented me from being inundated by my thesis. For all of this I am exceedingly grateful, but it is perhaps his friendship that has proven most invaluable.
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Chapter 1

INTRODUCTION

Warm ups are generally accepted as part of the pre-activity routine. However, there is little scientific evidence that explains the purpose, mechanisms and benefits of warming up before an activity (Bishop, 2003a). In order to appropriately study warm ups, they must be categorized according to their method and parameters. The method of a warm up can either be passive (raising temperature by external means) or active (raising temperature, as well as metabolic and cardiovascular markers by exercise or activity). Changing the parameters of the warm up (duration, intensity, recovery, continuity, mode) can make the combinations nearly infinite (Bishop, 2003a, 2003b).

A recent cycling study attempted to structure a warm up, varying intensity and rest duration in order to optimize subsequent exhaustive exercise tests. The results showed that a high intensity warm up coupled with a longer recovery produced the most improvement in exhaustion tests. However, if the recovery was shorter, either lesser improvement or decrement followed. The findings were believed to support the notion that warm ups affected the oxygen kinetics of the following exhaustion test (Bailey, Vanhatalo, Wilkerson, DiMenna, & Jones, 2009).

One of the main findings thought to be crucial to improved performance after heavy intensity warm ups is oxygen kinetics. Though improvements in the oxygen kinetics curve were observed, the mechanism did not necessarily lie in the curve itself. Instead the oxygen kinetics curve represents, to a certain degree, the state of metabolism. Several studies assert that the reduction in the amplitude of the slow component and the
elevation in baseline VO\textsubscript{2} indicate a sparing of finite anaerobic energy stores and heavier reliance on oxidative energy sources (Bishop, 2003b; Burnley, Doust, Ball, & Jones, 2002; Burnley, Jones, Carter, & Doust, 2000; Wilkerson, Koppo, Barstow, & Jones, 2004; Wittekind & Beneke, 2009). The exact mechanism of how this achieved is still widely contested.

Since the VO\textsubscript{2} response at the onset of exercise (i.e., oxygen kinetics curve) can provide valuable metabolic information, anything that has the potential to alter it for performance benefit warrants investigation. Some researchers cite the Bohr effect of hemoglobin as a possible mechanism explaining, at least in part, the improved VO\textsubscript{2} response (Jones, Koppo, & Burnley, 2003). Prior exercise has been demonstrated to raise blood lactate levels at the onset of a second bout of exercise. The drop in pH associated with blood lactate is thought to shift the oxy-hemoglobin dissociation curve rightward, facilitating more oxygen to be unloaded into the muscle. The decreased pH can also serve as a vasodilator, improving blood delivery and remedying tissue perfusion heterogeneity (Bailey, et al., 2009; Jones, Koppo, et al., 2003; Poole, Barstow, McDonough, & Jones, 2007).

One proposed mechanism is the altering of muscle activity and recruitment. Several studies cite the progressive recruitment of motor units, specifically type II fibers, in the first two minutes of exercise as being responsible for the existence of the VO\textsubscript{2} slow component (Sabapathy, Schneider, & Morris, 2005). As such, increasing motor unit recruitment at the onset of exercise may decrease the amplitude of the slow component. Because recruitment increases, the metabolic load is spread across more units and fibers.
Thus, each fiber has a lower metabolic demand, which in turn allows for an increased reliance on oxidative rather than anaerobic processes (Bailey, et al., 2009; Burnley, et al., 2002). Additionally, if the slow component and muscle motor unit recruitment are truly positively correlated, it may be possible to illustrate the specifics about fiber type recruitment. The increase in reliance on oxygen and oxidative processes implicates those fibers that use oxygen, primarily Type I and to some extent Type IIa fibers. Therefore, a high intensity warm up may not only alter the amount of motor units recruited, but also facilitate a heavier reliance on more efficient fibers in subsequent exercise (Burnley, et al., 2002).

Greater reductions in the slow component amplitude and therefore, more efficient motor unit recruitment, should theoretically improve subsequent performances. The problem with this notion is that it assumes a linear relationship between the slow component amplitude and exercise performance. In Bailey’s study, while up to 30% improvements in time to exhaustion tests were associated with reductions in the slow component amplitude and elevated baseline VO$_2$, the greatest reduction in slow component amplitude and elevation in baseline VO$_2$ did not produce the best performance in time to exhaustion. In fact, that condition was associated with the poorest time to exhaustion performance. This suggests that oxygen uptake kinetics may not be the sole factor in improving subsequent performance with warm up (Bailey, et al., 2009).

Another possible contributing factor is the notion of central fatigue. In this viewpoint espoused by Noakes and colleagues, fatigue is no longer a defined physical event delineated by physiological measures; rather it is a decrease in or cessation of
neural output from the central nervous system to the muscles based on subconscious interpretations of various forms of feedback regarding past experiences, current physiological conditions, projected goals and the maintenance of homeostasis (Noakes, St. Clair Gibson, & Lambert, 2004, 2005). The central nervous system output, or central motor drive, is what initiates what is known as fatigue. It works to preserve the human body and to prevent catastrophic failure of any system, thus allowing exercise to terminate in a relatively homeostatic condition. Therefore, the body and its reaction to any stressors, such as exercise, is centrally governed (Noakes, et al., 2004, 2005).

If this central governor model posited by Noakes is accurate, then altering how the central nervous system interprets afferent feedback can alter its initiation of fatigue. Research exists that suggests exercise can be a tool for altered pain perception (Koltyn, 2000; Koltyn & Umeda, 2006). Suggested mechanisms behind this include endogenous opioid systems acting on muscle afferents, in turn altering the central motor drive. Though a relatively new area of research, future studies may hold the key to understanding just how warm ups or prior exercise are capable of producing performance benefits, as seen in Bailey’s 2009 cycling study.

Another important warm up consideration is that the majority of studies in this area have been conducted with cycle ergometry. Few studies use running as the mode of warm up and exercise testing in regards to oxygen kinetics (Jones et al., 2008). One study attempted to explicate the similarities and differences between the oxygen kinetics of cycling and running at the onset of exercise (Carter et al., 2000). The author Carter states that the current research shows a decreased amplitude of the slow component while other
researchers question the existence of the slow component altogether in running when compared to cycling (Billat, Richard, Binsse, Koralsztein, & Haouzi, 1998). Carter’s research, and later Wittekind’s research in 2009, affirmed the former—that running oxygen kinetics displayed a smaller slow component amplitude (Wittekind & Beneke, 2009). Carter attributed this to the relatively greater recruitment of Type II fibers in cycling as compared with running, supporting the muscle activity and recruitment theory as the primary mechanism (Billat, et al., 1998; Wittekind & Beneke, 2009). The fact that the characteristics and existence of the slow component are still controversial in current research highlights a significant deficiency in the literature.

In spite of all this information, to the knowledge of these authors there is no such study that specifically investigated how these warm up findings apply to running. Future research will need to look at the effects of a protocol like that of Bailey’s 2009 study on runners. Such a study will not only help illustrate the effect of a structured warm up on a sample population of runners, but it may also elucidate the contributing factors and mechanisms of warm ups acting on exercise tolerance.
Statement of Purpose

The purpose of the current investigation was to examine the effect of a moderate and high intensity warm up on the physiological responses related to oxygen use, blood lactate and perceptions of exertion in trained runners during time to exhaustion tests at VO₂ max.

Significance of the Thesis

To date, substantial research exists detailing the oxygen kinetics, blood lactate responses, and the effect of prior exercise (i.e., warm ups) on subsequent exercise performance but very few studies attempt to combine those variables. Recent research has demonstrated relationships among these variables in cyclists, eliciting significant performance gains. However, in addition to its scarcity, the present state of research in this specific area is often performed on cyclists, not runners. This investigation will attempt to fill those gaps by supporting or disputing recent developments while also applying the principles to a different mode of exercise. While merely a stepping stone, this study has the potential to better explicate, manipulate and utilize the convention of warming up.
Limitations

(1) The results of the present study may only be generalized to populations similar to the study population.

(2) The response to warm up may vary according to each individual.

Delimitations

(1) The researcher limited the sample to 10 healthy, well-trained runners actively competing with a minimum two year competition history, under the age of 45 (males) or 55 (females).

(2) The researcher limited the sample to subjects from the greater Sacramento area.

(3) While the subjects received instruction and guidelines regarding both training and diet, the researcher did not have complete control over the training or diet, relying on three day training and diet logs prior to each visit.

(4) The researcher limited the study to include only a high intensity and moderate intensity warm up.

(5) The researcher limited the criterion bout to a high intensity time to exhaustion test.

Assumptions

(1) All subjects will follow pre-visit protocol and accurately track training and diet.

(2) All subjects will give a maximal effort in the VO$_2$ max and time to exhaustion tests.
Hypotheses

(1) There will be no significant difference in TTE among the groups.

(2) There will be no significant difference in oxygen kinetics during the first 3 minutes and at the end of the TTE as described by VO\textsubscript{2} and AOD values among the groups.

(3) There will be no significant difference in blood lactate concentrations post warm up, pre TTE and post TTE among the groups.

(4) There will be no significant difference in RPE during the first 3 minutes and at the end of TTE among the groups.
The Effects of Warm Up on Exercise Performance

Warm ups can be considered as one of two types: passive or active. Passive warm ups are usually defined as methods of increasing muscle temperature or core temperature by an external means (Bishop, 2003a). Because of the passive nature of these warm ups, the primary mechanism through which they affect the body is elevated temperature. Elevated temperature of the body and working muscles is thought to reduce stiffness and viscosity in the muscular skeletal system, facilitating ease of movement. Although the research in this area is relatively inconsistent in design and control, passive warm ups have shown some minor improvements in short (< 10 seconds) and intermediate term performance (> 10 seconds, < 5 minutes), while neutrally or negatively affecting long (> 5 minutes) term performance (Bishop, 2003a).

Alternatively, active warm ups are not only capable of increasing muscle and core temperature, but also eliciting greater metabolic and cardiovascular changes when compared to passive means. Again, performance gains in short duration activities tend to be elicited by increases in muscle and core temperature. Furthermore, performance decrements can follow an active warm up if the warm up is too intense or does not allow sufficient recovery. Intermediate and long term performance are generally improved by active warm ups, provided the warm up does not fatigue the individual but simultaneously elevates metabolic and cardiovascular processes (Bishop, 2003b).
Because activity is involved in active warm ups, more variables are introduced that can be manipulated to formulate an active warm up: duration, intensity and recovery. The manipulation of these variables can have profound effects on exercise performance. It has been suggested that an active warm up of sufficient intensity (40-60% VO$_2$ max) and duration (5-10 minutes) to raise muscle temperature coupled with a recovery of sufficient duration (5 minutes) to restore high energy systems is most beneficial for shorter term activities. Intermediate and longer term activities may require more intense warm ups (60-70% VO$_2$ max) for similar durations (5-10 minutes) but followed by shorter recoveries (≤ 5 minutes) to allow for elevated baseline VO$_2$. As indicated by the vagueness of the guidelines in research and the varying styles, methods and structures of warm up in use within a single sport alone, there is no clear consensus as to what works best and the mechanism behind it (Bishop, 2003b).

In an attempt to further optimize warm up and better understand its underlying mechanisms, a recent study was oriented toward studying the effects of specific warm ups on subsequent exercise performance. Bailey et al. (2009) studied eight male participants 18-24 years old. Through the study, it was deduced that prior high intensity cycle ergometry was capable of increasing tolerance to severe intensity cycle ergometry as long as the duration of the recovery period was long enough (≥ 9 minutes) to preserve the change in VO$_2$ kinetics elicited by the warm up and also long enough to provide for restoration of muscle homeostasis. The various conditions in this study were control with no warm up; heavy exercise with 3, 9 and 20 minute recoveries; severe exercise with 3, 9 and 20 minute recoveries. This resulted in seven conditions all followed by time to
exhaustion (TTE) tests at severe intensity, with heavy exercise for the warm up defined as 40% of the difference between gas exchange threshold (GET) and VO$_2$ max (40%Δ), severe exercise for the warm up defined as 70%Δ and severe intensity exercise for the TTE defined as 80%Δ. TTE was significantly increased in the severe intensity warm ups followed by both the 9 minute (70-9-80, 15% increase) and 20 minute recoveries (70-20-80, 30% increase), and impaired in the 70-3-80 (16% decrease) condition when compared to control. All three of the heavy intensity warm ups did not significantly affect TTE (Bailey, et al., 2009).

Of the several physiological responses measured in this study, oxygen kinetics was at the forefront. Previous research hypothesized that minimizing the oxygen deficit at the onset of exercise could prove beneficial to exercise performance (Bishop, 2003b; Burnley, Doust, & Jones, 2005; Burnley, et al., 2000; Wilkerson, et al., 2004; Wittekind & Beneke, 2009). Decreases in the VO$_2$ slow component, representing a decrease in the oxygen deficit, should therefore produce proportional increases in performance. However, Bailey’s study found that the while four of the conditions (40-3-80, 70-3-80, 70-9-80 and 70-20-80) had significant decreases in the slow component over control, only two conditions (70-9-80 and 70-20-80) showed significant improvement in TTE. This suggests that the primary mechanism for improvement may include more than just oxygen kinetics (Bailey, et al., 2009).

Another physiological marker measured was blood lactate. It has been suggested that elevations in blood lactate above baseline (~3-5 mM) prior to exercise are associated with enhanced performance and altered oxygen kinetics (Burnley, et al., 2005; Jones,
Wilkerson, Burnley, & Koppo, 2003). However, Bailey’s study found that while three of the conditions (40-3-80, 70-9-80 and 70-20-80) began with blood lactate concentrations of 3-5 mM, only two of the conditions (70-9-80 and 70-20-80) showed significant improvement in TTE and significant differences in oxygen kinetics (Bailey, et al., 2009). This also suggests there may be something more beyond the relationship between blood lactate and oxygen kinetics concerning exercise performance.

Integrated electromyography (iEMG) also played a significant role in bringing new perspective to the mechanisms of warm up in Bailey’s study in 2009. Previous research has demonstrated that iEMG activity is higher at the onset of the second of two bouts of high intensity exercise, followed by a blunted increase in iEMG as exercise progressed. This observation was also associated with improvements in oxygen kinetics (Burnley, et al., 2002). Additionally, the progressive recruitment of motor units throughout exercise has been implicated as a contributing factor to the development of the VO₂ slow component (Burnley, et al., 2002; Jones, Koppo, et al., 2003). Bailey’s study showed similar results, wherein the 70-20-80 condition reported a trend toward increased motor unit recruitment at onset, and a reduced recruitment later on. This was also associated with the improvement in oxygen kinetics and possibly indicates alterations in motor drive (Bailey, et al., 2009).

Undoubtedly, the physiological mechanisms of warm up are varied and not clearly understood.
Oxygen Kinetics

Oxygen kinetics—the dynamic manner in which VO$_2$ behaves during exercise—offers insight into the state of oxygen usage. At the onset of exercise, this becomes particularly important; there is a delay between the immediate increase in imposed work rate from rest to exercise and the mitochondrial adenosine triphosphate (ATP) provision response. The oxygen response at the onset of exercise can be characterized into three different phases. Phase I is the apparent delay in VO$_2$ increase, reflecting the transit time between the muscles and the lungs. Phase II represents a monoexponential increase in VO$_2$ to reach the required oxygen cost (steady state oxygen consumption) of the activity. Phase III is the steady state of oxygen consumption for the given intensity (Burnley, et al., 2000). The observed delay therefore encompasses both Phase I and Phase II and is termed the oxygen deficit.

The oxygen deficit is indicative of the degree of substrate-level phosphorylation that buffers the mitochondrial provision of ATP during abrupt changes in work rate (Bailey, et al., 2009; Jones, Koppo, et al., 2003). It would therefore follow that oxygen kinetics at the onset of exercise have important implications for exercise performance and tolerance. Essentially, more of the energy for the initial work would be provided by aerobic sources, reserving anaerobic sources for later (Bishop, 2003b).

Several studies have looked at the effect of prior exercise on subsequent exercise oxygen kinetics and how it might be altered. Altered oxygen kinetics was thought to be manifested by an overall speeding of oxygen kinetics and/or a reduction in the slow
component amplitude. Overall speeding of oxygen kinetics involves reducing the mean response time (MRT). The MRT, the time to reach 63% of the increase in VO\(_2\) above baseline, reflects the rates of VO\(_2\) response of Phase I and partially Phase II (Jones, Koppo, et al., 2003). At the onset of exercise, the MRT is part of the perceived delay in VO\(_2\) response. If this delay could be reduced, then theoretically the amount of work accomplished at the onset of exercise could be shifted to more aerobic sources rather than anaerobic, as indicated by increased oxygen usage. However, recent research has not demonstrated this phenomenon (Bailey, et al., 2009; Burnley, et al., 2002; Burnley, et al., 2000; Jones, Koppo, et al., 2003; Poole, et al., 2007).

Instead of altering the MRT, the majority of research points to the slow component of oxygen kinetics. The slow component is unique to exercise occurring between the lactate threshold (LT) and VO\(_2\) max, emerging after Phase II of oxygen kinetics. This slow component can either delay steady state oxygen consumption, or set oxygen consumption on a course to VO\(_2\) max above the predicted steady state value (Bailey, et al., 2009; Burnley, et al., 2000; Jones, Koppo, et al., 2003; Sabapathy, et al., 2005). Ultimately, both scenarios reflect increasing metabolic costs. Reducing the amplitude of the slow component should therefore reflect a more stable metabolic response and result in increases in exercise performance.

One of the primary and most often researched manners to achieve improved oxygen kinetics via a reduced slow component is prior exercise. It is thought that the reduction in the slow component is due to increased oxygen extraction at the muscle, possibly a consequence of increased enzyme function. Several studies confirm that prior
exercise can reduce the slow component, but not the MRT, of a subsequent bout of exercise (Bailey, et al., 2009; Burnley, et al., 2002; Burnley, et al., 2000; Jones, Koppo, et al., 2003; Poole, et al., 2007). However, very few studies have been conducted concerning how these findings apply to exercise performance (Jones, Koppo, et al., 2003).

As previously mentioned, Bailey’s 2009 study showed significant gains (15-30% in TTE) in performance after prior exercise. Of the six experimental conditions, varying between high (40%Δ) and severe (70%Δ) intensity and among 3, 9 and 20 minute recoveries, Bailey demonstrated a significant reduction in the slow component in four of the conditions: the high intensity warm up followed by a 3 minute recovery, and all three conditions for the severe intensity warm up. In spite of this, only two of the groups showed significant improvements in TTE. The severe intensity warm up followed by a 9 and 20 minute recovery showed 15% and 30% improvements, respectively in the TTE at an intensity of 80%Δ. Furthermore, the condition with the greatest reduction in the oxygen slow component amplitude (i.e., most improved oxygen kinetics) was the severe intensity warm up followed by a 3 minute recovery, yet it had the worst TTE, resulting in decrements below that of control. This suggests the relationship between exercise performance and the oxygen slow component is not linear and that there may be more physiological processes involved (Bailey, et al., 2009).

While oxygen kinetics is an important physiological measure, it merely provides a window into whole body oxygen demand.
**Metabolism**

Metabolism at the onset of exercise can be to an extent, graphically represented by the VO$_2$ curve. The constituent phases, and their subtleties in rate and mathematical descriptions primarily show how oxygen is being utilized by the body. The mismatch between the required oxygen consumption for a given work rate and the actual oxygen consumption is termed the oxygen deficit. It represents the amount of energy provided by anaerobic pathways at the onset of exercise, buffering the muscles’ aerobic contribution from mitochondrial oxidation. Therefore, a shift in the curve toward a more aerobic reliance may well indicate a shift in metabolism toward aerobic pathways.

The significant changes in the oxygen curve observed by Bailey may be due in part to blood lactate concentration. Some research proposes that the improvement in the VO$_2$ response to exercise is due to an alleviation of oxygen delivery and extraction limitations. This is thought to be facilitated by the accumulation of metabolites, namely lactate, in the blood. The drop in pH associated with increasing blood lactate levels is thought to cause vasodilation, thus improving blood delivery to exercising muscles and effectively eliminating any tissue perfusion heterogeneity. This would allow greater oxygen delivery to the muscle. Furthermore, the metabolic acidosis associated with increasing blood lactate levels may also cause a shift in the oxy-hemoglobin dissociation curve. Known as the Bohr Effect, this would allow more oxygen to be unloaded from the hemoglobin for a given partial pressure of oxygen in the blood (Bailey, et al., 2009; Jones, Koppo, et al., 2003; Poole, et al., 2007).
Jones reviewed the literature on the effects of prior exercise on metabolic and gas exchange responses to exercise. It was concluded that there was a weak relationship among blood lactate concentrations, improved performance and oxygen kinetics. Firstly, similar changes in oxygen kinetics can be elicited by both high (6.4mM) and low (3.4mM) initial blood lactate concentrations. Secondly, a reduction in the VO$_2$ slow component (an improvement in oxygen kinetics), can occur without an increase in initial blood lactate concentrations. Lastly, improvements in oxygen kinetics can persist, even after muscle pH and blood lactate levels return to baseline. This suggests that while often associated with one another, the relationship among blood lactate, improved oxygen kinetics and improved performance is not causative (Jones, Koppo, et al., 2003).

Though residual acidosis and the Bohr Effect have been demonstrated to improve oxygen extraction after a bout of heavy exercise, it is not closely tied to improved performance. Like the relationship between improved oxygen kinetics and performance, TTE improvements were absent in the presence of increased oxygen extraction (Bailey, et al., 2009). Additionally, improved oxygen extraction is presumed to be due to increased oxidative enzyme activity. However because oxidative enzyme activity, like that of pyruvate dehydrogenase (PDH), has been demonstrated to recover within approximately 4 minutes after heavy exercise, the theory that warm ups prime metabolic enzymes for subsequent high intensity activity falls short. In Bailey’s study, the greatest improvements in TTE after heavy and severe warm ups occurred in protocols utilizing 9 and 20 minute recoveries. With these significantly longer time frames for recovery, PDH activity would have most likely returned to baseline levels, theoretically negating any
priming from a warm up and dissociating enzyme activity from improved performance (Bailey, et al., 2009).

Beyond blood lactate levels functioning as a mechanism for improved performance, they provide insight into metabolism. In several warm up studies, blood lactate was measured immediately prior to the criterion bout of exercise (e.g., TTE or time trial) and at the end of the bout, having been preceded by varying types of warm up and recovery durations. The high intensity warm ups caused an increased blood lactate concentration at the onset of the criterion bout over control groups with no warm up. At the end of the criterion bouts, the blood lactate concentrations across the conditions were not significantly different. This demonstrates a blunted blood lactate response, where both the total change in and rate of blood lactate accumulation was significantly reduced over the respective control groups. This implies that either lactate production decreased, lactate clearance increased, or a combination of both occurred (Bailey, et al., 2009; Burnley, et al., 2005; Wittekind & Beneke, 2009).

Decreased lactate production may suggest the possibility of a decreased reliance on anaerobic energy sources, allowing the relatively finite amount of anaerobic energy to be utilized over a longer period of time. Increased clearance may suggest increased lactate utilization by organs that can oxidize it such as the brain, heart, liver and slow twitch muscle fibers (Bailey, et al., 2009; Shulman, 2005; Wittekind & Beneke, 2009). Furthermore, Brooks’ Lactate Shuttle Theory suggests a unification of the two processes, where lactate is moved from where it is synthesized in fast twitch fibers to slow twitch fibers where it can be oxidized (Brooks et al., 1991).
Therefore, the behavior of lactate may not be a mechanism in itself, but its decreased rate of accumulation due to decreased synthesis and increased clearance in muscle fibers may point to changes in motor unit recruitment.

Motor Unit Recruitment

Motor unit recruitment and more specifically, fiber type recruitment may provide a possible explanation for the blunted lactate response as well as the characteristic decreased VO$_2$ slow component of prior exercise studies. Given the previously mentioned evidence on lactate production, clearance, shuttling and its relation to fiber types, the relationship between motor unit recruitment and oxygen kinetics should also be examined. The appearance of the VO$_2$ slow component has been ascribed to the progressive recruitment of fast twitch or type II muscle fibers. Theoretically, the less aerobically efficient type II fibers cause a slow drift in oxygen consumption (the slow component) at the onset of exercise, requiring increased dependence on substrate level phosphorylation to compensate for the oxygen deficit (Burnley, et al., 2002; Jones, Koppo, et al., 2003). Recent research by Sabapathy has attempted to support this notion by measuring plasma ammonia (NH$_3$) concentrations (Sabapathy, et al., 2005).

Plasma NH$_3$ concentrations are thought to be directly related to type II fiber use and recruitment. Plasma NH$_3$ is a by-product of the deamination of adenosine monophosphate (AMP) in the purine nucleotide cycle. AMP itself is a by-product of maintaining the ATP/adenosine diphosphate (ADP) ratio during high rates of ATP use. However, an increase in AMP concentration in the muscle is never truly observed because of its deamination into NH$_3$ and inosine monophosphate (IMP) by AMP
deaminase. Type II fibers have a greater amounts of AMP deaminase activity— and therefore ability to produce NH₃— than type I fibers. The expectation would then be that a correlation exists between plasma NH₃ and the slow component (Sabapathy, et al., 2005).

Sabapathy’s study consisted of 7 minutes of constant, heavy load (50%Δ) cycling, expired gas analysis, plasma NH₃ measurements and iEMG. Across the eight subjects, plasma NH₃ significantly increased at the onset of the VO₂ slow component by 32.2 ± 2.9 μmol·L⁻¹. Moreover, the increase in plasma NH₃ from 3 minutes (just after the onset of the slow component) to 7 minutes was significantly correlated with the rise of the slow component (r = 0.79) as well as the amplitude of the slow component (r = 0.87). The mean power frequency (MPF) of the iEMG also increased significantly from the 3 minutes onward by 5.4 ± 0.8%. The iEMG increased significantly throughout the constant, heavy load cycling— abruptly through the first minute followed by a slower increase until 7 minutes. Though plasma NH₃ and the slow component share a correlation, the relationship cannot be considered causal. That relationship, taken together with the iEMG data and the characteristic ability of type II fibers to produce NH₃ suggests that the gradual and progressive recruitment of type II muscle fibers is the common factor relating plasma NH₃ and the VO₂ slow component (Sabapathy, et al., 2005).

With that relationship relatively established, if properly designed, studies that affect changes in VO₂ kinetics (specifically, the VO₂ slow component) can be related to muscle fiber type and motor unit recruitment. In Bailey’s aforementioned cycling study,
the iEMG data showed increased activity at the onset of the criterion bout in the 70-20-80 condition (the condition with the greatest improvement, 30%, in TTE) for the first 2 minutes. Additionally, the total change from baseline to termination was significantly less in this condition over control, demonstrating a blunted iEMG response. This blunted response—higher baselines followed by slower increases to the same terminal value—are also seen in the VO$_2$ response (increased Phase II amplitude followed by a slow component) and lactate response (Bailey, et al., 2009).

A previous study by Burhley demonstrated the same phenomenon. iEMG activity was increased by 19% at the beginning of the second of two bouts of severe cycle exercise. The higher initial increase was followed by a blunted increase in activity. The change in iEMG activity was not accompanied by changes in mean power, suggesting that the tension generated by each individual fiber was decreased, reducing the metabolic disturbance by spreading the metabolic load across more fibers, all reflected by the increased VO$_2$ response at the onset of exercise (increased Phase II amplitude, decreased slow component). Considering the concurrency of these events, a high intensity warm up or bout of prior exercise may cause increased motor unit recruitment and possibly a shift to a heavier reliance on type I fibers, all manifested by an increased VO$_2$ response and blunted lactate response (Burnley, et al., 2002).

While these studies intertwine oxygen kinetics with motor unit and even fiber type recruitment, the results from Bailey’s study relate all three to prior exercise and performance advantages of up to 30% improvement in TTE tests. But the addition of
motor unit and fiber type recruitment raises more questions regarding the underlying mechanism.

Central and Peripheral Fatigue

The increase in motor unit recruitment can implicate the perceptions of central and peripheral fatigue as well as the concept of central motor drive, yet another area of research that may explain the beneficial mechanisms of warm up.

Noakes proposed in a series of articles that the human body is governed by central processes in a feed forward mechanism, while also interpreting feedback information from different body systems (Noakes, et al., 2004). This feed forward mechanism of neural output from the central nervous system to the muscles would essentially constitute the concept of central motor drive (CMD). It is continuously adjusted throughout self-paced exercise based on the central nervous system, prior knowledge, afferent feedback and potentially several other variables. Therefore, fatigue becomes an interpretation of many central nervous system events processing subconscious variables and no longer a physical event defined by specific physiological measures. Additionally, the sensations of fatigue and exhaustion no longer represent catastrophic failures of physiological systems. Rather, they represent end points initiated by a central governor that precede catastrophic failure so as to preserve homeostasis (Noakes, et al., 2004, 2005).

Interestingly, if the central nervous system could be influenced to perceive and process the sensations of fatigue or exhaustion differently, could what is currently known as maximal effort or performance be redefined? Koltyn conducted a review of “Analgesia Following Exercise” in 2000, detailing several studies that delve into the capabilities of
exercise to influence the perception of pain (Koltyn, 2000). Several years later in 2006, Koltyn and Umeda conducted another review of “Exercise, Hypoalgesia and Blood Pressure” to a similar end, this time including blood pressure as a possible mechanism (Koltyn & Umeda, 2006).

A recent study by Amann highlights the possible benefits of altering opioid-mediated muscle afferents. When these afferents were blocked by intrathecal fentanyl, CMD as represented by iEMG was increased during the first half of a 5 kilometer cycle ergometer time trial, resulting in a significantly faster first half but relatively unchanged second half and final time. Though not exactly a performance benefit, it demonstrates the critical role muscle afferents place in self-pacing and therefore CMD (Amann, Proctor, Sebranek, Pegelow, & Dempsey, 2008).

Other studies also attempt to clarify the alleged “runner’s high” in an attempt to understand human fatigue more fully, whether it be due to opioidergic mechanisms (Boecker et al., 2008) or endocannabinoid systems (Sparling, Giuffrida, Piomelli, Rosskopf, & Dietrich, 2003).

Regardless of the avenue of research, the concept of CMD and perception of fatigue and their relationship to exercise performance is a relatively new field with few answers. However, within it may be the answer to the elusive mechanisms that underpin the beneficial practice of warming up.

Cycle Ergometry and Treadmill Running

Furthermore, the modality of exercise employed in this area of research is equally important, not only for real world applicability, but also for understanding the potential
mechanisms and processes involved. While the majority of studies looking specifically at warm ups, oxygen kinetics, exercise performance and those mentioned thus far have been done with cycle ergometry, very few have used treadmill running as the primary mode of exercise (Jones, et al., 2008). As evidenced by a few authors, the inherent difference in neural recruitment patterns between running and cycling may explain the dissimilarity in oxygen kinetics and further expound on the mechanisms of warming up.

One such study attempted to explicate the similarities and differences between the oxygen kinetics of cycling and running at the onset of exercise (Carter, et al., 2000). Carter states that most research has observed a decreased slow component amplitude in running, while other research questions the existence of the slow component entirely. Carter’s own research affirmed the former—that running oxygen kinetics displayed a smaller slow component amplitude relative to cycling at intensities greater than 50%Δ. Carter proposed that this difference in the slow component is due to fundamental differences in the modes of exercise, asserting that the greater degree of eccentric contractions in running exploits the elasticity of muscle. Because more elastic energy is stored in and returned from the muscle, individual muscle fibers have the potential to perform at relatively lower intensities, which may also delay or reduce the recruitment of type II muscle fibers. Conversely, the greater slow component observed in cycling was attributed to the relatively greater recruitment of type II fibers as compared with running, supporting the muscle activity and recruitment theory as a possible primary mechanism (Carter, et al., 2000; Wittekind & Beneke, 2009).
Billat’s 1998 study also espouses a similar viewpoint. Using ten triathletes performing both cycle ergometry and treadmill running to exhaustion at power outputs and speeds corresponding to 90% of VO₂ max, she found that the slow component was significantly lower during running when compared to cycling (20.9 ± 2 v. 268.8 ± 24 mL/min). The relatively large difference suggests that the presence and magnitude of the slow component is dependent on the mode of exercise and cannot be due to training status (hence the use of triathletes well trained in both disciplines) or differences in fiber composition (within subject design). As such, the fundamental differences between the modes may help explain the disparate oxygen kinetics. Billat suggests that the respective contraction regimen of each mode is responsible for the difference, stating that anywhere from 40% to 60% of the energy output for running can come from elastic energy stored in the muscle from eccentric contractions (Billat, et al., 1998).

Jones in his 2008 running study advocates the same viewpoints as both Carter and Billat concerning the decreased slow component in running. Interestingly, while his study is not performance oriented but rather strictly looks at the oxygen kinetics in primed and unprimed conditions, he demonstrated no improvement in oxygen kinetics after priming exercise. He speculates that improvement after priming exercise depends on the characteristics of the unprimed condition. If the oxygen kinetics are already fast (i.e., a minimized slow component) in the unprimed condition, further improvement may not be possible. Conversely, if the oxygen kinetics are relatively slow like in cycling, the effect of warming up may be more apparent (Jones, et al., 2008).
Nevertheless, the discrepancies between modes of exercise emphasize the deficiencies in the body of literature surrounding the interrelationship among warm ups, oxygen kinetics, physiological responses and performance benefits.

Summary

There is no clear consensus for how to structure warm ups, nor is there any clear understanding of its mechanisms. Bailey’s study has demonstrated the ability of a specifically structured warm up to elicit relatively large performance gains through the manipulation of several parameters. His study posits muscle motor unit recruitment and to some degree, oxygen kinetics as the underlying mechanisms. The results of his study explicate more clearly the relationship among exercise performance, oxygen kinetics, metabolism and motor unit recruitment while simultaneously opening the door to other approaches. Other research has focused on the endogenous opioid systems following exercise and their ability to alter perceptions of fatigue and central motor drive. Even so, the novelty of Bailey’s study and the majority of priming exercise research have been restricted to cycling and have yet to be replicated using other modalities of exercise.
Chapter 3
METHODOLOGY

The purpose of this research was to examine the effect of warm up intensity on subsequent time to exhaustion running performance in competitive distance runners by applying the findings and some design elements of Bailey’s study (Bailey, et al., 2009). The measured dependent variables included oxygen uptake (VO\textsubscript{2}), accumulated oxygen deficit (AOD), time to exhaustion (TTE), blood lactate concentration and rating of perceived exertion (RPE) across three independent conditions: control (no warm up), moderate intensity warm up, and high intensity warm up.

Subjects

Ten (one female) volunteer, well-trained runners actively competing and with a minimum 2 year history of competition were used for this study after completing an informed consent form detailing the anticipated procedures, benefits and risks. Subjects were 23.1 ± 5.3 years of age, 177.5 ± 8.3 centimeters in height, 65.0 ± 8.4 kilograms in body mass, with an average VO\textsubscript{2} max of 68.07 ± 4.46 mL·kg\textsuperscript{-1}·min\textsuperscript{-1}, training competitively for 7.7 ± 3.4 years.

To ensure proper subject treatment, the study passed through the Institutional Review Board at California State University, Sacramento. Subjects completed a medical history and screening form based off the American College of Sports Medicine (ACSM) guidelines so as to minimize the risk of cardiovascular complication throughout the duration of the study. Any subject classified higher than “low” by ACSM standards or presently affected by illness or injury was excluded. Subjects were recruited from
running clubs, schools and teams in the greater Sacramento area. No compensation, monetary or otherwise, was offered to the subjects for their participation.

Experimental Design

The study was a within subject control, repeated measures design. Each subject underwent each of the three conditions for the dependent variables. This design was chosen to minimize variability among subjects and to test the effects of warm ups on exercise performance. After the initial screening, all subjects were instructed to arrive to the laboratory for baseline VO₂ max testing fully rested & fully hydrated, three hours postprandial, and having avoided caffeine and alcohol 24 hours prior. All subsequent visits and tests were completed at least 48 hours later around the same time of day to minimize circadian rhythm influence (± 2 hours). The subjects also underwent a submaximal exercise test, the data from which was used to calculate theoretical oxygen costs and AOD. Each subject then completed all three conditions in a randomized order. This randomization functioned to reduce any learned effect from the ordering of the conditions. The control condition consisted of no warm up, 20 minute recovery and a TTE test, whereas the experimental conditions consisted of either a moderate intensity or high intensity warm up, 20 minute recovery and TTE test.

All exercise tests (baseline VO₂ max, submaximal tests, control and experimental conditions, TTE tests) were performed on a 425CP laboratory treadmill (Trackmaster, Kansas, United States). Gas exchange variables, heart rate (HR), and RPE were recorded throughout baseline VO₂ max testing, submaximal testing and TTE testing. Gas exchange variables were collected and displayed by a TrueOne 2400 metabolic cart (ParvoMedics,
Utah, United States). The cart was calibrated with gases of known concentration and a standard range of flow rates using a 3 liter calibration syringe (Hans Rudolph, Kansas, United States). Each subject wore a nose clip and ventilated through a Han Rudolph 2700B two-way valve mask secured by adjustable head gear (Hans Rudolph, Kansas, United States). Expired gas was directed by a 6-foot expired gas hose to a spirometer and mixing chamber. HR and RPE were collected by a heart rate monitor strapped to the subject’s chest (Polar Electro, Finland) and visual Borg RPE scale posted in front of the subject, respectively. Blood lactate was measured using a Lactate Pro test meter (h/p/cosmos sports & gmbh, Traunstein, Germany). Total time for each TTE test was recorded with a stop watch.

**Procedures**

*Baseline VO₂ Max Testing*

Baseline testing was used to assess the gas exchange threshold (GET) and VO₂ max. This was achieved by an incremental, velocity-based running protocol with a constant grade of 1.5% (Jones & Doust, 1996; Wittekind & Beneke, 2009) on a laboratory treadmill. The protocol was designed to increase speed every three minutes whereby the fourth stage a self-reported 5000 meter personal record pace was achieved (minutes 9 to 12).

Gas exchange variables, HR and RPE were recorded throughout baseline VO₂ max testing. From these initial data, the GET and VO₂ max were determined for each subject. These two markers later served as determinants for exercise intensity (Bailey, et al., 2009). GET was defined as an increase in the ventilatory equivalent for oxygen
(VE/VO₂) without an increase in the ventilatory equivalent for carbon dioxide (VE/VCO₂) with respect to VO₂. At least two of the following three criteria were used to define VO₂ max: less than 2 ml·kg⁻¹·min⁻¹ increase in VO₂ across an increase in workload (plateau), RPE greater than or equal to 17, and an RER greater than or equal to 1.05.

Submaximal Testing

Submaximal testing was used to calculate the theoretical oxygen cost for the high intensity TTE, which is essential for calculating AOD. This was achieved by performing six consecutive stages, all below the GET. Each stage was of sufficient duration to allow for one minute of steady state VO₂ (SS VO₂). Once one minute of SS VO₂ was achieved, the intensity was increased and the process was repeated until six stages had been completed.

Gas exchange variables and HR were recorded throughout submaximal testing. From these initial data, a regression equation of oxygen costs and running speed was developed for each runner.

Control

A 3 minute low intensity jog at a speed equivalent to 40% of VO₂ max speed preceded the 6 minute period of no warm up. The period of no warm up consisted of standing or sitting. Blood lactate was measured immediately post warm up.
Moderate Intensity Warm Up

A 3 minute low intensity jog at a speed equivalent to 40% of VO₂ max speed preceded the 6 minute moderate intensity warm up. The 6 minute moderate intensity warm up was conducted at a treadmill speed equivalent to the GET speed. Blood lactate was measured immediately post warm up.

High Intensity Warm Up

A 3 minute low intensity jog at a speed equivalent to 40% of VO₂ max speed preceded the 6 minute high intensity warm up. The 6 minute high intensity warm up was conducted at a treadmill speed equivalent to 70% of the difference in speed between GET and VO₂ max (70%Δ) (Bailey, et al., 2009). Blood lactate was measured immediately post warm up.

20 Minute Recovery

The recovery period was 20 minutes in duration. The subject remained in a rested condition for the first 17 minutes. At the start of the 18th minute, the subject then underwent 3 minutes of low intensity jogging at a speed equivalent to 40% of VO₂ max speed, before an abrupt step transition to the TTE intensity. Additionally, expired gas analysis was started and continued uninterrupted to the end of the TTE. Blood lactate was measured via fingertip blood sample immediately before the TTE.

Time to Exhaustion Test

Expired gas measures, HR and RPE were continuously monitored throughout the TTE test. HR and RPE were recorded every minute. TTE, RPE and blood lactate were recorded at exhaustion. The abrupt step transition to a treadmill speed equivalent to VO₂
max speed occurred immediately after the recovery, initiating the TTE test. Both the speed and a grade of 1.5% were held constant throughout the test. The subject was instructed to run until exhaustion. The time and speed were blinded from the subject throughout the test. The bout was terminated when the subject signaled for termination or failed to keep pace with the treadmill.

Data Analysis

AOD was determined for each condition by comparing the actual oxygen cost of the TTE test to the theoretical oxygen cost. The theoretical oxygen cost was determined using the data from the six submaximal stages. The steady state oxygen cost of each submaximal stage was plotted against work rate. The data points were then extrapolated to the TTE work rate (VO\(_2\) max), providing the theoretical oxygen cost at the given intensity. The actual oxygen cost of the TTE test was measured at 15 second increments for the first three minutes of the TTE test and subtracted from the theoretical cost. The resultant value was termed the AOD (theoretical oxygen cost – actual oxygen cost = AOD). Maximum accumulated oxygen deficit (MAOD) was calculated by summing the 15 second AOD values throughout the entire TTE bout.

The 15 second AOD values from each condition were time matched up to the same three minute point during the TTE test. This provided insight into the uptake and usage of oxygen at the onset of heavy exercise after each of the three independent conditions. The MAOD provided insight into the total oxygen contribution during the TTE bout.
Statistical Analysis

The independent variables were the control condition of no warm up and the experimental conditions of a moderate and high intensity warm up. The dependent variables were the exercise measures taken before, during and after the TTE. The continuously measured VO\textsubscript{2} data were used to compare the VO\textsubscript{2} after warming up, at the onset and end of the TTE, as well as calculate AOD.

All data were exported to Excel (Microsoft, Washington, United States) and STATISTICA (StatSoft, Oklahoma, United States) for statistical analysis.

A repeated-measures analysis of variance (ANOVA) was used to compare the means of TTE, VO\textsubscript{2}, AOD, blood lactate, and RPE with the corresponding experimental or control condition. In the event of significant difference, Scheffé’s test was applied to ascertain the source of the difference. Statistical significance was accepted at P < 0.05 (Bailey, et al., 2009).
Chapter 4

RESULTS

The purpose of this investigation was to examine the effect of warm up intensity on subsequent time to exhaustion running performance, oxygen uptake, metabolism and perceived exertion in competitive distance runners. Ten healthy (nine males and one female), well trained subjects, volunteered to participate in this study (mean ± SD: 23.1 ± 5.3 years of age; 177.5 ± 8.3 centimeters in height; 65.0 ± 8.4 kilograms in body mass; VO$_2$ max of 68.07 ± 4.46 mL·kg$^{-1}$·min$^{-1}$; 7.7 ± 3.4 years of competitive training). All testing was conducted at the I.E. Faria Exercise Physiology Research Laboratory (Sacramento, CA).

Time to Exhaustion

Figure 4.1 shows the mean TTE values in seconds across the three warm up conditions. Figures 4.2 and 4.3 graphically display the change in TTE for each individual, as well as the group mean, across all conditions. Statistical analysis revealed a significant effect of warm up intensity on TTE. Post hoc testing detected a significant mean increase in TTE in the moderate intensity group over the control group (287.7 ± 56.5 s v. 236.7 ± 64.9 s, 21% increase, P = 0.006430). No other significant relationships among the conditions existed. However, there was a trend towards significance in the high intensity group over the control group (270.5 ± 46.3 s v. 236.7 ± 64.9 s, 14% increase, P = 0.076644).
Figure 4.1. Group mean times to exhaustion in the control, moderate intensity & high intensity warm up conditions. *Significantly different from control (P < 0.006430).
Figure 4.2. Individual (dashed lines) and group mean (solid line) times to exhaustion in the moderate intensity condition versus the control condition. *Significantly different from control (P = 0.006430).
Figure 4.3. Individual (dashed lines) and group mean (solid line) times to exhaustion in the high intensity condition versus the control condition.
Oxygen Uptake

Figure 4.4 shows the group mean VO\textsubscript{2} values in mL·kg\textsuperscript{-1}·min\textsuperscript{-1} every 15 seconds for the first three minutes of the TTE bout across the three warm up conditions. Analysis indicated no statistical difference in either the VO\textsubscript{2} at the onset or end of the TTE testing among the three different treatments (P = 0.49836). As expected, VO\textsubscript{2} increased significantly with respect to time (P = 0.0000), attaining a mean VO\textsubscript{2} end value within one standard deviation of the mean VO\textsubscript{2} max.

\textbf{Figure 4.4.} 15 second group means for VO\textsubscript{2} during the first 3 minutes and the final value reached at the end of the TTE test.
Accumulated Oxygen Deficit

Figure 4.5 shows the 15 second group mean AOD values for the first three minutes of TTE testing. There was no significant effect of warm up condition on AOD during the first three minutes of a TTE bout (P =0.82130). As expected, AOD significantly decreased with respect to time. Additionally, the average MAOD calculated at the end of the TTE bout was not significantly different across the three warm up conditions.

*Figure 4.5. 15 second group means for AOD during the first 3 minutes and the maximum accumulated value (MAOD) at the end of TTE test.*
Blood Lactate

Figure 4.6 shows the mean blood lactate concentrations immediately post warm up, prior to and immediately post TTE across the three conditions. There was a significant effect of warm up on blood lactate, with post hoc analysis demonstrating a mean increase in blood lactate concentration immediately post high intensity warm up when compared to the control and moderate conditions (P = 0.000000 & 0.000029, respectively). After the warm up recovery period there was no significant difference in either the pre or post TTE blood lactate values among the three conditions. The post high intensity warm up lactate value was not statistically different than the post TTE lactate value in the control (P = 0.671297), moderate (P = 0.942784) or high (P = 0.276554) conditions. Furthermore, there was no significant difference in the pre TTE blood lactate values among the three conditions.
Blood Lactate Concentration

\[ p = 0.00004 \]

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Control
Low
High

*, **

Figure 4.6. Post warm up, pre time to exhaustion & post time to exhaustion group mean blood lactate concentrations for the control, moderate intensity & high intensity conditions. *Significantly different from control (\( P = 0.000000 \)). **Significantly different from moderate intensity (\( P = 0.000029 \)).
Perceived Exertion

Figure 4.7 shows the mean RPE values during the first three minutes of TTE testing, as well as the final rating at exhaustion for all three warm up conditions. Analysis revealed no significant effect of warm up condition on RPE during the onset and at the end of the TTE bout ($P = 0.33333$). As expected, RPE increased significantly with respect to time.

Figure 4.7. 1 minute RPE values during the first 3 minutes and the final value reached at the end of the TTE.
Based on the results of this research, hypothesis number one (there will be no significant difference in TTE among the groups) was rejected. Hypothesis number three (there will be no significant difference in blood lactate concentrations post warm up, pre TTE and post TTE among the groups) was also rejected. Hypotheses number two (there will be no significant difference in oxygen kinetics during the first 3 minutes and at the end of the TTE as described by VO$_2$ and AOD values among the groups) and number four (there will be no significant difference in RPE during the first 3 minutes and at the end of TTE among the groups) were both accepted.
Chapter 5

DISCUSSION

The primary finding of this research was a moderate intensity warm up significantly increased TTE nearly 21% in comparison to a control condition of no warm up. Similarly, a high intensity warm up produced a non-significant improvement in TTE by nearly 14% over control. None of the warm up protocols had significant effects on VO₂, AOD or RPE. However, blood lactate concentration was significantly elevated immediately post high intensity warm up over both the control and moderate intensity conditions.

Physiological Responses to Warming Up

Oxygen Kinetics

In the current study, oxygen kinetics was represented by 15 second VO₂ averages over the first three minutes of the TTE bout, as well as the final value reached at the end. Additionally, AOD and MAOD were also calculated to further characterize the VO₂ response, using 15 second deficit averages over the first three minutes of TTE, as well as the total accumulated throughout the entire TTE bout. For these variables, no significant changes were elicited by any of the three conditions, even in the moderate intensity warm up condition where TTE was significantly improved.

Because no significant differences in values in either the VO₂ or AOD were observed, especially in the moderate intensity group, the oxygen kinetics as measured by the current investigation were relatively unchanged. The lack of significant difference in VO₂ and AOD during the first 3 minutes and at the end of TTE suggests no real change in
oxygen uptake kinetics. Rather, the subjects may simply be able to sustain actual VO\(_2\max\) oxygen consumption for a longer period of time.

This has certain implications for both the MRT and VO\(_2\) slow component. Previous research suggests that decreasing the slow component, not the MRT, beneficially affects exercise performance (Bailey, et al., 2009; Burnley, et al., 2002; Burnley, et al., 2000; Jones, Koppo, et al., 2003; Poole, et al., 2007). With no significant differences in AOD or VO\(_2\) across time, especially in the moderate intensity group, the current investigation does not support changes in oxygen kinetics as the primary mechanism that aids high intensity TTE efforts.

This observation may be due two main factors: modality and intensity. An important difference between this study and the majority of the literature is the chosen exercise modality. Authors Carter and Billat have demonstrated significantly different oxygen uptake responses to exercise between running (this study’s choice of modality) and cycling (the choice of modality for majority of the aforementioned research). Both authors observed a decrease in or a near absence of a slow component in running when compared to cycling at the same intensity. They postulate that the apparent dissimilarity is due to the fundamental differences in movement patterns between running and cycling—particularly the higher amount of eccentric contraction present in running when compared to cycling. Ultimately, more energy can be stored and returned in running simply due to its mechanics, which may explain the oxygen uptake response at the onset of exercise (Billat, et al., 1998; Carter, et al., 2000).
Furthermore, evidence exists that improved oxygen kinetics from priming or warming up is dependent on the unprimed condition’s (control) oxygen kinetics during the criterion bout (e.g., TTE). Jones theorizes that if the kinetics are already fast (a minimized slow component, such as the case with running, especially when compared to cycling) in the unprimed criterion bout, further improvement may not be possible regardless of any priming exercise. Conversely, if the kinetics are relatively slow (such as in cycling when compared to running), the effect of warming up on oxygen kinetics may be more evident (Jones, et al., 2008).

Similarly, the intensity of the criterion bout cannot be overlooked. While the majority of previous studies consulted in the current investigation used submaximal intensities (i.e., <100% VO₂ max), this study used 100% VO₂ max as the target intensity for the TTE. Because the TTE is conducted at a velocity designed to elicit maximal oxygen consumption, it acts as a ceiling within the TTE bout. This relatively high intensity may also increase the demand of oxygen at the onset of exercise such that its uptake is accelerated when compared to lower intensities. As depicted by the oxygen uptake curve (Figure 4.4), the subjects approached VO₂ max in less than 2 minutes during the TTE bout.

The maximal effort may decrease the difference between Phase II (the monoexponential increase in VO₂) and Phase III (steady state VO₂) of oxygen uptake at the onset of exercise by increasing the Phase II amplitude. In effect, this reduces the slow component amplitude resulting in inherently fast oxygen kinetics. Considering this acceleration of oxygen kinetics due to intensity in the unprimed condition, as well as
Jones’ argument concerning modality, priming exercise may not result in improved oxygen kinetics (Jones, et al., 2008).

Therefore, whether by modality, intensity or a combination of both, the oxygen kinetics of the TTE bout in this study may have been intrinsically fast. This would result in a considerably diminished opportunity to improve oxygen kinetics with priming exercise or warm up conditions. Although the current study lacks a measured improvement in oxygen kinetics in comparison to other studies (Bailey, et al., 2009; Burnley, et al., 2002; Burnley, et al., 2000; Jones, Koppo, et al., 2003; Poole, et al., 2007), it may indicate that the oxygen response to high intensity exercise may not be sufficient to explain the differences in exercise tolerance or performance (Jones, et al., 2008).

The above mentioned effects of warm up on oxygen kinetics and TTE with regard to modality and intensity are conjectural. Further investigations to delineate the role that oxygen kinetics plays in both warm ups and high intensity exercise are needed.

Another aspect that cannot be disregarded is the use of 15 second VO$_2$ averages. Unlike the majority of the previously discussed literature that uses breath-by-breath analyzers to quantify oxygen kinetics, the present study was limited by the available equipment, which was only capable of 15 second VO$_2$ averages. To supplement the VO$_2$ measurement, AOD was also calculated. In spite of this, both are essentially indirect measures of oxygen kinetics, and as such are fundamentally different from breath-by-breath analysis. However, the brief exercise duration and rapid VO$_2$ response elicited by the high intensity TTE may not warrant breath-by-breath analysis.
Blood Lactate

The only apparent effect of warm up on blood lactate values was immediately post warm up, where the high intensity warm up produced significantly higher concentrations than both the control and moderate intensity warm up conditions. Among the conditions, the remaining pre and post TTE sampling points yielded no significant differences. Despite the significant increase in lactate after the high intensity warm up, the recovery period of 20 minutes was sufficient to reduce these elevated lactate concentrations back down to the pre TTE values of the control and moderate intensity groups. Thus, the Bohr Effect may not be the considered as a primary mechanism to improved performance in the current investigation, despite the suggestions of prior research (Bailey, et al., 2009; Jones, Koppo, et al., 2003; Poole, et al., 2007). Furthermore the warm up condition did not alter or augment the post TTE values. These findings do however support the assertions of previous reviews that the correlation between improved exercise performance and elevated baseline blood lactate concentrations is weak (Jones, Koppo, et al., 2003).

The lack of a significant difference in both pre and post TTE concentrations despite the difference in TTE duration indicates a relatively constant absolute change in pre and post TTE values among all three conditions. Notably, the moderate intensity warm up condition had a significantly longer TTE over the control and high intensity conditions. Because of this, it can be inferred that although the absolute change in blood lactate concentration is essentially the same across all conditions, the rate of its accumulation is reduced in the moderate intensity condition because it occurs over a
longer time frame (i.e., significantly longer TTE). This observation can signify a decreased production of lactate, an increased clearance of lactate, or some combination of both, falling in line with previous research findings (Bailey, et al., 2009; Burnley, et al., 2005; Wittekind & Beneke, 2009).

The decreased production of lactate may be indicative of a decreased reliance on anaerobic energy sources. This sparing of anaerobic energy sources can allow that relatively finite amount of energy to be utilized over a longer time period, potentially contributing to the increased TTE observed in the moderate intensity condition. Conversely, the increased clearance of lactate can denote increased utilization of lactate as a substrate, particularly by slow twitch fibers, as suggested by previous research (Shulman, 2005; Wittekind & Beneke, 2009). The lack of change in the AOD and VO$_2$ suggests the energy contribution from nonoxidative sources was relatively unchanged, pointing towards a change in clearance rather than production. This remains speculative until future studies investigate lactate kinetics in the context of warming up and high intensity exercise.

**Perceived Exertion**

Although the various conditions yielded no significant effect on RPE during the first 3 minutes of TTE and the final value reached, it is important to note that this includes the moderate intensity warm up condition—the lone condition with a significant increase TTE duration. Much like the blood lactate findings, for there to be no significant difference in RPE initially and at the end of a TTE, despite the increased duration between measurements (significant increase in TTE), signifies a change in the rate of the
perception of exertion. While not synonymous, this alteration in the perception of exertion is closely tied to fatigue, and by extension improved performance.

This finding falls in line with Noakes’ idea of central motor drive (CMD), where instead of fatigue being defined by physiological markers and catastrophic failure, it becomes a central process that initiates an end point to CMD in response to the interpretation of several variables to preserve homeostasis (Noakes, et al., 2004, 2005). The argument then can be posited that if alterations to the perception of those variables (e.g., exertion) can be made, then performance can be enhanced by unleashing the CMD. This relationship is bolstered by Koltyn’s reviews of exercise altering perception of afferents, as well as Amann’s work with opioid-mediated muscle afferents (Amann, et al., 2008; Koltyn, 2000; Koltyn & Umeda, 2006). Therefore, while this current investigation lacks a true measurement of CMD, its use of RPE can indirectly approximate it, ultimately supporting the previously described literature.

When taken together, the responses of this study’s variables offer a possible explanation for an improved TTE after a moderate intensity warm up. Despite the lack of altered oxygen kinetics, the blunted lactate response and increased TTE may be explained in part by increased motor unit recruitment. Increased motor unit recruitment, as measured by iEMG, could explain the blunted lactate response, by increased slow twitch fiber recruitment. This would both decrease lactate production and increases its clearance (Brooks, et al., 1991; Shulman, 2005; Wittekind & Beneke, 2009).

Amann’s recent work provides a possible explanation for the increased TTE, by connecting increased iEMG (as a measure for CMD) to blunted afferent feedback (a
theoretical measure for perception) (Amann, et al., 2008). In that scenario, the increased motor unit recruitment would allow for the maintenance of VO$_2$ max power, increased clearance of blood lactate, caused by the alteration in perception of effort which effectively increases CMD. Because the current investigation does not fully address all of these variables, these proposed mechanisms are purely hypothetical. Future research should address these issues in order to expound on the mechanisms behind real world applications of warming up.

Conclusion

This study indicates that a moderate intensity warm up at the gas exchange threshold is the most effective for subsequent TTE exercise occurring at or near VO$_2$ max in a sample population of well trained, competitive runners. However, this neither precludes high intensity warm ups nor affirms moderate intensity warm ups. Recall that the improvement in TTE in the high intensity condition trended toward significance. Additionally the individual results for each subject indicate a high degree of inter- and intravariability regarding the response to the warm up conditions. While on average the moderate intensity warm up prolonged TTE significantly, on an individual basis three of the ten subjects actually improved TTE more with a high intensity warm up than with a moderate intensity warm up (Figures 4.2 and 4.3).

But with no statistically distinguishable difference in any of the measured physiological measures among the conditions, especially between the moderate and high intensity conditions, despite the significant increase in TTE following the moderate intensity warm up, the mechanism behind the observed improvement is still unknown.
This apparent dissociation of physiological markers with exercise performance only further emphasizes the need for additional research that explores the effects of warming up on not just physiological measures like those included in this study, but also on neurological aspects that were not utilized such as CMD, iEMG and perception.
APPENDICES
APPENDIX A

Informed Consent
INFORMED CONSENT
Human Performance Research Laboratory
California State University, Sacramento

Kevin Dinglasan, BS ATC is an exercise science graduate student and Roberto Quintana, PhD is a professor at California State University, Sacramento (CSUS). Both are interested in conducting research that studies “The Effect of Warm Up Intensity on Time to Exhaustion at VO$_2$ Max in Runners.” You were selected as a possible participant for this study because of your current health status and your interests in participating in this research.

Purpose of the Research
The goal of this research is observe the effect of a moderate & high intensity warm up on the human body’s use of oxygen and performance during a race-like scenario.

Explanation of the Treatments and Tests to Be Administered
If you decide to participate, you are required to report to the Human Performance Research Laboratory (HPRL) at CSUS on several occasions to complete several tests. For the three days leading up to each of your visits, you will record what you eat as well as your exercise routine. There are five total visits, each of which is explained as follows:

A. Report to HPRL to fill out a medical history questionnaire and undergo a medical screening to determine whether you are suitable for enrollment in the study. Also during this time you will be able to become familiar with the procedures of the study and equipment that will be used. If you qualify for the study you will complete a maximum oxygen consumption treadmill running exercise test (VO$_2$ max). This treadmill test is an incremental running exercise test until you reach maximal fatigue. The test can last anywhere from 8 to 15 minutes. Your heart rate (HR), rating of perceived exertion (RPE), and expired gases will be measured every stage of the exercise test. The results of this test will determine the intensity at which you perform certain aspects of the remainder of the study.

B. Two days after this test, you will return to the lab for the second visit to undergo submaximal testing. This testing is less intense than VO$_2$ max testing. It requires that you perform six stages of low to moderate intensity exercise on a treadmill while your expired gases are measured. Each stage will last approximately 3 minutes.

C. Two days after the second visit you will return for the third visit. Visits 3 through 5 will be separated by five to seven days and require you to undergo one of the three conditions for warming up during each visit, in a randomized order. The three warm up conditions will be 6 minutes in duration and will either be control (no warm up), moderate intensity or high intensity. The warm up will be followed by a 20 minute recovery. You will then be placed on the treadmill to run at a speed that corresponds to your VO$_2$ max until you reach exhaustion. This high intensity time to exhaustion (TTE) test can last anywhere from 3-15 minutes depending on your level of fitness and requires you to maintain your VO$_2$ max as long as possible. The TTE test will be
terminated if you give the termination signal, or appear to fail to keep up with the treadmill.

D. Immediately after the warm up, and before and after the TTE test, your blood lactate will be measured by taking blood samples from the finger tip via a spring loaded, sterilized lancet. This will involve a momentary, sharp pain in the form of a pin prick. Throughout the test, your HR, RPE and expired gases will be measured. Your final time will also be recorded.

E. The total time commitment for the study will be approximately 5 visits of 1 hour each completed within three weeks.

Risks from Procedures
Exercise tests to the point of fatigue are associated with a risk of death (<0.01%) and complications with the heart (<0.1%) (i.e., irregular heart rhythm, inadequate blood to the heart, and heart attack). The risk of incidents occurring is much less for individuals who are young, exercise regularly, and are in good health. Completion of the questionnaire prior to the beginning of the study will help minimize the risks of any cardiac event. Also, associated with an exercise test of this nature are leg and breathing discomfort (100%), as well as increases in body temperature (100%). If any adverse reactions occur due to exercise testing, you will be referred to your personal physician or the CSUS Student Health Center if you are a student. In case of severe or acute signs and symptoms we will follow CSUS Guidelines for Emergencies and when necessary Adult CPR/automated external defibrillator procedures.

Responsibilities of the Participant
The information you provide about your health and well-being may affect the safety and value of your cardiorespiratory fitness and health evaluation. It is important to provide any unusual feelings/discomforts during the cardiorespiratory fitness and health evaluation. The accurate reporting of your health and unusual symptoms that you may experience during the evaluation will minimize the occurrence of any adverse events and aid in evaluating your health. Your full cooperation and adherence to directions are appreciated during all aspects of these procedures. All tests are voluntary and you may request to stop the procedures at any time.

Benefits of Participation
There will be no compensation, monetary or otherwise, provided to you for your participation. The benefits to yourself for participating in this study include knowledge of your maximal ability to consume oxygen, your maximal heart rate and the effect that warm ups have on your body before a race-like scenario. This information can be used to help optimize your training and understand your body’s response to exercise. Other than the items listed above, there may be no direct benefit to you.
Your Rights and Confidentiality
If you decide to participate, you are free to withdraw your consent and to stop participation at any time with no penalty to you. Any information which is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission. The data will be identified only with numeric codes, not the names of the participants.

Questions
If you have any questions, please feel free to call the primary researcher Kevin Dinglasan at (xxx) xxx-xxxx between 9am and 5pm, the major professor Roberto Quintana PhD at (916) 278-4495, or the Office of Research and Contract Administration at (916) 278-7565.

Statement of Permission
You are making a decision whether to participate or not to participate. Your signature indicates that you have decided to participate having read the information provided above. Your signature also affirms that the medical history you have provided is complete and true to the best of your knowledge. You will be given a copy of this form to keep. You understand that you will not receive any compensation for participating in this study.

I have read this form and I understand the test procedures that I will perform, as well as the attendant risks and discomforts. Having had the opportunity to ask questions that have been answered to my satisfaction, I consent to participate in this test.

_________________________  ____________________________
Date                                   Signature of Participant

_________________________  ____________________________
Date                                   Signature of Investigator
APPENDIX B

Subject Information & Medical History
SUBJECT INFORMATION AND MEDICAL HISTORY

Human Performance Research Laboratory
California State University, Sacramento

NAME: ___________________________________________ DATE: ______________
ADDRESS: ______________________________________ PHONE: ______________
________________________________________________ EMAIL: ______________

OCCUPATION: _________________________________
GENDER: M ___ F ___ AGE: ______ years DATE OF BIRTH: ______________
TOTAL CHOLESTEROL: _____ mg/dL HDL _____ mg/dL LDL _____ mg/dL
FASTING BLOOD GLUCOSE: __________ mg/dL Other blood results: __________

We will take the following 4 measurements (do not answer):
WEIGHT:________kg HEIGHT:________cm BP:_____/____mmHg HR:________beats/min

MEDICAL HISTORY: (Please Circle your Answers)
Are you currently taking any medications? Yes or No:
If yes, please list: ____________________________________________________________

Please list all medical conditions (e.g. ulcers, arthritis, mono, hepatitis, HIV, musculoskeletal injury)?
_____________________________________________________________________________

Do you have any of the following hospitalizations and/or surgeries?
_____________________________________________________________________________

Have you ever been diagnosed with a breathing problem such as asthma? Yes or No:
If yes, please explain: _________________________________________________________

Have you ever been diagnosed with a heart problem or condition? Yes or No:
If yes, please explain: _________________________________________________________

Do you have any of the following symptoms at rest or with low to moderate physical activity? Yes or No:
Lightheadedness Shortness of Breath Chest Pain Numbness
Fatigue Coughing Wheezing Other: ____________________________

Do you have any of following cardiovascular disease risk factors? Yes or No
Family History of Heart Attacks Hypertension High Cholesterol
Sedentary Lifestyle (refer to next page) Diabetes Current cigarette smoker
Obesity (Calculate BMI=________kg/m²)
If yes, please explain: _________________________________________________________
Do you have an immediate family member with any of the following diseases? Yes or No:

- Diabetes
- Hypertension
- High Cholesterol
- Obesity

If yes, please explain:

Are there any other conditions that might affect your health/exercise ability? Yes or No:

If yes, please explain:

**Training History**

What type of athlete are you? Please circle the best answer:

- A) Professional/National Class
- B) Competitive at Regional-Local level
- C) Age or Class Competitor
- D) Well Trained
- E) Other: ______________________

How many years have you been training competitively? ______________________

Over the last year, what has been your weekly mileage? ______________________

Over the last year, what percentage of your overall training is at a pace faster than “somewhat hard” or >70% of VO₂max? ______________________

What are your 3 best performances? (include date and event/course)

1: ______________________

2: ______________________

3: ______________________

Please give your best performance over the last 18 months (include date, time & course)

---

*These questions concern your training over the past 20 weeks:*

What is the average number of exercise sessions per week? ______________________

What is the average duration of your exercise sessions? ______________________

What is the average intensity of your exercise bouts? ______________________

Could you give us the respective volume of easy, moderate (“somewhat hard” or 70% VO₂max) and hard workouts (>“Hard” or 85% VO₂max) per week (miles per week)?

- Easy: _______ Moderate: _______ Hard: _______

What is the total volume of your workouts per week (miles per week)? ______________________

Any recent significant injuries which have limited your training? ______________________

---

**Additional Information:**

Have you ever performed a fitness or maximal exercise test? Yes or No:

If yes, what were the results of your tests?

Protocol: _______ VO₂ max: _______ Speed/Power: _______ Lactate Threshold: _______

Overall Interpretation: ______________________
Women Only:

Please indicate how many menstrual cycles you have had within the past 12 months: __________________

Are you taking oral contraceptives or estrogen replacement therapy? Yes or No:

If yes, indicate type & brand: __________________________________________________________

COMMENTS & OBSERVATIONS: ______________________________________________________

OVERALL RISK STRATIFICATION: ___________________________________________________

EXERCISE & EXERCISE TEST RECOMMENDATIONS: ________________________________
APPENDIX C

Diet & Exercise Log
# DIET & EXERCISE LOG

Human Performance Research Laboratory  
California State University, Sacramento

The following diet and exercise log will allow you to keep track of both what you eat and your exercise routine for the three days leading up to each of your five visits. It is important to try to minimize the variation of your diet & exercise during this time frame among each of your visits.

Remember to:
1. Abstain from strenuous or long bouts of exercise three days before your visit
2. Abstain from alcohol and caffeine 24 hours before your visit
3. Abstain from eating within 3 hours before your visit
4. Arrive to your visit well rested and well hydrated

**VISIT #1**

<table>
<thead>
<tr>
<th>Days Out</th>
<th>Time</th>
<th>Description of Meal (includes type &amp; amount/weight)</th>
<th>Time</th>
<th>Description of Exercise (includes type, distance/duration &amp; intensity)</th>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day of Visit</td>
<td>No eating within 3 hours before; arrive rested and hydrated</td>
<td></td>
<td></td>
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</table>
## DIET & EXERCISE LOG

**Human Performance Research Laboratory**

**California State University, Sacramento**

### VISIT #2

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<tr>
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<th>Time</th>
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<th>Time</th>
<th>Description of Exercise (include type, distance, duration &amp; intensity)</th>
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<td>No mention of long bout of exercise</td>
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<tr>
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<td></td>
<td>Day of Visit</td>
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<td>No eating within 3 hours before, well rested and hydrated</td>
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## DIET & EXERCISE LOG
Human Performance Research Laboratory
California State University, Sacramento

### VISIT #3

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<th>Time</th>
<th>Description of Exercise (include type, distance/duration &amp; intensity)</th>
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<td>No stress or long bouts of exercise</td>
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<td>No stress or long bouts of exercise; no alcohol or caffeine</td>
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<tr>
<td>Day of Visit</td>
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<td></td>
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<tr>
<td>No eating within 3 hours before; arrive rested and hydrated</td>
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# DIET & EXERCISE LOG

Human Performance Research Laboratory  
California State University, Sacramento

## VISIT #4

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<th>Description of Exercise (include type, distance, duration, &amp; intensity)</th>
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## DIET & EXERCISE LOG
Human Performance Research Laboratory  
California State University, Sacramento

### VISIT #5

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<th>Description of Exercise (include type, distance, duration &amp; intensity)</th>
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<td>Day of Visit</td>
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APPENDIX D

Visit Schedule
### Visit Schedule

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<td>High</td>
<td>Control</td>
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<td>High</td>
<td>Control</td>
<td>Moderate</td>
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</tr>
<tr>
<td>4</td>
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APPENDIX E

Rating of Perceived Exertion Scale
### BORG RPE SCALE

**Rating of Perceived Exertion**

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<th>Rating</th>
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<tr>
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<td>Extremely light</td>
</tr>
<tr>
<td>8</td>
<td>Very light</td>
</tr>
<tr>
<td>9</td>
<td>Light</td>
</tr>
<tr>
<td>10</td>
<td>Somewhat hard</td>
</tr>
<tr>
<td>11</td>
<td>Hard (heavy)</td>
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<tr>
<td>12</td>
<td>Very hard</td>
</tr>
<tr>
<td>13</td>
<td>Extremely hard</td>
</tr>
<tr>
<td>20</td>
<td>Maximal exertion</td>
</tr>
</tbody>
</table>
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