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PRE-RACE BLOOD LACTATE LEVELS AND PERFORMANCE

IN MIDDLE DISTANCE RUNNERS

by

Scott D. Williams

An Abstract

of a thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in the School of Health Sciences and Human Performance at Ithaca College

May 2001

Thesis Advisor: Dr. Betsy Keller

ABSTRACT

This study examined the effects of pre-race blood lactate levels on the performance of middle distance runners. Eight middle distance runners from the Ithaca College men's track and field team specializing at 400, 800, or 1500 m, volunteered for participation in the study. All subjects first completed a maximal oxygen consumption (VO₂ max) test. Subsequent testing sessions were conducted in the field at collegiate track meets. Each subject competed in four track meets during which performance, heart rate (HR), tympanic temperature (T), and lactate concentration ([LA]) were measured. Heart rate, T, and [LA] were measured at three times during each meet, 1) baseline, prior to the start of warm-up (WU); 2) pre-race, an estimated 5 minutes prior to the start of a race; and 3) post-race, within 7.5 minutes of race completion. Performance was recorded as the time necessary to cover the race distance. Despite significant differences in pre-race [LA] or percent change in [LA] from baseline to pre-race (% chg. [LA]), there were no differences in corresponding performance or post-race [LA]. Similarly, despite significant differences in performance, there were no differences in the corresponding pre-race [LA] or % chg. [LA]. However, there were significant differences in corresponding post-race [LA]. A strong relationship was found between performance in 800 m and post-race [LA]. Warm-up sometimes resulted in a decrease in [LA] from baseline to pre-race, which coincided with the best performance for some subjects, while other subjects performed best with an increase or minimal change in [LA]. In addition, a considerable degree of variability in pre-race [LA] corresponding to either the worst or best performance was found between subjects. The findings suggest a degree of individuality between athletes for an optimal WU in enhancing middle distance running performance.

PRE-RACE BLOOD LACTATE LEVELS AND PERFORMANCE

IN MIDDLE DISTANCE RUNNERS

A Thesis Presented to the Faculty of the School of Health Sciences and Human Performance Ithaca College

In Partial Fulfillment of the

Requirements for the Degree

Master of Science

by

Scott D. Williams

May 2001

Ithaca College Graduate Program in Exercise and Sport Sciences Ithaca, NY

CERTIFICATE OF APPROVAL

MASTER OF SCIENCE THESIS

This is to certify that the Thesis of

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submitted in partial fulfillment of the requirements for the degree of Master of Science in Exercise and Sport Sciences at Ithaca College has been approved.

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Chapter 1

INTRODUCTION

Development of Problem

Nearly all in sport, from recreational exercisers to professional athletes, partake in some sort of warm-up (WU) prior to engaging in their activity. The wide acceptance and use of a WU is rooted in the beliefs that by doing so, the risk of injury will be reduced and performance enhanced (Powers & Howley 1997, chap. 25). Aside from the questionable prophylactic potential pertaining to injury, the proposition that performance may be enhanced by a WU is enticing. However, with regards to the latter, the efficacy of a WU remains debatable.

In studies of WU, heart rate (HR), temperature (T), and lactate concentration ([LA]) are physiologic variables typically monitored. Little is known about the effects of pre-performance [LA] on performance. The presence of LA is of physiological significance that can be both beneficial and detrimental to performance. With as little as ten seconds of supramaximal activity causing a significant increase in existing [LA] (Jacobs, Tesch, Bar-Or, Karlsson, & Dotan, 1983), the potential for [LA] to be modified through a WU routine is quite possible.

Scope of Problem

The use of a WU is just as prevalent amongst track and field athletes as with athletes of any other sport. Anecdotal evidence indicates that some individuals will spend as long as 60 minutes in WU for less than 60 seconds of racing. Performance in middle distance track events, more so than other distances, has been shown to be dependent on the amount of LA that can be produced; such that, within a respective middle distance

event, higher post-race [LA] coincided with a better performance (Hautier et al., 1994; Lacour, Bouvat, & Barthélémy, 1990; Svendenhag & Sjödin, 1984). Lacour et al. (1990) specifically found significant relationships between post-race [LA] and performance in 400 and 800 m races; such that a better performance coincided with higher post-race [LA].

A number of studies found peak [LA] to vary as a result of prior exercise (Buono & Roby, 1982; Genovely & Stamford, 1982; Houmard et al., 1991; Ingjer & Strømme, 1979; Karlsson, Bonde-Petersen, Henriksson, & Knuttgen, 1975; Klausen, Knuttgen, & Forster, 1972; Martin, Robinson, Wiegman, & Aulick, 1975; Mitchell & Huston, 1993; Robergs et al., 1990). Interestingly, many of these same studies found peak [LA] to vary as a function of pre-existing [LA] (Buono & Roby, 1982; Genovely & Stamford, 1982; Karlsson et al., 1975; Klausen et al., 1972; Mitchell & Huston, 1993; Robergs et al., 1990), which suggests that WU may impact middle distance performance.

Statement of Problem

The purpose of this study was to assess the effects of pre-race blood [LA] on the performance of middle distance runners.

Hypotheses

1. Warm-up will result in an increase of HR, T, and [LA], above baseline values.

2. In accordance with previous findings, performance in 400 and 800 m races will be related to post-race [LA], but not the 1500 m races.

3. Post-race [LA] will be inversely related to both pre-race [LA] and percent change in [LA] from baseline to pre-race.

4. Performance will be inversely related to both pre-race [LA] and percent change in [LA] from baseline to pre-race.

5. The worst and best performance will be different, as will the corresponding pre-race [LA], post-race [LA], and percent change in [LA] from baseline to pre-race for the trials exhibiting the worst and best performance.

6. The highest and lowest percent change in [LA] from baseline to pre-race will be different, as will the corresponding post-race [LA] and performance, for the trials exhibiting the largest and smallest changes.

7. The highest and lowest pre-race [LA] will be different, as will the corresponding post-race [LA] and performance, for the trials exhibiting the highest and lowest pre-race [LA].

Assumptions of Study

For purposes of this study the following assumptions were made:

1. Blood LA sampled from a fingertip is indicative of existing levels in the muscle.

2. A maximal effort is given for each race.

3. Pre-race [LA] is representative of [LA] in the muscle at the start of the race.

4. Warm-up is typical and characteristic for each athlete.

Definition of Terms

The following items were defined for the purpose of this study:

<u>Criterion exercise (CE)</u>: refers specifically to any type of exercise challenge used to elicit physiologic responses or measure of performance; it does not refer to warm-up activity.

<u>Middle distance running</u>: refers to a classification of events in track and field, that include 400, 800, and 1500 m races; all of which have a substantial anaerobic component necessary for energy production.

<u>Peak [LA]</u>: blood lactate concentration measured upon completion of a criterion exercise; synonymous with post-race lactate concentration.

<u>Pre-existing [LA]</u>: blood lactate concentration resulting from warm-up that is measured prior to the criterion exercise; synonymous with pre-race lactate concentration.

Supramaximal activity: activity fueled predominantly by anaerobic energy systems.

<u>Warm-up (WU):</u> exercise conducted prior to a performance, whether or not muscle or body temperature is elevated.

Delimitations of Study

The following decisions served as delimitations for this study:

1. Data collection occurred during actual competitions in the meets prior to the 2000 NYSCTC Outdoor Championships.

2. Eight male middle distance runners of the Ithaca College Track and Field team served as subjects, whose ages ranged from 19-22 years.

3. Two subjects specialized at 400 m, four specialized at 800 m, and two specialized at 1500 m.

4. Each subject was tested on four separate occasions during those meets in which they competed at their specialty distance.

5. Resting, pre-race, and post-race measurements were each made for HR, T, and [LA].

6. Tympanic T served as an index of body T.

7. HR was monitored via palpation and HR monitors.

8. Blood samples (25 μ l) were taken and immediately mixed with 50 μ l of preservative agent.

9. The preserved blood samples were analyzed within 24 hours after blood draw.

Limitations of Study

The following decisions served as limitations to the study:

1. Only male middle distance runners from Ithaca College's track and field team were used, therefore these results may not be generalized to females and athletes other than collegiate male middle distance runners.

2. The WU routine used by each subject across the different meets was not controlled.

3. The time lapse between the pre-race measures and the start of the race may have varied slightly between meets, but was always estimated to be 5 minutes.

4. Weather conditions between the different meets could not be controlled.

5. Pre-race [LA] may not have been entirely indicative of the conditions existing immediately prior to the race due to involvement in activity by the subject between the time of sampling and the start of the race.

Chapter 2

REVIEW OF LITERATURE

Introduction

This review of literature begins by presenting general findings with regards to WU and performance, specifically comparing performance when preceded by WU to performance without WU. This is followed by a discussion of the effects of WU on T, HR, oxygen consumption (VO₂), and performance. Finally, the physiologic significance of lactic acid and the impact on performance is discussed.

Performance and Warm-up

A WU is typically engaged in with the hope of enhancing performance. Thus it follows that in evaluating the worth of a WU, performance be assessed. In a number of studies, the performance in a CE was greater in the trials preceded by some type of WU compared to the trials where no WU was performed (Andzel, 1982; De Vries, 1959; Grodjinovsky & Magel, 1970; Stewart & Sleivert, 1998).

De Vries (1959) assessed the effects of four different WU modalities on the swim performance for various strokes. The WU modalities included: an active swim, a hot shower, various calisthenics, or a massage, all of which were compared to a control condition of no WU. Considering the group as a whole, regardless of the stroke swum, performance in a 100 yard time trial was faster only when preceded by the active swim WU compared to the control. However, in some cases, when grouping the subjects by the stroke specialization, performance was improved to a greater extent through other WU modalities rather than through active swimming. For instance, breaststrokers and dolphin swimmers showed their greatest improvement in performance when they engaged in

calisthenics, whereas the performance of free stylers was slower than that of the controls, when preceded by calisthenics.

The studies by Grodiinovsky and Magel (1970), Andzel (1982), and Stewart and Sleivert (1998) compared running performance preceded by various active WU intensities to a control of no WU. In all cases, performance was enhanced by one of the active WU conditions compared to that of the control. A regular WU consisting of a five minute jog and calisthenics was as effective as a vigorous WU consisting of a five minute jog, calisthenics, and a maximal sprint over 176 yards. WU improved 60 and 440 yard run times above that of a control. While performance in the one mile run was only improved through the use of the vigorous WU, performance after the regular WU was equal to that of the control (Grodjinovsky & Magel, 1970). Andzel (1982) found one mile run performance improved only after a WU of two minutes at a HR of 140 beats per minute (bpm), whereas a WU for two minutes at a HR of 120 bpm produced similar performance times to that of the control. When assessing a 15 minute WU at intensities of 60, 70, or 80 % of maximal oxygen consumption (VO₂ max), time to fatigue was improved only after WU at intensities of 60 or 70 %, while WU at 80 % produced times to fatigue similar to no WU (Stewart & Sleivert, 1998).

It appears that some degree of active WU has an ergogenic effect on performance beyond that of no WU. In an attempt to explain potential mechanisms by which performance is enhanced following WU, various physiologic variables have been studied.

Temperature and Warm-up

The ability of a muscle to perform work is largely influenced by enzyme activity. Enzyme activity is a function of the rate at which it is able to convert substrates into

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"biologically useful energy" (Powers & Howley, 1997, chap. 3, pg. 26). The rate of enzyme activity is dependent on a number of factors. Temperature is one of these factors. The rate of reaction of an enzyme, or efficiency, increases with increasing T. However, enzyme activity will eventually peak with increasing T, after which a further increase in T will denature the protein structure of the enzyme and hinder its function (Holum, 1994, chap. 22; Lehninger, Wilson, & Cox, 1993, chap. 8; Powers & Howley, 1997, chap. 3).

Muscular work can increase body T. A T slightly above resting body T is optimum for increasing the activity of most enzymes (Powers & Howley, 1997, chap. 3). Therefore, a slight elevation in body T above resting could enhance performance through increased enzyme activity.

When body T was elevated above resting by 1.5 degrees Celsius (°C), performance was equal to that of no WU, and in some cases impaired (Genovely & Stamford, 1982; Stewart & Sleivert, 1998). Whereas a smaller increase in body T above resting (.7 - .9 °C), enhanced anaerobic performance (Stewart & Sleivert, 1998). These results suggest that enzyme activity, and thus performance, can be enhanced by a slight increase in body T above resting, while a more significant T elevation is detrimental.

Heart Rate, Oxygen Consumption, and Warm-up

Heart rate and VO_2 are two variables often measured simultaneously. In many studies (De Bruyn-Prevost, 1980; De Bruyn-Prevost & Lefebvre, 1980; Gutin, Stewart, Lewis, & Kruper, 1976; Martin et al., 1975; Robergs et al., 1991; Watt & Hodgson, 1975), both HR and VO_2 were elevated after WU. It is not surprising that elevated HR coincided with higher VO_2 given the close relationship between HR and VO_2

(Fox, Bowers, & Foss, 1989, chap. 12). However, of importance is the fact that as a result of WU, $\dot{H}R$ and VO_2 were elevated both prior to and during a CE.

Since VO₂ is an indication of energy expenditure, a higher VO₂ after WU is indicative of a greater reliance on, or activation of aerobic pathways for the energy needs necessary to complete a given task. In comparing the net adenosine triphosphate (ATP) generation from one molecule of glycogen through either glycolysis or oxidative phoshporylation, it is evident that aerobic pathways far exceed the efficiency of anaerobic pathways (Powers & Howley, 1997, chap. 3). Maximal glycolytic activity could deplete glycogen stores in two minutes (Hultman & Sahlin, 1980), while in comparison, the activation of aerobic pathways for ATP generation would have a glycogen sparing effect allowing for a couple hours of activity prior to depletion (Powers & Howley, 1997, chap. 4).

Based on results from the aforementioned studies pertaining to HR and VO₂, it appears conclusive that WU can enhance performance through some sort of mobilization effect that reduces the time necessary to recruit the aerobic energy system at the onset of an exercise task. A WU can therefore decrease the oxygen deficit experienced at the onset of exercise through a shortened adjustment period to exercise. In addition, a greater reliance on aerobic versus anaerobic pathways to meet the energy demands of an exercise task will result in a lower accumulation of LA. Studies to date suggest that performance may benefit by the elevation of T, HR, and VO₂ following a WU. It is less clear, however, what effects pre-existing [LA] may have on performance.

Lactic Acid

Lactic acid is a substance produced by the liver, intestines, red blood cells, and most abundantly by skeletal muscle as the end-product of anaerobic energy production through glycolysis and glycogenolysis (Brooks, 1988). The pKa of lactic acid is 3.8. Therefore, at physiologic pH values, lactic acid is almost completely dissociated to a LA anion and a proton/hydrogen ion (H+) such that LA and H+ are formed in equal amounts (Brooks, 1991; Sahlin, 1986). The LA ion itself does not have any detrimental effects on metabolism or muscular contraction, but it is the decrease in pH associated with the increased [H+] that erodes performance (Sahlin, 1986).

The accumulation of lactic acid from muscular activity is responsible for more than 85% of H+ produced. Thus, the major factor in determining changes in muscle pH is the accumulation of the lactic acid generated H+ (Hultman & Sahlin, 1980; Sahlin, 1986). Both lactate and H+ cross over the muscle membrane at about equal rates, therefore lactate measurements will give information on H+ activity and thus pH (Fox et al., 1989, chap. 21; Hultman & Sahlin, 1980). The efflux of LA from the muscle into circulation is a function of LA accumulation within the muscle. Lactate release from the muscle to circulation increases as [LA] increases within the muscle. However, there appears to be a limit on the translocation of LA from the muscle to the circulation, which reaches a peak efflux of 4-5 mmol min⁻¹ at a concentration of 4 mmol·kg⁻¹ wet muscle. Therefore, [LA] in the muscle can, at times, be two to three fold greater than [LA] in blood (Hultman & Sahlin, 1980). Despite this controversy over the relationship between blood and muscle [LA], blood LA measurements are a widely accepted means by which to quantify [LA] in the muscle. Moreover, they are easy to do, requiring only a few drops such as the ear or finger, and the measurements are highly reproducible when taken from the same site (Billat, 1996; Dassonville et al., 1998; Jacobs, 1986). Based on the apparent time lag in translocation of LA from muscle to circulation, to more accurately quantify the maximal [LA], blood samples should be drawn at about 7.65 minutes following the cessation of exercise (Fujitsuka, Yamamoto, Ohkuwa, Saito, & Miyamura, 1982).

The [LA] measured in the blood is dependent on both production and removal (Billat, 1996; Brooks, 1991; Jacobs, 1986; Katz & Sahlin, 1988; Powers & Howley, 1997, chap. 4). Skeletal muscle produces the most LA, especially during supramaximal work. However, skeletal muscle is not the sole contributor to existing [LA]. The liver, intestines, and red blood cells all contribute to LA production during both rest and exercise (Brooks, 1988, 1991). As little as ten seconds of supramaximal activity elicits a significant elevation in muscle [LA] (Jacobs et al., 1983). However, supramaximal and anaerobic exercise are not the only activities during which LA is produced. Activity at intensities corresponding to 50-75% VO₂ max also results in elevation of blood [LA] (Fox et al., 1989, chap. 13; Katz & Sahlin, 1988; McArdle, Katch, & Katch, 1991, chap. 11; Powers & Howley, 1997, chap. 4).

Lactate removal from circulation is achieved via slow-twitch skeletal muscle, cardiac muscle, and the liver (Brooks, 1988, 1991). While slow twitch muscle and cardiac muscle can metabolize LA and use it as a source of energy, the liver utilizes LA to produce glucose via gluconeogenesis in the Cori-cycle (Fox et al., 1989, chap. 3; Hutlman & Sahlin, 1980, McArdle et al., 1991, chap. 6; Powers & Howley, 1997, chap. 4). Skeletal muscle can also aid in LA removal not only through oxidation and efflux into circulation but also by reforming glucose and glycogen (Fox et al., 1989, chap. 3; Hultman & Sahlin, 1980). Lactate removal by both skeletal and cardiac muscle is greater during submaximal exercise compared to rest (Hultman & Sahlin, 1980). Removal of LA by the splanchnic region is also greater during exercise compared to removal during resting conditions. Lactate removal is greatest during the first 5-15 minutes of steadystate, submaximal exercise (Hultman & Sahlin, 1980). The kidneys are also integral in the removal of LA. The kidneys remove LA via oxidative and gluconeogenic pathways in addition to renal excretion. The activity of the kidneys in the removal of LA is increased under acidotic conditions (Hutlman & Sahlin, 1980).

Enzyme Activity

Lactic acid is of a physiological significance that can be both detrimental and beneficial for athletic endeavors. Muscular fatigue is most commonly associated with the production of lactic acid. The fatigue experienced upon lactic acid accumulation is a result of decrements in both metabolic and contractile functioning. The decrease in metabolic functioning that is associated with acidosis resides in the activity of phosphofructokinase (PFK). Phosphofructokinase is the enzyme involved in the conversion of fructose 6-P to fructose-1,6-diP, which is the key rate-limiting step in the glycolytic pathway for ATP production (Fox et al., 1989, chap. 2; Hultman & Sahlin, 1980; Lehninger et al., 1993, chap. 14; McArdle et al., 1991, chap. 6; Powers & Howley, 1997, chap. 3; Sahlin, 1986). As mentioned earlier, the activity level of an enzyme is dependent on a number of factors. The pH is another key factor in determining the rate of enzyme activity. Just as each enzyme has an optimal T for maximum activity, there is an optimum pH at which the functioning of an enzyme is maximized (Lehninger et al., 1993,

chap. 4). As lactic acid accumulates and dissociates into LA and H+, the pH begins to drop. This drop in pH results in an inhibition of the enzyme PFK and restricts further energy production via glycolysis (Fox et al., 1989, chap. 5; Hultman & Sahlin, 1980; Sahlin, 1986; McArdle et al., 1991, chap. 6; Powers & Howley, 1997, chap. 3).

Contractile Function

The process of excitation-contraction coupling, which dictates muscle function, is another site where fatigue can develop due to lactic acid accumulation. The release of calcium (Ca++) from the sarcoplasmic reticulum (SR) into the cytoplasm, the binding of Ca++ with the contractile protein troponin, and the activity of myosin-ATPase, are all integral steps in the contractile process which are hampered by lactic acid accumulation (Fox et al., 1989, chap. 5; Hultman & Sahlin, 1980; Sahlin, 1986). When compared to normal pH, acidotic conditions cause increased binding of Ca++ to proteins in the SR. Therefore, under acidotic conditions, the amount of Ca++ released per excitatory impulse is less. In addition, regulatory protein and myosin ATP-ase activity are less responsive per unit of Ca++ in acidotic conditions compared to normal pH. In sum, there is a decrease in the tension that can be developed for each excitatory signal that traverses the neuromuscular junction under acidotic conditions (Fox et al., 1989, chap. 5; Hultman & Sahlin, 1980; Sahlin, 1986).

Perfusion

Muscle perfusion is a process enhanced by the presence of lactic acid. The amount of blood flow through a given area of the body is regulated by the radius of the vessels supplying that area. The presence of various metabolic products can vary the degree of constriction or dilation of the arterioles through intrinsic regulation. During exercise, adequate blood flow is maintained via active hyperemia. Active hyperemia is the vasodilatory response that is produced in active musculature as a result of the presence of various metabolic factors (Sherwood, 1993, chap. 10). The decrease in pH typically associated with the H+ generated from lactic acid dissociation, is one of these metabolic factors which causes a local vasodilatory response during exercise. The decrease in pH causes relaxation of arteriolar smooth muscle, thus reducing vascular resistance and increasing blood flow (Delp & Laughlin, 1998; Fox et al., 1989, chap. 10; McArdle et al., 1991, chap. 16; Powers & Howley, 1997, chap. 9; Saltin, Rådegran, Koskolou & Roach, 1998; Sherwood, 1993, chap. 10). In resting muscle, it is estimated that only one of every 30 to 40 capillaries is open. With exercise-induced hyperemia, muscle blood flow (McArdle et al., 1991, chap. 16; Powers & Howley, 1997, chap. 9). There is also an increase in surface area between muscle tissue and blood over which oxygen can be delivered and waste products removed (McArdle et al., 1991, chap. 16).

Oxygen Dissociation

In addition to the vasodilatory effects of lactic acid, it also plays an integral role in facilitating the dissociation of oxygen from its carrier protein hemoglobin. Known as the Bohr effect, hemoglobin's affinity for oxygen is decreased when in the presence of various metabolic factors produced during muscular work. The drop in pH caused by lactic acid production, is a metabolic factor that aids in decreasing hemoglobin's affinity for oxygen (Fox et al., 1989, chap. 9; Grassi, Quaresima, Marconi, Ferrari, & Cerretelli, 1999; Holum, 1994, chap. 23; Hultman & Sahlin, 1980; Lehninger et al., 1993, chap. 7; McArdle et al., 1991, chap. 13; Powers & Howley, 1997, chap. 10; Sherwood, 1993,

chap. 13; Stringer et al., 1994). Under acidotic conditions, various amino acids of hemoglobin become protonated resulting in the formation salt bridges which configure hemoglobin in such a manner that the bonds with oxygen are broken (Lehninger et al., 1993, chap. 7). Less oxygen bound to hemoglobin allows for greater oxygen diffusion into the muscle and availability for aerobic pathways to produce energy. Whereas, under normal pH, oxygen bound to hemoglobin is not readily available to the mitochondria (Fox et al., 1989, chap. 9; Hultman & Sahlin, 1980; McArdle et al., 1991, chap. 13; Powers & Howley, 1997, chap. 10; Sherwood, 1993, chap. 13).

<u>Analgesia</u>

The opioid peptide β-endorphin is commonly known for its analgesic-producing effects (Schwarz & Kindermann, 1992; Sforzo, 1988). Interestingly, it has been suggested that the release of β-endorphin into circulation may be linked to exercise intensity, where greater exercise intensity is accompanied by a greater release of βendorphin (Colt, Wardlaw, & Frantz, 1981; McMurray, Forsythe, Mar, & Hardy, 1987; Rahkila, Hakala, Alén, Salminen, & Laatikainen, 1988). In fact, it has been shown that βendorphin release increases concomitantly with various indicators of acidosis including that of LA (Rahkila et al., 1988; Taylor et al., 1994).

Warm-up and Peak Lactate

The diverse physiologic responses resulting from LA accumulation, as well as the potential for [LA] to be modified through WU, have been well established. However, the relationship between pre-existing [LA] and peak [LA] remains to be elucidated. Many studies have shown peak [LA] to vary as a function of pre-existing [LA] (Buono & Roby,

1982; Genovely & Stamford, 1982; Karlsson et al. 1975; Klausen et al., 1972; Mitchell & Huston, 1993; Robergs et al., 1990).

Both Klausen et al. (1972) and Karlsson et al. (1975) have shown greater peak [LA] as a result of higher pre-existing [LA]. For instance, when pre-existing [LA] was ten fold greater than resting conditions, Klausen et al. (1972) found corresponding peak [LA] to measure 15.3 mmol·L⁻¹ compared to 12.9 mmol·L⁻¹ measured in trials where preexisting [LA] equaled resting values. Similar results were demonstrated in the study by Karlsson et al. (1975), where peak [LA] corresponding to elevated pre-existing [LA] was 14.0 mmol·L⁻¹ compared to 10.6 mmol·L⁻¹ measured in trials where pre-existing [LA] equaled resting values. The elevated pre-existing [LA] in both studies, resulted from a maximal bout of exercise prior to the CE. However, in the Klausen et al. (1975) study, the same exercise mode was used for WU and the CE, whereas in the Karlsson et al. (1975) study, WU and the CE modalities involved arm and leg exercises, respectively. Both blood and muscle peak [LA] were elevated during pre and post measurements of trials involving only leg muscles during the CE despite the use of only the arms during the prior exercise (Karlsson et al. 1975).

Similar effects on peak [LA] after WU were also observed by Mitchell and Huston (1993). In this study, pre-existing [LA] increased due to a WU routine rather than a maximal bout of exercise prior to the CE. Pre-existing and peak [LA] of 6.97 mmol·L⁻¹ and 13.66 mmol·L⁻¹, respectively, for subjects who performed a WU, compared to pre-existing and peak values of 1.73 mmol·L⁻¹ and 10.04 mmol·L⁻¹, respectively, for no WU.

In contrast, Robergs et al. (1990), Genovely and Stamford (1982), and Buono and Roby (1982) reported lower peak [LA] following a higher pre-existing [LA]. Robergs et al. (1990) found pre-existing [LA] of 3.1 and 1.7 mmol·L⁻¹ to correspond with peak [LA] of 10.7 and 12.8 mmol·L⁻¹, respectively. Genovely and Stamford (1982) showed preexisting LA of 4.2 and 1.2 mmol·L⁻¹ to correspond with peak LA of 9.4 and 13.7 mmol·L⁻¹, respectively. Buono and Roby (1982) found pre-existing LA of 5.59 and 1.11 mmol·L⁻¹ to correspond with peak LA of 10.68 and 11.84 mmol·L⁻¹, respectively. Robergs et al. (1990) utilized different WU routines to elicit differences in pre-existing LA between testing conditions, as did Genovely and Stamford (1982). There was no specific WU routine implemented in the study by Buono and Roby (1982). Instead, the CE was performed twice and the second trial followed 25 minutes after the first. Pre-existing [LA] prior to the first trial was equal to resting values, while the pre-existing [LA] prior to the second trial was elevated due to LA that remained in circulation as a result of the first trial of the CE.

Unlike the aforementioned studies in which peak [LA] was found to either increase or decrease as a result of pre-existing [LA], Robergs et al. (1991) and Mitchell and Huston (1993) have shown peak [LA] to be unaffected when pre-existing [LA] was greater than resting levels. Robergs et al. (1991) showed peak [LA] to be equal between two trials, despite different pre-existing [LA] of 5.2 and 1.7 mmol·L⁻¹. Similarly, Mitchell and Huston (1993) found different pre-existing [LA] of 6.15 and 1.56 mmol·L⁻¹, to result in equal peak [LA].

A few studies have found peak [LA] to change as result of WU despite any appreciable rise in pre-existing [LA] above resting levels (Martin et al., 1975; Ingjer & Strømme, 1979; Houmard et al., 1991). In all cases the WU resulted in pre-existing [LA] equal to no WU. Ingjer and Strømme (1979) found peak [LA] for CE performed after

WU to be 6.5 mmol·L⁻¹ while no WU resulted in a peak [LA] of 9.9 mmol·L⁻¹ for the same CE. Consistent with these findings, Houmard et al. (1991) showed a WU prior to a CE resulted in peak [LA] of 4.3 mmol·L⁻¹ compared to 6.2 mmol·L⁻¹ for no WU. Similarly, Martin et al. (1975) found peak [LA] was 25% lower as a result of WU compared to no WU. In addition to peak [LA], other physiological variables have also been found to vary as a result of elevated pre-existing [LA].

Warm-up and Other Responses

Elevated pre-existing [LA] has been shown to result in greater VO₂ at the onset of a CE (Mitchell & Huston, 1993), during the first couple of minutes of a CE (Weltman, Stamford, & Fulco, 1979; Buono & Roby, 1982; Robergs et al., 1991), and cumulatively upon completion of a CE (Buono & Roby, 1982; Weltman et al., 1979). Weltman et al. (1979) and Buono and Roby (1982) also reported lower carbon dioxide production (VCO₂) and respiratory exchange ratio (RER) during the CE as a result of elevated preexisting [LA]. Robergs et al. (1990) found elevations in pre-existing [LA] and [H+] to result in smaller [CO₂] and [H+] in blood upon completion of a CE. These findings suggest elevated pre-existing [LA] results in greater reliance on aerobic pathways for energy production and decreased disturbance in acid-base status, both of which may influence performance.

Warm-up and Performance

Perceived performance enhancement is often the motivating factor for WU. Few studies have measured performance in response to WU-induced changes in pre-existing [LA]. Instead, studies used a standardized work bout to assess the effects of WU (Bruyn-Prevost, 1980; Buono & Roby, 1982; Houmard et al., 1991; Ingjer & Strømme, 1979;

Martin et al., 1975; Mitchell & Huston, 1993; Robergs et al., 1990; Robergs et al., 1991). Few studies of pre-existing [LA] incorporated a quantifiable performance measure. Karlsson et al. (1975) and Genovely and Stamford (1982) found performance was hampered when pre-existing [LA] was elevated, while Klausen et al. (1972), Weltman et al. (1979), and Mitchell and Huston (1993) did not find any differences in performance as a result of elevated pre-existing [LA].

Middle Distance Running Performance

Andersen, Bolstad, and Sand (1960) demonstrated that after running either 400 or 800 m, oxygen debt and [LA] were generally higher compared to running either 100 or 200 m. Similarly, Svendenhag and Sjödin (1984) found [LA] to be higher upon completion of races in runners specializing at 400 m, 800 m, 800 - 1500 m compared to runners who specialized at 1500 - 5000 m, 5000 - 10000 m, or 10000 m - marathon. Upon assessing the percent contribution of the various energy systems to ATP production over 400, 800, or 1500 m, it is no surprise that peak [LA] is greater after races representative of these distances since a substantial amount of energy for these races is generated via glycolytic pathways (Powers & Howley, 1997, chap. 21).

In elite track athletes specializing at 100, 200, 400, 800, or 1500 m, both the peak [LA] and performance have been measured throughout a competitive season (Hautier et al., 1994; Lacour et al., 1990). The performances of the 400 and 800 m runners were found to be significantly related to the post-race [LA]. Correlation coefficients of r=.89 and r=.71 were found between the post-race [LA] and the average velocity of the performances for the 400 and 800 m runners, respectively (Lacour et al., 1990). Similar trends between performance and post-race [LA] have also been demonstrated in a number of swimmers competing in events comparable to the 400 and 800 m track events (Sawka, Knowlton, Miles, & Critz, 1979). Given the substantial variability in peak [LA] that has prior to been shown to result from elevated pre-existing [LA], a predicament arises for individuals whose performance resides in peak [LA].

Summary

In general, an active WU appears to benefit performance. However, little is known regarding the optimum length and intensity of WU to maximize performance. Though the [LA] at a given time is dependent on a number of factors, it is readily affected by physical activity such as that engaged in during WU. The physiologic responses to LA may be beneficial or detrimental to performance. With regards to the peak LA response, some have found peak [LA] to increase in accordance with elevated pre-existing [LA], while others have found peak [LA] to decrease, and still others have found peak [LA] to be unaffected. Such variability in the peak LA response caused by modification of preexisting [LA] through WU, suggests implications for those whose performance is highly dependent on energy produced via glycolytic pathways, such as middle distance runners.

Chapter 3

METHODS

Subjects

Volunteers from the men's track and field team at Ithaca College competing in middle distance events were sought for the study. Eight members volunteered for participation in the study ranging in age from 19 to 22 years. Permission to seek volunteers from the track and field team was granted by the coach (Appendix A). Prior to their participation in the study, all subjects were informed of the nature of the testing procedures and gave their written informed consent (Appendix B). Procedures for this study were approved by the Human Subjects Research Committee at Ithaca College.

Procedures

Each subject completed a total of five test sessions. During the first test session a VO_2 max test was performed. Subsequent test sessions (2-5) occurred at track meets, during which performance time was recorded, and HR, [LA], and T were measured at rest, prior to, and following a race.

VO₂ Max Test Protocol

The VO₂ max test consisted of continuous running on a motorized treadmill to volitional exhaustion. An elevation-based protocol was utilized where the elevation of the treadmill increased 3% every two minutes beginning at a grade of 0%, while the speed remained constant at a self-selected pace. Oxygen consumption, VCO₂, HR, RPE, and [LA] were measured during the test.

Gas Analysis

During the test, the subject wore a two-way breathing valve (Hans Rudolph, Kansas City, MO) for collection of expired gases. Oxygen consumption and VCO₂ were determined with a computerized metabolic measurement system (ParvoMedics TrueMax 2400, Sandy, UT). The system was turned on for a minimum of 30 minutes prior to calibration. Ambient temperature (°C), relative humidity, and barometric pressure (mmHg) were entered into the system on each day of testing. The flowmeter and gas analyzers were calibrated each day before testing commenced, as well as within the same day after every fifth test. The flowmeter was calibrated with a 3-L syringe (ParvoMedics, Sandy, UT). The calibration was accepted when verification flow values were within \pm 3% of the predicted values. The gas analyzers were calibrated with a factory prepared gas standard (4% CO₂, 16% O₂, balance N₂) (ParvoMedics, Sandy, UT). The calibration was accepted when verification analysis values for CO_2 and O_2 were within $\pm .02$ of the standard gas values. During testing, the gas analyzer sampled expired gas every 15 seconds from which minute averages were calculated. The final four 15 second samples recorded upon completion of the test were averaged for determination of VO₂ max.

Heart Rate Analysis

Heart rate was monitored with the use of HR monitors (SensorDynamics Gemini, Fremont, CA). With ten seconds remaining in each stage, a one-second steady state HR value was recorded. In addition, a one-second HR value was attained upon termination of the test.

Exertion Analysis

Rating of perceived exertion (RPE) was measured with the 6-20 Borg scale (Borg, 1982). Rating of perceived exertion was recorded with 15 seconds remaining in each stage as well as upon termination of the test.

Lactate Analysis

Peak blood [LA] was determined within 7.5 minutes after the completion of the test. Determination of [LA] required a sample of blood (25 μ l) to be drawn from the subject's fingertip, which was sterilized with alcohol swab prior to sampling. The 25 μ l blood sample was collected in a capillary tube and subsequently analyzed with an automated lactate analyzer (YSI 1500 Sport Tester, Yellow Springs, OH). Prior to each analysis the analyzer was calibrated with a 5 mmol·L⁻¹ lactate standard (YSI 5 mmol·L⁻¹ Lactate Standard, Yellow Springs, OH). Calibration was accepted when verification analysis of the 5 mmol·L⁻¹ lactate standard was within ± 1%.

Field Testing Protocol

Testing sessions 2-5 took place at actual collegiate track meets. Heart rate, [LA], and T were measured at three times (baseline, pre-race, and post-race) during each meet. Baseline measurements were taken prior to the beginning of a pre-race warm-up. Pre-race measurements were made an estimated 5 minutes prior to the start of a race. The order of events for each meet ran according to a time schedule from which an estimate was made for a time in which to take pre-race samples. Post-race measurements were taken within 7.5 minutes after the completion of the race.

Heart Rate Analysis

All HR measurements were taken with HR monitors (SensorDynamics Gemini, Fremont, CA) with the exception of the baseline measurements. Baseline HR measurements were taken by palpating the radial artery for 20 seconds after the subject had been seated and rested for a minimum of five minutes. Pre-race HR was recorded as a single second value and measured at five minutes prior to the start of a race. Post-race HR was recorded as a single second value and measured immediately upon completion of a race.

Temperature Analysis

Tympanic T was measured with an infrared thermoscan device (Braun Pro-1, Kronberg, Germany) and used as an index of body T. Two measurements in the same ear were taken, from which an average value was calculated and recorded. Baseline temperature was taken after the subject had been seated and rested for a minimum of five minutes. Pre-race T was measured five minutes prior to the start of a race.

Lactate Analysis

Each LA determination required a 25 µl sample of blood to be drawn from the subject's fingertip in the same manner as previously described. Since the LA analyzer was not available for immediate analysis within the field for sessions 2-5, each blood sample was preserved. The preservative agent lysed the red blood cells and inhibited any further LA production. The preservative agent was prepared beforehand as described in Appendix C.

Each 25 μ l sample of blood was added to its own collection vile that contained 50 μ l of the preservative agent. The samples were then refrigerated and later analyzed in the same manner as previously described within 24 hours of sampling.

Since all whole blood samples were diluted with the preservative agent, adjustments were required for values generated by the LA analyzer. Pilot testing was performed to determine a correction factor by which to multiply the value obtained from analysis of a diluted, preserved sample in order to estimate the actual blood [LA] at the time of blood draw. Two, 25 μ l blood samples were drawn from the same site and at the same time. One sample was analyzed immediately. The other was placed in a collection vile containing 50 μ l of preservative agent and later analyzed after 24 hours of refrigeration. The two samples taken from the same site at the same time were then compared. This procedure was performed a total of 20 times.

Simple linear regression analysis was performed between the preserved, diluted blood samples and the non-preserved blood samples in order to predict the [LA] in the blood at the time of blood draw from the LA analysis of a preserved, diluted blood sample. The regression analysis generated the equation "y = 2.52x + 0.03" for predicting the blood [LA] at the time of blood draw (y) from the LA analysis of a preserved, diluted blood sample (x). The data for the regression analysis and the results are summarized in Appendix D.

Performance Analysis

The performance measured for each subject was the time necessary for completing a race. The time was measured and recorded by meet officials through the use of fully automatic timing.

Data Management

For comparing the physical characteristics between events, event (400, 800, and 1500 m) was the independent variable (IV), while age, height, weight, and VO₂ max were dependent variables (DV). To assess changes in physiologic variables due to WU, measurement (baseline and pre-race) and meet (1-4) were the IV's, while HR, T, and LA were the DV's.

Three categories were formed to rank order pre-race [LA] (category₁), performance (category₂), and percent change in [LA] from baseline to pre-race (category₃) for analysis. Within each category, four groups were identified by within subject rank ordering.

The groups in category₁ were established by rank ordering the pre-race [LA] from lowest to highest. Group₁ (lowest, intermediate-1, intermediate-2, and highest) and event (400, 800, and 1500m) were the IV's. The pre-race [LA] and corresponding post-race [LA] and performance were the DV's.

Groups in category₂ were established by rank ordering the performance from worst to best. Group₂ (worst, intermediate-1, intermediate-2, and best) and event (400, 800, and 1500m) were the IV's. The performance and corresponding pre-race [LA], percent change in [LA] from baseline to pre-race, and post-race [LA] were the DV's.

Groups in category₃ were established by rank ordering the percent change in [LA] from baseline to pre-race. Group₃ (smallest, intermediate-1, intermediate-2, and largest) and event (400, 800, and 1500m) were the IV's. The percent change in [LA] from baseline to pre-race and the corresponding post-race [LA] and performance were the DV's.

Data Analysis

The alpha level for all analyses was set at 0.05. One-way ANOVA was performed between event (IV) and the physical characteristics (DV). Separate two-way repeated measures ANOVA's between measurement (IV, within subjects) and meet (IV, within subjects) were performed on the physiologic variables (DV). For each category, separate two-way repeated measures ANOVA's between event (IV, between subjects) and group (IV, within subjects) were performed on the category-defining variable and its corresponding measures (DV). Pearson correlation analyses were performed between the following variables: pre-race [LA], percent change in [LA] from baseline to pre-race, post-race [LA], and performance.

Chapter 4

RESULTS

Introduction

This study was conducted to investigate the effects of pre-race blood lactate on middle distance running performance. Each subject (N=8) completed four trials during which HR, LA, T, and performance were measured. The effects of pre-race blood lactate on performance were assessed through ANOVA and correlation analyses between the following variables: pre-race [LA], percent change in [LA] from baseline to pre-race, post-race [LA], and performance.

Physical Characteristics

A one-way ANOVA was performed to assess differences in the physical characteristics of the subjects. The middle distance event (400, 800, or 1500 m) served as the IV, while age (years), height (cm), weight (kg), and VO₂ max (ml·kg⁻¹·min⁻¹) were the DV's. The physical characteristics of the subjects, grouped by event, are reported in Table 1. The analyses revealed there were no significant differences between the 400 m (<u>n</u>=2), 800 m (<u>n</u>=4), or 1500 m runners (<u>n</u>=2) for age (<u>F</u>=5.19, <u>p</u>>0.05), height (<u>F</u>=0.13, <u>p</u>>0.05), weight (<u>F</u>=1.03, <u>p</u>>0.05), or VO₂ max (<u>F</u>=1.64, <u>p</u>>0.05). A summary of the analyses is presented in Appendix E (Tables A, B, C, and D, respectively for age, height, weight, and VO₂ max).

Ambient Meet Conditions

Since each meet was contested in an outdoor environment, it was possible that the ambient conditions during competition may have influenced WU, performance, or both. Therefore, mean, maximum, and minimum temperatures from the cities in which the

Table	1

Physical Characteristics of Subjects Grouped by Event						
Event		Age	Height	Weight	VO ₂ max	
(m)	<u>n</u>	(years)	(cm)	(kg)	(ml•kg ⁻¹ •min ⁻¹)	
400	2	21.5 ± 0.7	179 ± 12	$70.9~\pm~3.8$	56.2 ± 1.1	
800	4	19.3 ± 0.5	176 ± 2	71.5 ± 3.8	59.8 ± 2.2	
1500	2	20.0 ± 1.4	176 ± 5	68.2 ± 1.9	64.1 ± 9.0	
Total	8	20.0 ± 1.2	177 ± 6	70.5 ± 2.7	60.0 ± 4.8	

Physical Characteristics of Subjects Grouped by Event

<u>Note.</u> Physical characteristics are represented as mean \pm standard deviation.

No significant differences were found between events for any of the characteristics.

meets took place are reported, as well as precipitation (Table 2). The temperature readings were reported by the National Weather Service as single values, which limited the ability to perform any statistical analysis of the values, however, it is evident that there was some degree of variation in the ambient conditions between meets.

Baseline and Pre-race Measures

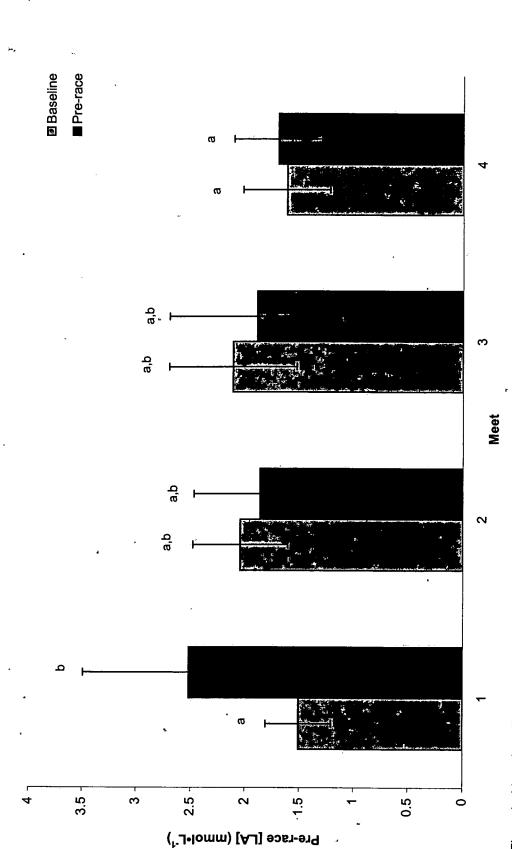
A 2x4 ANOVA (measurement x meet) with repeated measures on both factors, was performed on the dependent variable [LA] to assess the effects of WU on [LA]. No significant differences in [LA] were found for the main effects of measurement (\underline{F} =0.69, \underline{p} >0.05) or meet (\underline{F} =1.39, \underline{p} >0.05), however, there was an interaction between measurement and meet (\underline{F} =7.75, \underline{p} <0.05) (Figure 1). A summary of the analysis is presented in Appendix E (Table E). Tukey Honestly Significant Difference (HSD) posthoc analysis revealed significant differences in [LA] (\underline{p} <0.05) between meet 1 baseline (1.51±0.30 mmol·L⁻¹) and meet 1 pre-race (2.52±0.98 mmol·L⁻¹), meet 1 pre-race and meet 4 baseline (2.05±0.43 mmol·L⁻¹), and meet 1 pre-race and meet 4 pre-race (2.52±0.98 mmol·L⁻¹). The results are illustrated by Figure 1.

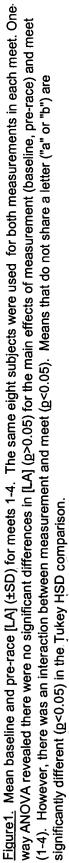
A 2x4 ANOVA (measurement x meet) with repeated measures on both factors, was performed on the dependent variable HR to assess the effects of WU on HR. There were significant differences in HR for the main effects of measurement (\underline{F} =145.86, p<0.05) and meet (\underline{F} =7.58, p<0.05), as well as an interaction between measurement and meet (\underline{F} =3.62, p<0.05) (Figure 2). A summary of the analysis is presented in Appendix E (Table F). Tukey HSD post-hoc analysis revealed that regardless of meet, all baseline HR's were significantly different than pre-race HR's (p<0.05). In addition, meet 1 prerace (115.9±23.5 beats min⁻¹) was significantly different than both meet 3 pre-race

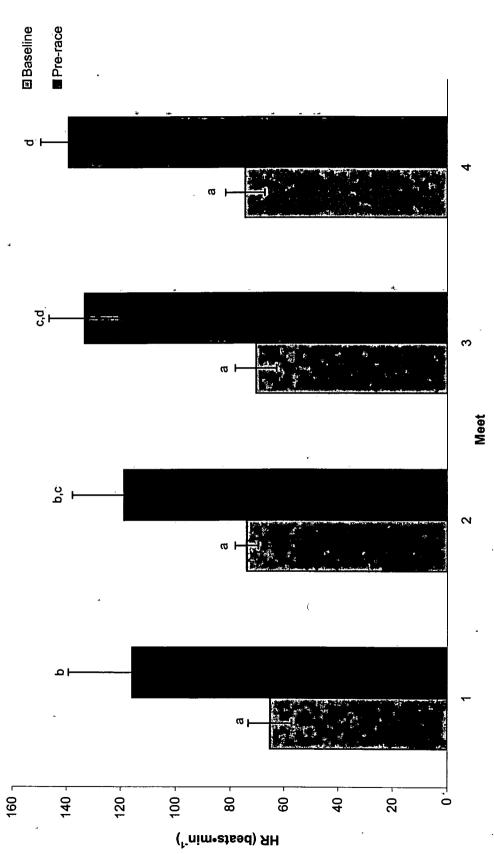
Date	Location	Mean	Max	Min	
(Month/Day)	(City, State)	(°C)	(°C)	(°C)	Precipitation
4/1	Rochester, NY	8.3	16.9	1.1	N
4/8	Ithaca, NY	9.4	· 17.2	3.1	Y
4/15	Allentown, PA	11.9	15.0	8.1	Y
4/22	Rochester, NY	6.4	14.4	4.4	Y

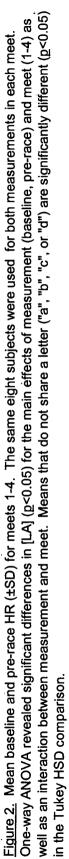
Geographic Ambient Outdoor Meet Weather Conditions

Note. Mean, Maximum (Max), and Minimum (Min) temperatures were the average, highest and lowest temperatures, respectively, recorded over the 24 hour period for the day of the meet in the respective city. A yes (Y) in the precipitation column indicates that it rained at some point during the meet, while a no (N) indicates that there was no rainfall at any time during the meet. All temperatures were recorded and reported by the National Weather Service. The researchers made a note of any precipitation during the meets.









 $(133.5\pm13.2 \text{ beats} \cdot \text{min}^{-1})$ and meet 4 pre-race $(139.5\pm10.3 \text{ beats} \cdot \text{min}^{-1})$. Similarly, meet 2 pre-race $(118.9\pm19.1 \text{ beats} \cdot \text{min}^{-1})$ was significantly different than meet 4 pre-race. The results are illustrated by Figure 2.

Due to instrument malfunction, baseline and pre-race T were measured in only one meet for each subject. Therefore, a dependent t-test was used to assess differences between the two measurements. The dependent t-test revealed no significant differences between baseline and pre-race T (\underline{t} =-0.85, \underline{p} >0.05). A summary of the analysis is presented in Appendix E (Table G).

Collectively, WU resulted in increased HR, while [LA] and T remained unchanged. The mean baseline and pre-race values for HR, [LA], and T are presented in Table 3.

Post-race Lactate and Performance

A Pearson correlation analysis was performed to assess the relationship between post-race [LA] and performance (average velocity). The analysis revealed a significant relationship (r= 0.858, p<0.01, n=16) between the average velocity of a race (m·s⁻¹) and the post-race [LA] (mmol·L⁻¹) for 800 m. The relationship between post-race [LA] and performance in 400 m did not reach statistical significance (r=0.700, p=0.053, n=8). There was also no significant relationship between average velocity and post-race [LA] for 1500 m (r=0.332, p>0.05, n=8). Only the 800 m performance was strongly related to [LA] measured in the blood upon completion of a race. The results are presented in Table 4 and illustrated in Figure 3.

Mean and SD of Baseline and Pre-race Measures for Heart Rate (HR),

Lactate (LA), and Temperature (T)					
	HR	LA	Т		
Measurement	(beats•min ⁻¹)	$(\text{mmol} \cdot L^{-1})$	(°C)		
Baseline	$71.1 \pm 7.7_{a}$	$1.83 \pm 0.50_{a}$	$35.0 \pm 0.6_{a}$		
Pre-race	$126.9 \pm 19.3_{b}$	$2.00 \pm 0.76_{a}$	$35.1 \pm 0.5_{a}$		

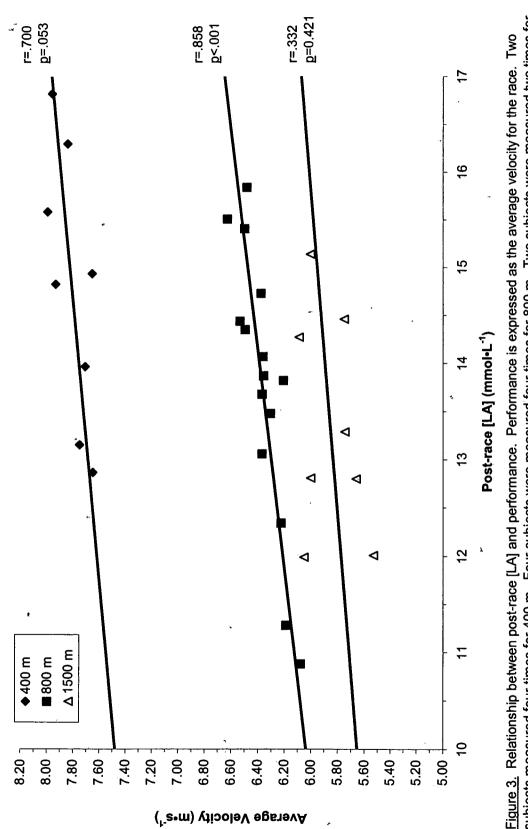
<u>Note.</u> Baseline and pre-race HR and LA were measured during four trials for each subject (<u>N</u>=8). Due to instrument problems in the field, a total of only 8 trials were recorded for baseline and pre-race T amongst the subjects (<u>N</u>=8). Means in the same column that do not share subscripts differ at p<0.05. Temperature was compared using a dependent t-test. Repeated measures ANOVA was used to compare baseline and pre-race measures for both HR and LA.

Average Vel	ocity (m•s ⁻¹)	and Post-race	[LA] (mmol•L ⁻¹)
Event	Trials	<u>r</u>	p
· 400	8	0.700	0.053
800	16	0.858	0.000
1500	. 8	0.332	0.421
Total	32	0.504	0.003

Summary of Pearson Correlation Analysis between

Note. 2-400 m, 4-800 m, and 2-1500 m runners each

completed four trials in their respective event.



subjects measured four times for 400 m. Four subjects were measured four times for 800 m. Two subjects were measured two times for 1500 m. Least squares regression analysis between performance and post-race [LA] generated the trend lines for the respective race distances.

Pre-race and Post-race Lactate

A Pearson correlation analysis was performed to assess the relationship between pre-race [LA] and post-race [LA]. The analysis revealed no significant relationship between the pre-race [LA] (mmol·L⁻¹) and post-race [LA] (mmol·L⁻¹) for 400 m (r=0.620, p>0.05, $\underline{n}=8$), 800 m (r=0.303, p>0.05, $\underline{n}=16$), or 1500 m (r=0.321, p>0.05, $\underline{n}=8$). Regardless of event, post-race [LA] is not significantly related to pre-race [LA]. The results are summarized in Table 5.

Pre-race Lactate and Performance

A Pearson correlation analysis was performed to assess the relationship between pre-race [LA] and performance (average velocity). The analysis revealed no significant relationship between the pre-race [LA] (mmol·L⁻¹) and average velocity of a race (m·sec⁻¹) for 400 m (r=0.228, p>0.05, n=8), 800 m (r=0.320, p>0.05, n=16), or 1500 m · (r=-0.110, p>0.05, n=8). Regardless of event, performance is not significantly related to the pre-race [LA]. The results are summarized in Table 6.

Percent Change in Lactate and Post-race Lactate

A Pearson correlation analysis was performed to assess the relationship between the percent change in [LA] from baseline to pre-race and post-race [LA]. The analysis revealed no significant relationship between the percent change in [LA] and the post-race [LA] (mmol·L⁻¹) for 400 m (r=0.232, p>0.05, n=8), 800 m (r=0.456, p>0.05, n=16), or 1500 m (r=0.456, p>0.05, n=8). Regardless of event, post-race [LA] is not significantly related to the percent change in [LA] from baseline to pre-race. The results are summarized in Table 7.

Total

Pre-race [LA] ($mmol \cdot L^{-1}$) and Post-race [LA] ($mmol \cdot L^{-1}$) Event Trials p ŗ 400 8 0.620 0.101 0.303 0.254 800 16 0.438 0.321 1500 8

Summary of Pearson Correlation Analysis between

Note. 2-400 m, 4-800 m, and 2-1500 m runners each

0.150

0.412

completed four trials in their respective event.

Average Velocity (m•s ⁻¹) and Pre-race [LA] (mmol•L ⁻¹)						
Event	Trials	<u>r</u>	p			
400	. 8	0.228	0.587			
800	16	0.320	0.227			
1500	8	-0.110	0.814			
Total	32	- 0.283	0.117			

Summary of Pearson Correlation Analysis between

Note. 2-400 m, 4-800 m, and 2-1500 m runners each

completed four trials in their respective event.

Summary of Pearson Correlation Analysis between

Percent Change in [LA] from Baseline to Pre-race and

Event	Trials	<u>r</u>	p
400	8	0.232	0.580
800	16	0.456	0.076
1500	8	0.456	0.256
Total	32	0.276	0.126

Post-race [LA] $(mmol \cdot L^{-1})$

Note. 2-400 m, 4-800 m, and 2-1500 m runners each

completed four trials in their respective event.

Percent Change in Lactate and Performance

A Pearson correlation analysis was performed to assess the relationship between the percent change in [LA] from baseline to pre-race and performance (average velocity). The analysis revealed no significant relationship between the percent change in [LA] and average velocity (m·sec⁻¹) for 400 m (r=-0.135, p>0.05, <u>n</u>=8), 800 m (r=0.335, p>0.05, <u>n</u>=16), or 1500 m (r=0.134, p>0.05, <u>n</u>=8). Regardless of event, performance is not significantly related to the percent change in [LA] from baseline to pre-race. The results are summarized in Table 8.

Worst and Best Performance

A 3x4 ANOVA (event x group₁) was performed on each of the DV: performance, pre-race [LA], percent change in [LA] from baseline to pre-race, and post-race [LA], to assess if differences in performance coincided with differences in the corresponding prerace [LA], percent change in [LA] from baseline to pre-race, or post-race [LA]. Event (400, 800, and 1500m) was a between subjects factor, while group₁ (worst (W), intermediate-1 (I-1), intermediate-2 (I-2), and best (B)) was a within subjects factor.

The analysis revealed significant differences in performance between the groups₁ (\underline{F} =10.32, \underline{p} <0.05). Tukey HSD post-hoc analysis revealed significant differences in performance between W (142.5±80.5 s) and B (137.3±78.4 s), W and I-2 (139.0±78.4 s), and I-1 (140.6±79.4 s) and B. There were also significant differences in performance between the events (\underline{F} =830.04, \underline{p} <0.05). A summary of the analysis is presented in Appendix E (Table H).

There were also significant differences in the corresponding post-race [LA] between groups₁ (<u>F</u>=3.54, p<0.05). Tukey HSD post-hoc analysis revealed significant

Summary of Pearson Correlation Analysis between

Average Velocity (m•s⁻¹) and Percent Change in [LA]

	٠		
Event	Trials	<u>r</u>	p
400	8	-0.135	0.749
800	16	0.335	0.205
1500	8	0.134	0.752
Total	32	- 0.158	0.367

from Baseline to Pre-race

Note. 2-400 m, 4-800 m, and 2-1500 m runners each

completed four trials in their respective event.

differences in post-race [LA] between W (12.69±1.65 mmol·L⁻¹) and B (14.65±0.82 mmol·L⁻¹), and W and I-2 (14.22±1.55 mmol·L⁻¹). There were no differences in post-race [LA] between the events (\underline{F} =2.27, \underline{p} >0.05). A summary of the analysis is presented in Appendix E (Table I)

There were no significant differences in the corresponding pre-race [LA] either by group₁ (<u>F</u>=1.05, <u>p</u>>0.05) or by event (<u>F</u>=0.62, <u>p</u>>0.05). Similarly, there were no differences in percent change in [LA] from baseline to pre-race either by group₁ (<u>F</u>=0.67, <u>p</u>>0.05) or by event (<u>F</u>=0.45, <u>p</u>>0.05). Summaries of the analyses for pre-race [LA] and percent change in [LA] are presented in Appendix E (Table J and Table K, respectively).

Despite significant differences in performance between groups₁, there were no differences in the corresponding pre-race [LA] or percent change in [LA] from baseline to pre-race between groups₁. There were, however, significant differences in corresponding post-race [LA] between groups₁. The results are presented in Table 9. Performance was the only DV to vary by event.

Lowest and Highest Pre-race Lactate

A 3x4 ANOVA (event x group₂) was performed on each of the DV: pre-race [LA], post-race [LA], and performance, to assess if differences in pre-race [LA] coincided with differences in the corresponding post-race [LA] or performance. Event (400, 800, and 1500 m) was a between subjects factor, while group₂ (lowest (L), intermediate-1 (I-1), intermediate-2 (I-2), and highest (H)) was a within subjects factor.

The analysis revealed significant differences in pre-race [LA] between groups₂ (<u>F</u>=25.33, p<0.05). Tukey HSD post-hoc analysis revealed significant differences between L (1.52 ± 0.39 mmol·L⁻¹) and H (2.71 ± 0.91 mmol·L⁻¹), L and I-2 (1.95 ± 0.62

Mean and SD for Performance and Corresponding Pre-race [LA], Percent Change in [LA] from Baseline to Pre-race

(% chg. [LA]), and Post-race [LA]	st-race [LA]			
	Performance	Pre-race [LA]	% chg. [LA]	Post-race [LA]
Group ₁	(s)	(mmol•L ⁻¹)	(%)	(mmol•L ⁻¹)
M	$142.5 \pm 80.5_{a}$	$1.97 \pm 0.82_{a}$	$6.5 \pm 48.1_{a}$	$12.69 \pm 1.65_{a}$
I-1	$140.6 \pm 79.4_{a,b}$	$1.82 \pm 0.31_{a}$	$31.4 \pm 41.3_{a}$	$14.07 \pm 0.82_{a,b}$
I-2	$139.0 \pm 78.4_{b,c}$	$1.92 \pm 0.94_{a}$	$15.1 \pm 67.1_{a}$	$14.22 \pm 1.55_{b}$
B	$137.3 \pm 78.4_{\rm c}$	$2.28 \pm 0.89_{a}$	$11.6 \pm 51.0_{\rm a}$	$14.65 \pm 0.82_{\rm b}$
<u>Note.</u> The groups ₁ : w	Note. The groups ₁ : worst (W), intermediate-1 (I-1), intermediate-2 (I-2), and best (B), were formed by rank ordering the four	1), intermediate-2 (I-2), and	d best (B), were formed by	rank ordering the four
performance trials of each subject (<u>N</u> =8)	each subject (<u>N</u> =8) from wo	orst to best based on time.	from worst to best based on time. The corresponding pre-race [LA], post-race [LA], and	[LA], post-race [LA], and

% chg. [LA] from the specific meet in which performance was measured, were assigned to a particular group based solely on rank of the performance for that meet. Means in the same column not sharing a subscript (a,b,c,d) differ at p<0.05 in the Tukey HSD comparison. Critical D's for Performance, Pre-race [LA], % Chg. [LA], Post-race [LA] = 2.46, 0.82, 67.20, 1.43, respectively. mmol·L⁻¹), I-1 (1.82±0.56 mmol·L⁻¹) and H, and I-2 and H. There were no differences in pre-race [LA] between events (<u>F</u>=0.63, p>0.05). A summary of the analysis is presented in Appendix E (Table L).

There were no significant differences in the corresponding post-race [LA] either by group₂ (<u>F</u>=1.92, <u>p</u>>0.05) or by event (<u>F</u>=2.27, <u>p</u>>0.05). There were also no differences in performance between groups₂ (<u>F</u>=0.43, <u>p</u>>0.05). There were, however, differences in performance between events (<u>F</u>=830.04, <u>p</u><0.05). A summary of the analyses for postrace [LA] and performance are presented in Appendix E (Table M and Table N, respectively).

Despite significant differences in pre-race [LA] between groups₂, there were no differences in the corresponding performance or post-race [LA] between the groups₂. The results are presented in Table 10. Performance was the only DV to vary by event.

Smallest and Largest Percent Change in Lactate

A 3x4 ANOVA (event x group₃) was performed on each of the DV: percent change in [LA] from baseline to pre-race, post-race [LA], and performance, to assess if differences in the percent change in [LA] coincided with differences in the corresponding post-race [LA] or performance. Event (400, 800, and 1500 m) was between subjects factor, while group₃ (smallest (S), intermediate-1 (I-1), intermediate-2 (I-2), and largest (L) was a within subjects factor.

The analysis revealed significant differences in the percent change in [LA] between groups₃ (\underline{F} =18.33, \underline{p} <0.05). Tukey HSD post-hoc analysis revealed significant differences between S (-25.6±23.1 %) and L (72.0±57.3 %), S and I-2 (16.6±29.8 %), I-1 (1.6±31.8 %) and I-2, I-1 and L, and I-2 and L. There were no differences in percent

Mean and SD for Pre-race [LA], and Corresponding Post-race [LA] and

Performance

	Pre-race [LA]	Post-race [LA]	Performance
Group ₂	$(mmol \cdot L^{-1})$	$(mmol \cdot L^{-1})$	(s)
L	$1.52 \pm 0.39_{a}$	$13.19 \pm 0.89_{a}$	$140.6 \pm 79.0_{a}$
× I-1	$1.82 \pm 0.56_{a,b}$	$13.44 \pm 1.82_{a}$	$140.8 \pm 78.3_{a}$
I-2	$1.95 \pm 0.62_{b}$	$14.63 \pm 1.40_{a}$	$139.1 \pm 80.6_{a}$
Н	$2.71 \pm 0.91_{\rm c}$	$14.38 \pm 1.09_{a}$	$138.9 \pm 78.8_{a}$

<u>Note:</u> The groups₂: lowest (L), intermediate-1 (I-1), intermediate-2 (I-2), and highest (H), were formed by rank ordering the pre-race [LÅ] from the four meets for each subject (<u>N</u>=8) from lowest to highest pre-race [LA]. The corresponding performance and post-race [LA] from the specific meet in which the pre-race [LA] was measured were assigned to a particular group based solely on the rank of the pre-race [LA] from that meet. Means in the same column not sharing a subscript (a,b,c,d) differ at <u>p</u><0.05 in the Tukey HSD comparison. Critical D's for Pre-race [LA], Post-race [LA], Performance = 0.40, 1.72, 3.90, respectively. change in [LA] between events ($\underline{F}=0.45$, $\underline{p}>0.05$). A summary of the analysis is presented in Appendix E (Table O).

There were no significant differences in the corresponding post-race [LA] either by group₃ (<u>F</u>=0.82, <u>p</u>>0.05) or by event (<u>F</u>=2.27, <u>p</u>>0.05). There were also no differences in the corresponding performance between groups₃ (<u>F</u>=1.02, <u>p</u>>0.05). There was, however, a significant difference in performance between events (<u>F</u>=830.04, <u>p</u><0.05). A summary of the analyses for post-race [LA] and performance are presented in Appendix E (Table P and Table Q, respectively).

Despite significant differences in the percent change in [LA] from baseline to prerace between groups₃, there were no differences in the corresponding post-race [LA] or performance between groups₃. The results are presented in Table 11. Performance was the only DV to vary by event.

Summary

No relationship was found between performance and either pre-race [LA] or percent change in [LA] from baseline to pre-race. These results were further illustrated when despite significant differences in performance between groups₁, no differences were found in the corresponding pre-race [LA] or percent change in [LA] between groups₁. There was, however, a significant difference in corresponding post-race [LA] between groups₁. In addition, a significant relationship was found between performance in 800 m and post-race [LA]. However, post-race [LA] was not related to either pre-race [LA] or percent change in [LA]. Despite significant differences in pre-race [LA] between groups₂, there were no differences in corresponding post-race [LA] between groups₂. Similarly, despite significant differences in percent change in [LA] between groups₃, no

and Corresponding Post-race [LA] and Performance % chg. [LA] Post-race [LA] Pérformance

Mean and SD for Percent Change in [LA] from Baseline to Pre-race (% chg. [LA])

	70 chg. [LA]		renormance		
Group ₃	(%)	$(mmol \cdot L^{-1})$	(s) ·		
S	$-25.6 \pm 23.1_{a}$	$13.27 \pm 1.68_{a}$	$141.4 \pm 80.4_{a}$		
I-1	$1.6 \pm 3\dot{1}.8_a$	$13.72 \pm 1.58_{a}$	$140.2 \pm 78.8_{a}$		
I-2	$16.6 \pm 29.8_{b}$	$14.32 \pm 1.31_{a}$	$138.6 \pm 78.9_{a}$		
L	$72.0 \pm 57.3_{c}$	$14.32 \pm 1.02_{a}$	$139.3 \pm 78.7_{a}$		
Note: The groups ₃ : smallest (S), intermediate-1 (I-1), intermediate-2 (I-2), and					
largest (L), were formed by rank ordering the % chg. [LA] from the four meets for					

each subject (N=8) from lowest to highest % chg [LA]. The corresponding performance and post-race [LA] from the particular meet in which the % chg. [LA] was measured were assigned to a particular group based solely on the rank of the % chg. [LA] from that meet. Means in the same column not sharing a subscript (a,b,c,d) differ at p<0.05 in the Tukey HSD comparison. Critical D's for % Chg. [LA], Post-race [LA], Performance = 40.04, 2.00, 4.31, respectively.

differences were found in corresponding post-race [LA] between groups₃. Collectively, baseline [LA] did not significantly differ from pre-race [LA]. However, [LA] was shown to sometimes increase, and other times decrease from baseline to pre-race. Pre-race HR was consistently higher than baseline HR. There were no differences in age, height, weight, VO₂ max, pre-race [LA], percent change in [LA], or post-race [LA] between events. As expected, performance significantly varied between events.

Chapter 5

DISCUSSION

Introduction

The purpose of this study was to assess the effects of pre-race blood [LA] on the performance of middle distance runners. Results from this study demonstrated that there was no relationship between pre-race blood [LA] and performance in middle distance runners. In addition, there was no relationship between middle distance running performance and the percent change in [LA] due to WU.

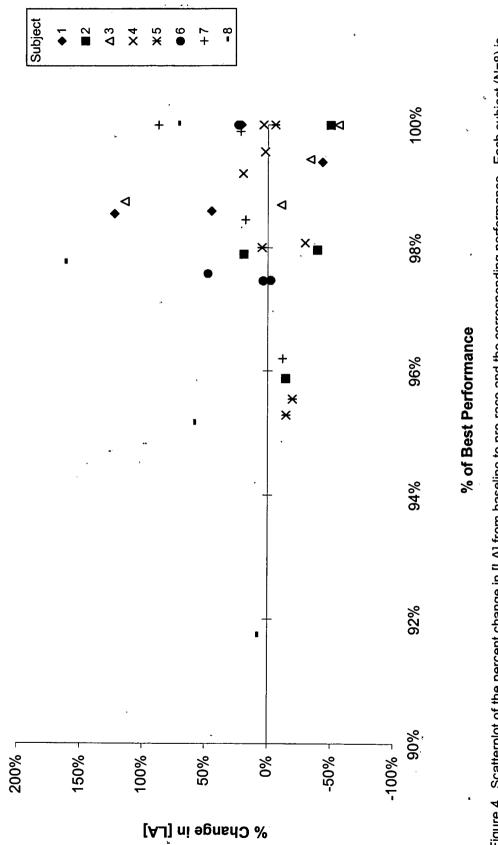
Effects of Warm-up on Lactate

Collectively, over the four meets, there was no difference between baseline and pre-race [LA] (Table 3). However, there was an interaction between measurement and meet (Figure 1). Meet 1 was the only meet where baseline and pre-race [LA] were significantly different. Amiable weather conditions in meet 1 compared to other meets (Table 2), which may have fostered a more intense WU, may explain the significant increase in [LA]. Chronologically, meet 1 was the first meet of the season. It is possible that the athletes were more excited during their WU in preparation for the first race of the season more so than for WU in future meets.

Ranking the four trials for each subject from smallest to largest percent change in [LA] from baseline to pre-race, it becomes evident that in some instances [LA] did change (Table 11). Of importance though, was the fact that there were no differences in performances corresponding to the smallest and largest percent changes in [LA], suggesting that the percent change in [LA] with WU was not predictive of performance.

Unlike the current study, many studies to date have used a structured WU regimen with a resultant increase or no change in [LA] (Martin et al., 1975; Ingjer & Strømme, 1979; De Bruyn-Prevost, 1980; De Bruyn-Prevost & Lefebvre, 1980; Genovely & Stamford, 1982; Robergs et al., 1990; Houmard et al., 1991; Robergs et al., 1991; Mitchell & Huston, 1993). Interestingly, the present study found that WU sometimes resulted in a decreased [LA] from baseline to pre-race (Table 11). Though a decrease was unforeseen, it is not unusual because a decrease in [LA] following physical activity is known to occur for several reasons. Submaximal exercise enhances LA removal (Bonen & Belcastro, 1976; Mazzeo, Brooks, Shoeller, & Budinger, 1986; Bond, Adams, Tearney, Gresham, & Ruff, 1991; Taoutaou et al., 1996), and intensity of exercise and VO₂ are both integral factors in determining the rate of LA removal (Bonen, Campbell, Kirby, & Belcastro, 1979; Mazzeo et al., 1986). Thus, the wide range of change in [LA] from baseline to pre-race (-56% to +161%) as illustrated by Figure 4, could have resulted from different WU activities practiced both within and between subjects. Interestingly, some subjects demonstrated considerable variability in changes in [LA] due to WU between the four trials (Figure 4).

Lactate removal in the body is accomplished mainly through either oxidative or glyconeogenic pathways (Pagliassotti & Donovan, 1990a; Donovan & Pagliassotti, 2000; Brooks, 2000; Gladden, 2000). The majority of LA removal (75-80%) is accomplished through oxidation (Brooks, 2000). The means by which LA is removed during rest and exercise is due in part to muscle fiber type. Type I fibers are responsible for oxidative removal of LA, while type II fibers remove LA via glyconeogenic pathways (Pagliassotti & Donovan, 1990b; Gladden, 2000; Donovan & Pagliassotti, 2000). Thus, the



axis, this includes the best performance for each subject which is represented as 100%. The other three performance scores for each subject represented by a specific symbol. A total of four trials were completed by each subject. Individual performance scores are plotted on the x-Figure 4. Scatterplot of the percent change in [LA] from baseline to pre-race and the corresponding performance. Each subject (<u>N</u>=8) is are represented as a percentage of the individual best pest performance score.

recruitment of muscle fiber to perform submaximal work will facilitate LA removal from both circulation and muscle. Oxidative fibers uptake LA at faster rates and lower thresholds compared to glycolytic fibers (Donovan & Pagliassotti, 2000; Gladden, 2000). Mitochondrial content, and existing levels of pyruvate dehydrogenase, malate-aspartate shuttle enzymes, lactate dehydrogenase (heart type), and monocarboxylate transport enzymes, all influence the oxidative capacity of slow twitch fibers and thus, both the rate of LA removal from circulation and translocation of LA from the muscle (Bonen, 2000; Gladden, 2000). The liver also aids in LA removal through glyconeogenesis (Wasserman, Connolly, & Pagliassotti, 1991).

In the present study, all baseline measures were taken prior to WU and were assumed to be indicative of resting state. Interestingly, baseline [LA] was consistently higher than what is generally accepted as resting level (1.83 vs. 1.00 mmol·L⁻¹). Given that the subjects were athletes, possible consumption of a high carbohydrate meal prior to competition may have affected baseline [LA]. It has been shown that diets high in carbohydrate elevate resting levels of pyruvate, the key intermediate in the mass action driven reaction for LA production (Yoshida, 1984). High carbohydrate diets have also been shown to result in elevated [LA] after competition (Reilly & Woodbridge, 1999).

All blood samples in the current study were drawn from the fingertip. Another factor that may explain the elevated baseline [LA] is the sampling site. Blood taken from the fingertip has been found to have a higher [LA] than blood taken from the ear lobe (Feliu et al., 1999). However, the fact that blood was consistently sampled from the same site should have ensured some degree of standardization between samples from which comparisons were made (Dassonville et al., 1998).

Due to time and equipment limitations associated with field-testing, all blood samples collected in the field were preserved and later analyzed in the lab. The value generated from analysis of a preserved blood sample should have in theory, been multiplied by a factor of three due to dilution, to estimate the actual [LA] in the blood at the time of blood draw. However, regression analysis of pilot data suggested otherwise (Appendix D). It is evident that at a 68% confidence interval, estimating the actual [LA] from a preserved sample may have only been at best within ± 0.51 mmol·L⁻¹.

Effects of Warm-up on Heart Rate

Following WU, pre-race HR was consistently higher than baseline HR (Table 3). Pre-race HR was measured approximately five minutes prior to the start of the race in meets 1 and 2. However, due to complications with recording HR, pre-race HR was measured immediately prior to the start of the race for meets 3 and 4. Figure 2 illustrates higher HR for meets 3 and 4 compared to 1 and 2. The higher HR immediately prior to the start of the race in meets 3 and 4 compared to the HR measured five minutes prior to the start of a race in meets 1 and 2 suggests that physiological adjustments continued after the five minute pre-race sampling period. However, it is not clear if the difference in HR between the two measurement times resulted from continued WU activity or anticipatory rise in HR as the race neared. This variance in HR between the two sampling periods (immediately and five minutes prior to the start of a race) suggests that [LA] measured five minutes prior to the start of a race may not have been indicative of immediate pre-race conditions. Instead, the [LA] may have changed between the time of pre-race sampling and the start of the race.

Effects of Warm-up on Temperature

It was expected that T would increase from baseline to pre-race due to involvement in physical activity during WU (Genovely & Stamford, 1982; Robergs et al., 1991; Stewart & Sleivert, 1998). However, there was no difference between baseline and pre-race T as a result of WU (Table 3). The device which measured tympanic temperature malfunctioned in three of four track meets, therefore temperature measurements were made in only one meet. In addition, tympanic temperature may have been influenced by ambient air conditions as indicated by the rather low range of temperatures $(33.9 - 35.6^{\circ}C)$ compared to normal body temperature $(37.0^{\circ}C)$.

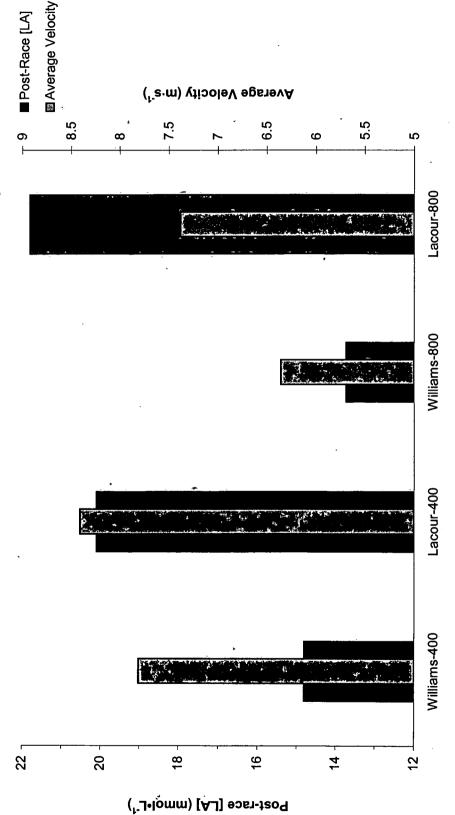
Performance and Corresponding Lactate Parameters

Rank ordering subject performances from worst to best, revealed significant differences between the worst and best performances (Table 9). Despite these differences in performance, there were no differences in the corresponding pre-race [LA] or percent change in [LA] from baseline to pre-race. However, the best performance did coincide with significantly greater post-race [LA] compared to post-race [LA] of the worst performance.

These results are consistent with Lacour et al. (1990) who found a significant correlation of r=0.71 between performance represented as the average velocity (m·sec⁻¹) over 800 m, and post-race [LA] (mmol·L⁻¹) (p<0.01). In the current study this correlation was r=0.86 (p<0.01) (Figure 3, Table 4). However, while Lacour et al. (1990) also demonstrated a significant correlation of r=0.89 between average velocity over 400 m and post-race [LA] (p<0.01), the corresponding correlation in the present study of r=0.70 did not reach statistical significance (p=0.053) (Figure 3, Table 4). The failure of the present study to demonstrate a comparable correlation between average velocity over 400 m and post-race [LA] may reside in the fact that only two subjects competed at this distance compared to four subjects studied by Lacour et al. (1990). Neither study found any significant relationship between average velocity over 1500 m and post-race [LA]:

Athletes in the study by Lacour et al. (1990) were of national caliber, while those in the current study were not. Mean values for both average velocity and post-race [LA] were higher for the national caliber 400 and 800 m runners compared to those in the current study (Figure 5). The combined data from the current study and Lacour et al. (1990) suggests a continuum along which performance in 400 and 800 m running events is largely dependent on anaerobic energy production indicated by peak [LA]. However, it remains unclear whether or not the ability to generate LA, or the ability to accumulate LA before suffering from the adverse metabolic effects of acidosis is most influential in performance.

Robergs et al. (1990), Genovely and Stamford (1982), and Buono and Roby (1982) reported a decrease in peak [LA] as a result of elevated pre-existing [LA]. Thus, it was believed that an inverse relationship existed between pre-race [LA] and post [LA]. This was not observed in the present study. Only Genovely and Stamford (1982) incorporated a quantifiable measure by which to assess performance, where the decrease in peak [LA] that resulted from an elevated pre-existing [LA] hindered performance. It was also hypothesized that the decrease in post-race [LA] resulting from an elevated pre-race [LA] would hinder performance given the close relationship between post-race [LA] and performance over 400 and 800 m. However, there was no apparent relationship



Study-Event

(1990) were members of a national team (high caliber), while subjects in the present study (Williams) were members of a Division III collegiate et al. (1990) for 800 m is representative of a total of 18 trials between five subjects, while four subjects each completed four trials in the current representative of a total of ten trials between four subjects, while two subjects each completed four trials in the current study. Data of Lacour Figure 5. Post-race [LA] and average velocity over 400 and 800 m in high and middle caliber runners. Subjects in the study by Lacour et al. team (middle caliber). The values plotted are mean values from the respective studies. Data of Lacour et al. (1990) for 400 m is study.

between pre-race [LA] and performance at any middle distance, despite the fact that 800 m performance was significantly correlated with post-race [LA].

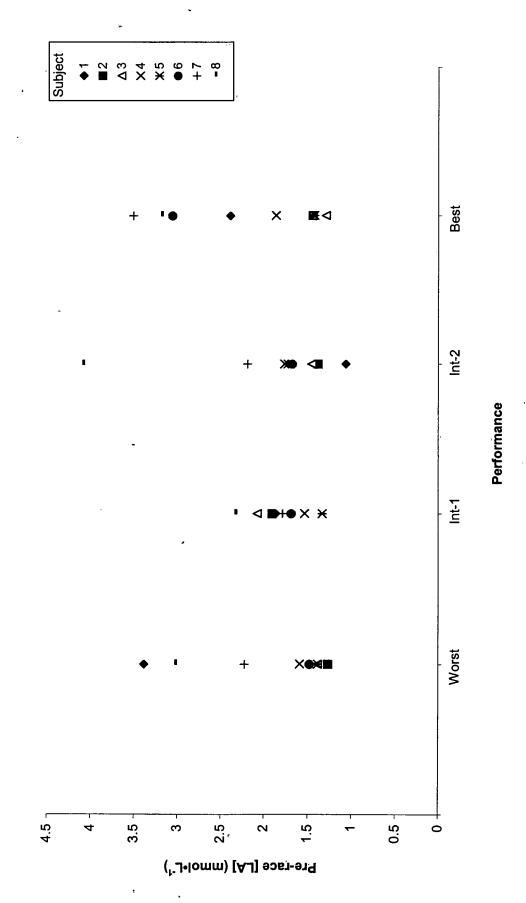
Table 10 illustrates that there were no differences between the corresponding post-race [LA] or performance despite significant differences between lowest and highest pre-race [LA]. The difference between lowest and highest pre-race [LA] in the current study was 1.19 (1.52 to 2.71) mmol·L⁻¹. Whereas the differences between lowest and highest pre-existing [LA] in the studies by Robergs et al. (1990), Genovely and Stamford (1982), and Buono and Roby (1982) were: 1.4 (1.7 to 3.1), 3.0 (1.2 to 4.2), and 4.48 (1.11 to 5.59) mmol·L⁻¹, respectively. The smaller range of values observed in the present study may explain why peak [LA] failed to decrease with an increase in pre-existing [LA]. However, Mitchell and Huston (1993) and Robergs et al. (1991) reported findings similar to the present findings despite greater differences between lowest and highest preexisting [LA] than the current study. Corresponding performance and peak [LA] were similar despite a difference in pre-existing LA of 4.59 (1.56 to 6.15) mmol·L⁻¹ (Mitchell & Huston, 1993). Similarly, Robergs et al. (1991) found there was no effect on peak [LA] as a result of difference in pre-existing [LA] of 4.0 (1.7 to 5.7) mmol·L⁻¹.

Since all previous studies pertaining to WU demonstrated either no change or an increase in [LA] following WU, a decrease in [LA] has never been a benefit associated with WU let alone a response to WU. However, a decrease in [LA] with WU, could be justly labeled both a potential response to WU as well as a benefit due to the fact that some subjects demonstrated a decrease in [LA] with WU, which coincided with a best performance (Figure 4). Figure 4 shows that some subjects performed best with a decrease in [LA] from baseline to pre-race, while others performed best with an increase

or minimal change. It is possible that the prevalence of decreased [LA] with WU as well as the variability between subjects for percent change in [LA] with WU corresponding to the best performance may have resulted from varied baseline [LA]. However, with regards to an absolute value, Figure 6 illustrates considerable variability between subjects for the pre-race [LA] that coincided with the worst performance (1.27-3.39 mmol·L⁻¹) as well as the best performance (1.30-3.52 mmol·L⁻¹). The present data indicate there to be no optimal percent change in [LA] with WU as well as no optimal pre-race [LA] for the collective performance enhancement of middle distance runners in this study. However, the results do suggest optimal WU-induced modifications in [LA] as well as optimal prerace [LA] are likely to be highly individual between athletes.

Summary

Performance in the 800 m track event is strongly related to peak [LA]. Collectively, there is no optimal pre-race [LA] or percent change in [LA] with WU for enhancing middle distance running performance. Instead, performance enhancement through WU appears to be highly individual.



Intermediate-1 (Int-1) and Intermediate-2 (Int-2) were the second slowest and second fastest performances, respectively. Each subject is Figure 6. Performance and corresponding pre-race [LA]. Each subject (<u>N</u>=8) completed a total of four trials. Individual performance was ranked within each subject from slowest (Worst) to fastest (Best) according to time, and plotted against the corresponding pre-race [LA] represented by a specific symbol.

Chapter 6

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

This study was concerned with assessing the effects of pre-race blood [LA] on performance in middle distance runners. The study was unique in that all measurements were taken during actual competition while other studies pertaining to WU occurred in a laboratory environment. Not only were the subjects allowed to WU as they normally would in preparation for competition, but subsequent performance was measured in the race during actual competition toward which the subjects geared their WU. Though there was minimal intervention by the researchers in terms of standardizing and controlling the WU, therefore limiting the ability to generalize and describe a specific, optimal WU, the results must be considered nonetheless authentic.

Collectively, neither pre-race [LA] nor the percent change in [LA] with WU, predicted performance. In addition, peak [LA] was not affected by either pre-race [LA] or percent change in [LA] with WU. Peak [LA] was, however, strongly related to performance in 800 m. Interestingly, WU sometimes resulted in an unforeseen decrease in [LA] from baseline to pre-race, which coincided with the best performance for some subjects; whereas other subjects performed best with an increase or minimal change in [LA] with WU. In addition, there was considerable variability between subjects for prerace [LA] corresponding to worst and best performances.

Conclusions

Almost by default, it is common to think that there is one 'optimal' condition under which the best results are produced. In accordance with this line of thought,

studies have typically implemented a WU regimen intended to be optimal for performance enhancement of all subjects. However, as the current study has suggested, and given the likely diversity of each individual, it is plausible that what is optimal for one athlete may not be for another.

The likelihood for a high degree of individuality with regards to optimal WU for performance enhancement is great. The key to which may reside in muscle fiber characteristics of an individual. To date, seven fiber types have been identified in human skeletal muscle. However, coupled with differential degrees of adaptability between individuals, the variability of fiber characteristics within and between individuals potentiate the existence of "innumberable fiber type transients" (Simoneau & Bouchard, 1989; Staron, 1997).

Just as the same WU activity may elicit differential responses between individuals, different pre-existing [LA] may result in variant performance outcomes between individuals, as was suggested by this study. The ability to influence performance through WU is dependent not only on the nature of the WU activity, but also the metabolic profile of musculature, which is likely to be highly variable between individuals. On one extreme, an increase in LA with WU may benefit performance in individuals with an enhanced capacity to clear LA. The increase in LA may activate or prime the pathways for LA removal such that at the onset of a CE, LA does not immediately accumulate and cause a disturbance in homeostasis, but instead is promptly removed. On the other extreme, individuals with less enhanced capacities for LA removal, may limit their performance through an increase in LA with WU. An increase in LA prior to a CE, which may not be readily cleared, could disturb homeostasis and ultimately limit glycolytic energy production and hinder performance. Regardless, the optimal WU for performance enhancement in middle distance running, suggested by the findings in the present study, is likely to be unique for each athlete.

<u>Recommendations</u>

Though sampling blood immediately prior to commencement of a race during actual competition is difficult, attempts to do so would be more indicative of immediate pre-race conditions without influence from further WU or time lag. In addition, measuring other parameters such as blood pH, may help further understand the influence of WU in modifying such variables as well as the effects on performance due to the precompetition conditions.

Ambient outdoor conditions during the present study may have not only influenced WU, but also affected performance. Therefore, minimizing any such influences from environmental conditions by assessing WU in a competitive indoor environment may yield interesting results.

Further investigation is needed to understand the high degree of individuality of optimal WU. In addition, examining the repeatability of WU in a competitive setting is warranted. The present findings illustrate wide variability for some subjects across the four trials with regards to responses elicited with WU, which may serve to explain variability in performance. However, it is unknown whether or not the variance in responses within a subject was due to intentional manipulation of WU. Future research efforts on WU may also consider assessing the perceived efficacy of WU and subsequent performance. The psychology of preparedness for a race may influence performance as well.

APPENDIX A

Coach's Consent Form

Dear Coach Nichols,

As part of my master's program here at Ithaca College I am required to complete a thesis. I am interested in the effects of warm-up on performance. I would like to request permission from you to meet with your athletes to seek volunteers.

The proposed study will investigate the effects of pre-race blood lactate levels on performance in middle distance runners. The subjects sought for the study will be those athletes who will race 400, 800, or 1500 m during the 2000 outdoor season. The study will have two phases.

The first phase will entail testing the maximum oxygen consumption of the subjects at least 72 hours prior to the first scheduled contest. This test will require the subject to run until volitional exhaustion on a treadmill at a self-determined speed. The grade of the treadmill will increase 3% every 2 minutes starting at a grade of 0%. A small 25 μ l sample of blood will be drawn from the fingertip of the subject 7.5 minutes after the test for analysis of blood lactate.

The second phase will take place during those meets prior to the NYSCTC Outdoor Championships. At these meets the measurements that will be made are heart rate, tympanic (ear) temperature, and blood lactate. The group of variables (heart rate, body temperature, and blood lactate) will be measured at three times during these meets for each subject. The first set of measurement will be taken prior to the start of the athletes warm-up routine, the second set of measurements will be taken 5 minutes prior to the start of the race, and the final set of measurements will be taken within 7.5 minutes after the completion of the race. Each measurement of blood lactate will require a small 25 μ l sample of blood to be drawn from the fingertip of the subject. Heart rate will be measured with the use of a heart rate monitor. Temperature will be taken with a device that easily measures temperature in the ear canal.

Participation will be entirely voluntary and your athletes are free to dropout at any time without prejudice should they choose to do so. Your athletes will be free to warmup as they see fit. The names of the subjects will not be used in any papers or presentations that may arise from this study in order to maintain confidentiality. Results will be available to your athletes upon completion of the study.

I would appreciate your consent to seek volunteers from the track and field team, and will gladly answer any questions you may have.

Yours in Sport,

Scott Williams 277-5263 swillia1@ic3.ithaca.edu

Coach's Consent Form

_____ I give permission for my athletes to be in your study.

_____ I do not give permission for my athletes to be in your study.

Signed:

Date:

APPENDIX B

Informed Consent Form

Pre-race blood lactate levels and performance in middle distance runners.

1. Purpose of the study:

The purpose of the study is to assess the influence that warm-up has on performance, specifically investigating the effects of pre-race blood lactate on performance in middle distance runners.

2. Benefits of the study:

By participating in the study you will learn what your maximum oxygen consumption is (VO₂ max), a commonly used index of training status, and a test which can cost about \$150 on the open market. Secondly, this data may help with guiding your warm-up in future seasons in order to try and replicate those responses of a particular warm-up that may have led to an enhanced performance while avoiding those that may have deterred performance.

3. Your Participation Requires:

The first testing session will require you to report to the laboratory to complete a VO_2 max. In this test you will run on a treadmill at a speed determined by yourself. Every 2 minutes the grade of the treadmill will increase by 3% starting at a grade of 0%. The test will be terminated when you feel you can no longer continue. During the test you will be required to wear a headgear device with a breathing valve so that we can measure your expired gases. Your heart rate will be monitored throughout the test as will your rating of perceived exertion. 7.5 minutes after the conclusion of the test, we will prick your fingertip to draw a small sample of blood (25 μ l) to determine your blood lactate level.

The subsequent testing sessions will take place at those meets prior to the NYSCTC Outdoor Championships. During these meets, the set of variables: heart rate, body temperature, and blood lactate, will be measured three times. The first set of measurements will be taken prior to you commencing any warm-up routine for your race. The second set of measurements will be taken at 5 minutes prior to the start of your race. The final set of measurements will be taken within 7.5 minutes after the completion of the race. Blood lactate measurements will be taken as previously described while heart rate will be measured with heart rate monitors, and body temperature with a device that measures temperature in your ear canal. Your performance will also be recorded and represented as a time.

4. **Risks of Participation:**

You may feel some muscle soreness 24 to 48 hours after the VO₂ max test. Due to the vigorous nature of the VO₂ max test, there exists a small chance of musculoskeletal injury, lightheadedness, nausea, or even death (1 in 10,000 tests). In addition, you may experience some soreness in your fingertip(s) where the blood was drawn. As with any sampling of blood, there is a small chance of infection, for which proper sterilization procedures will be taken to prevent.

5. If You Would Like More Information about the Study:

For information at any time prior to, during, or after the study contact either Scott Williams at 277-5263, e-mail: swillia1@ic3.ithaca.edu; or Betsy Keller at 274-1683, e-mail: keller@ithaca.edu

6. Withdrawal from the Study:

You are free to withdraw from this study at any time without prejudice.

7. Confidentiality:

All data collected will be coded to insure your confidentiality. Your name will not appear in any reports from this study.

I have read and understood the above document. I agree to participate in this study and realize that I can withdraw at anytime. I also understand that I can and should address questions related to this study at any time to the researchers involved. I also verify that I am at least 18 years of age.

Name of Subject (please print)

Signature of Subject

Date

APPENDIX C

U.S.O.C. Sports Physiology Dept. Lactic Acid Analysis Preparation of Buffer and "Cocktail"

Supplies

- 200 μl 500 μl microcentrifuge tubes with cap. We use Curtin Matheson Scientific Inc., # 068-742 (500 μl). Be careful not to order tubes that are too tall or too thin.
- Triton X-100 (Sigma Chemical Co. # T-6878)
- YSI phosphate buffer (YSI # 2357)
- Sodium fluoride-NaF Anhydrous MW 42.0 (Sigma Chem. Co. # S-1504)
- 1 ml Tuberculin Syringe (Becton, Dickison & Co. # 5602) No needle.
- 30 ml Nalgene bottle
- 25 μl syringepet (micro pipet) (YSI # 2361)

Procedures

Buffer: to flush the 23L YSI analyzer and as base for "cocktail".

- 1) Empty contents of one Buffer 7C package into a 500 ml YSI mixing bottle.
- 2) Add 450 ± 25 ml distilled water and shake vigorously.

Note- it is NOT necessary (nor advisable) to add the detergent (Triton X-100) or the preservative (NaF) to this stock. The effect of Triton X-100 on the analyzer membrane is unknown.

This stock should probably not be used more than one week.

- "Cocktail": stock solution to fill microcentrifuge tubes. Breaks red blood cells and inhibits lactate conversion to pyruvate until samples can be analyzed.
 - 1) Add appropriate volume of stock buffer in 30 ml clear Nalgene bottle (see table below).
 - 2) Use tuberculine syringe to add triton X-100 to solution.
 - 3) Empty NaF from microcentrifuge tube into bottle and shake.

Tubes Needed	Stock Buffer	<u>Triton X-100</u>	<u>NaF</u> *
200	10.0 ml	0.022 ml	Approx. ¹ / ₂ tube
500	25.0 ml	0.055 ml	1 tube
1000	50.0 ml	0.110 ml	2 tubes

*NaF is firmly packed into a 500 µl microcentrifuge tube

Prior to testing, pipet 50 μ l of "cocktail" into microcentrifuge tubes. Cap tubes until ready to use. Add 25 μ l of blood being careful to mix blood with "cocktail" by flushing "pipet into mixture a few times. Cap tubes.

Blood samples (25 μ l blood to 50 μ l "cocktail") can be stored 1-3 days at room temperature or up to 10 days refrigerated.

APPENDIX D

Summary of	of Linear R	egression for Pre	dicting Tru	e Blood [LA] from Analysis
of [LA] in	Blood Mix	ed with Preserva	tive	· · · · · · · · · · · · · · · · · · ·
ŗ	<u>R</u> ²	adjusted $\underline{R^2}$	<u>SEE</u>	Regression line equation
0.99	0.99	0.99	0.51	y = 2.52x + .03

<u>Note.</u> Twenty pairs of samples were used in the regression analysis. Each pair consisted of a non-preserved blood sample and a preserved blood sample, both of which were drawn from the same site at the same time.

Blood Sample-Regression Raw Data

Blood Draw	Immediate Sample (mmol/L)	Cocktail Sample (mmol/L)
1	11.20	4.39
2	9.37	4.02
3	5.07	2.15
4	13.59	5.53
5	13.71	5.70
6	8.99	3.73
7	10.86	4.33
8	13.87	5.25
9	10.25	4.35
10 ⁻	14.01	5.15
11	2.22	0.96
12	· 9.15	3.31
13	, 7.85	2.95
14	7.07	2.57
15	1.14 .	0.47
16	1.02	0.43
17	1.14	0.41
18	6.04	2.52
19	1.81	0.74
20	0.93	0.40

Table A

One-way ANOVA Summary Table for Age (years) of Subjects (N=8)

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Grouped By Event

Source	<u>SS</u>	<u>df</u>	MS	Critical <u>F</u>	Critical \underline{F} Obtained \underline{F}	먼
Between	6.75	5	3.37	5.79	5.19	0.060
Within	3.25	S	0.65		-	
Total	10.00	٢				v
<u>Note.</u> IV: Event (400, 800, 1500 m); DV: Age.	(400, 800, 150(0 m); DV: Ag	e.			r -

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Table B

One-way ANOVA Summary Table for Height (cm) of Subjects (N=8)

Grouped By Event

Source	<u>SS</u>	df	<u>WS</u>	Critical <u>F</u>	Critical \underline{F} Obtained \underline{F}	ਰ
Between	10.27	2	5.14	5.79	 0.13 	0.885
Within	205.99	.	41.20			
Total	216.26	7				
Note. IV: Event (4)	(400, 800, 1500 m); DV: Height.	ı); DV: Height.				

Table C

One-way ANOVA Summary Table for Weight (kg) of Subjects (N=8)

Grouped By Event	

			3			
Source	<u>ŠS</u>	df	<u>WS</u>	Critical <u>F</u>	Critical \underline{F} Obtained \underline{F}	਼ੁਰ
Between	15.17	2	7.58	5.79	1.03	0.442
Within	36.77	5	7.35			•
Totàl	51.94	٢				
Note NV: Briant (100 800 1500 m); DV: Weicht		1.110.10	Maiaht			

Note. IV: Event (400, 800, 1500 m); DV: Weight.

Table D

One-way ANOVA Summary Table for VO2 max (ml·kg⁻¹·min⁻¹) of

Subjects (N=8) Grouped By Event

	Underts (11-0) Oronped by Even	Π			ų	
	SS	df	WS,	Critical <u>F</u>	Critical \underline{F} Obtained \underline{F}	đ
	63.37	. 2	31.69	5.79	1.64	0.283
	96.45	S	19.29			
	159.82	٢				
ant (Note IV: Event (100 800 1500 m): DV: VO mov	V. VU. (m. (

<u>Note.</u> IV: Event (400, 800, 1500 m); DV: VO₂ max.

(2x4) Repeated Measures ANOVA Summary Table of Baseline and Pre-race [LA]	JOVA Summa	rry Table of	<u>Baseline an</u>	1 Pre-race [LA	Ţ	
Source	<u>SS</u>	<u>df</u>	<u>WS</u>	Critical <u>F</u>	Obtained F	ದ
Subject	5.53	7	0.79	·		
Measurement	0.47	1	0.47	5.59	0.69	0.432
Subj x Measurement	4.70	٢	0.67			÷
Meet	1.30	°,	0.43	3.07	, 1.39	0.275
Subj x Meet	6.56	21	0.31			
Measurement x Meet	4.01	3	1.34	3.07	7.75	0.001
Residual	3.62	21	0.17			
Note. IV: Measurement (baseline, pre-race), Meet (1-4); DV: Pre-race [LA].	eline, pre-race	e), Meet (1-4); DV: Pre-	race [LA].		

Table E

Table F

(2x4) Repeated Measures ANOVA Summary Table of Baseline and Pre-race HR

Source	SS	<u>df</u>	<u>WS</u>	Critical <u>F</u>	Obtained \overline{F}	ੇ ਦ ਾ
Subject	2955.50	L	422.21			
Measurement	49952.25	1	49952.25	5.59	145.86	0.000
Subj x Measurement	2397.25	٢	342.46			
Meet	2412.13	, m	804.04	3.07	7.58	0.001
Subj x Meet	2227.38	21	106.07	`		
Measurement x Meet	1130.38	3	376.79	3.07	3.62	0.030
Residual	2187.13	21	104.15			
Note. IV: Measurement (baseline, pre-race), Meet (1-4); DV: Pre-race HR.	baseline, pre-race	(), Meet (1-4);	DV: Pre-race HR			

APPENDIX E (continued)

Table G

Summary of Dependent T-test Comparing Baseline and Pre-race

Temperature (T)

Variable	<u>df</u>	Critical <u>t</u>	Obtained <u>t</u>	p
Т	7	2.37	-0.85	0.423

Table H

(3x4) ANOVA Summary Table of Performance with Repeated Measures on Group,

			-			
Source	SS	<u>df</u>	W	Critical \underline{F} .	Obtained \underline{F}	, ਰ
Event	174967.21	2	87483.60	5.79	830.04	0.000
Subj w event	526.98	S	105.40			
Group	90.18	3	30.06	3.29	10.32	0.001
Event x Group ₁	31.51	9	5.25	2.79	1.80	0.166
Residual	43.71	15	2.91		,	
<u>Note.</u> IV: Event (400, 800,		oup ₁ (worst	1500 m), Group ₁ (worst, int-1, int-2, best); DV: Performance.	DV: Performanc		

(3x4) ANOVA Summary Table of Post-race [LA] with Repeated Measures on Group,	ry Table of Po	st-race [LA]	with Repea	ited Measures	<u>on Group</u> 1	
Source	<u>SS</u>	<u>df</u>	<u>SM</u>	Critical <u>F</u>	Obtained \underline{F}	ਰ
Event	9.49	5	4.75	5.79	2.27	0.199
Subj w event	10.46	S	2.09			
Group1	10.40	, С	3.46	3.29	3.54	0.040
Event x Group ₁	10.81	9	1.80	2.79	1.85	0.157
Residual	14.64	15	0.98			
<u>Note.</u> IV: Event (400, 800, 1500 m), Group ₁ (worst, int-1, int-2, best); DV: Post-race [LA].	800, 1500 m),	Group ₁ (wo	rst, int-1, in	t-2, best); DV:	Post-race [LA]	

Table I

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Table	

(3x4) ANOVA Summary Table of Pre-race [LA] with Repeated Measures on Group₁

Source	SS	df	<u>WS</u>	Critical <u>F</u>	Critical \underline{F} Obtained \underline{F}	<u>ମ</u> ୍ଭ
Event	1.88	2	• 0.94	5.79	0.62	0.576
Subj w event	7.61	5	1.52			;
Group ₁	1.02	3	0.34	3.29,	1.05	0.400
Event x Group ₁	2.74	9	0.46	2.79	1.41	0.275
Residual	4.88	15	0.33			
Note. IV: Event (400, 800, 1500 m), Group ₁ (worst, int-1, int-2, best); DV: Pre-race [LA].	0, 800, 1500	m), Group	1 (worst, int	-1, int-2, best);	DV: Pre-race []	LA].

Table K

(3x4) ANOVA Summary Table of Percent Change in [LA] from Baseline to Pre-race Values

with Repeated Measures on Group,

Source Site Stritical F Obtained F P Event 3946.59 2 1973.30 5.79 0.45 0.661 Event 3946.59 2 1973.30 5.79 0.45 0.661 Subj w event 21917.88 5 4383.58 0.651 0.583 Group1 4365.70 3 1455.23 3.29 0.67 0.583 Fvent x Group1 19447.03 6 3241.17 2.79 1.49 0.246 Residual 32557.13 15 2170.48 2.70 1.49 0.246 Note. IV: Event (400, 800, 1500 m), Group, (worst, int-1, int-2, best); DV: Percent Change in [LA]. 1.40 1.49 1.40	with Acpeated Inteasures on Oroup	TICS OIL OTOUP1	• ,				
Event3946.5921973.305.790.450.661Subj w event21917.8854383.580.670.583Group14365.7031455.233.290.670.583Event x Group119447.0363241.172.791.490.246Residual32557.13152170.48 </th <th>Source</th> <th><u>SS</u></th> <th>df .</th> <th>W</th> <th>Critical <u>F</u></th> <th>Obtained <u>F</u></th> <th>a</th>	Source	<u>SS</u>	df .	W	Critical <u>F</u>	Obtained <u>F</u>	a
Subj w event 21917.88 5 4383.58 Group1 4365.70 3 1455.23 3.29 0.67 0.583 Event x Group1 19447.03 6 3241.17 2.79 1.49 0.246 Residual 32557.13 15 2170.48 1.49 0.246	Event	3946.59	7	1973.30	5.79	0.45	0.661
Group1 4365.70 3 1455.23 3.29 0.67 0.583 Event x Group1 19447.03 6 3241.17 2.79 1.49 0.246 Residual 32557.13 15 2170.48 Note. IV: Event (400, 800, 1500 m), Group1 (worst, int-1, int-2, best); DV: Percent Change in [LA].	Subj w event	21917.88	S	4383.58			
Event x Group1 19447.03 6 3241.17 2.79 1.49 0.246 Residual 32557.13 15 2170.48 Note. IV: Event (400, 800, 1500 m), Group1 (worst, int-1, int-2, best); DV: Percent Change in [LA].	Group1	4365.70	e.	1455.23	3.29	0.67	0.583
Residual 32557.13 15 2170.48 Note. IV: Event (400, 800, 1500 m), Group ₁ (worst, int-1, int-2, best); DV: Percent Change in [LA].	Event x Group ₁	19447.03	9	3241.17	2.79	1.49	0.246
Note. IV: Event (400, 800, 1500 m), Group ₁ (worst, int-1, int-2, best); DV: Percent Change in [LA].	Residual	32557.13	15	2170.48			
	Note. IV: Event (400	, 800, 1500 m), Gi	oup ₁ (worst	t, int-1, int-2,	best); DV: Per	rcent Change in	[[TA

APPENDIX E (continued)

(3x4) ANOVA Summary Table of Pre-race [LA] with Repeated Measures on Group ₂	<u>mmary Tabl</u>	e of Pre-race	e [LA] with F	<u>Repeated Measur</u>	es on Group ₂	
Source	SS	<u>df</u>	<u>WS</u>	Critical <u>F</u>	Obtained F	đ
Event	1.90	2	0.95	5.79	0.63	* 0.573
Subj w event	7.61	5	0.52			
Group ₂	5.84	ю	1.95	3.29	25.33	0.000
Event x Group ₂	1.17	6	0.19	2.79	2.53	0.068
Residual	1.15	15	0.08			
Note. IV: Event (400, 800, 1500 m) Group, (lowest, int-1, int-2, highest); DV: Pre-race [LA].	100, 800, 15	00 m) Grout	o, (lowest, in	t-1, int-2, highes	t); DV: Pre-race	[LA].

Table L

APPENDIX E (continued)

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Source	SS	<u>df</u>	<u>WS</u>	Critical <u>F</u>	Obtained \underline{F}	ਰ
Event	9.49	2	4.75	5.79	2.27	0.199
Subj w event	10.46	5	2.09		t	
Group ₂	8.17	б	2.72	- 3.29	1.92	0.170
Event x Group ₂	9.46	9	1.58	2.79	1.11	0.401
Residual	, 21.27	15	1.42			
Note. IV: Event (400, 800, 1500 m) Group ₂ (lowest, int-1, int-2, highest); DV: Post-race [LA].	0, 800, 1500 r	n) Group ₂	(lowest, int-1	l, int-2, highest);	DV: Post-race [[A].

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Table N

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Source	<u>SS</u>	df	<u>MS</u>	Critical <u>F</u>	Obtained <u>F</u>	ਕ
Event	174967.21	5	87483.60	5.79	830.04	0.000
Subj w event	526.98	5	105.40			
Group ₂	9.35	Э	3.12	3.29	0.43	0.737
Event x Group ₂	56.75	9	9.46	2.79	1.30	0.317
Residual	109.47	15	7.30			
Note. IV: Event (400, 800, 1500 m) Group, (lowest. int-1. int-2. highest): DV: Performance.	0.800.1500 m) Gr	oun, (lowest	int-1. int-2. highe	st): DV: Perform	lance.	

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Table O

(3x4) ANOVA Summary Table of Percent Change in [LA] from Baseline to Pre-race Values

with Repeated Measures o	rres on Group ₃					£
Source	SS	<u>df</u>	WS	Critical <u>F</u>	Obtained <u>F</u>	đ
Event	3946.59	5	1973.30	5.79	0.45 .	0.661
Subj w event	21917.88	S	4383.58			
Group ₃	42391.90	ß	14130.63	3.29	18.34	0.000
Event x Group ₃	2613.03	9	435.51	2.79	0.57	0.752
Residual	11558.13	15	770.54`			
Note. IV: Event (400, 800, 1500 m), Group ₃ (smallest, int-1, int-2, largest); DV: Percent Change in [LA].), 800, 1500 m), G	roup ₃ (smal	llest, int-1, int-2, la	rgest); DV: Perce	ent Change in [LA]	

(3x4) ANOVA Summary Table of Post-race [LA] with Repeated Measures on Group ₃	<u>mary Table of I</u>	<u>ost-race [L</u>	A] with Repe	ated Measures	on Group ₃	
Source	<u>SS</u>	<u>df</u>	MS	Critical \underline{F}	Obtained <u>F</u>	đ
Event	9.49	2	4.75	5.79	2.27	0,199
Subj w event	10.46	'n,	2.09			
Group ₃	4.71	ю	1.57	3.29	0.82	0.504
Event x Group ₃	7.61	9	1.27	2.79	0.66	0.682
Residual	28.79	15	1.92			
Note. IV: Event (400, 800, 1500 m), Group ₃ (smallest, int-1, int-2, largest); DV: Post-race [LA].	0, 800, 1500 m), Group ₃ (s	mallest, int-1	, int-2, largest)	; DV: Post-race	[LA].

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Table P

Table Q

0.000 0.411 0.869 a Obtained \underline{F} 830.04 1.02 0:40 Note. IV: Event (400, 800, 1500 m), Group₃ (smallest, int-1, int-2, largest); DV: Performance. Critical **F** 3.29 2.79 5.79 105.40 9.12 3.55 8.93 87483.60 <u>MS</u> 15 9 2 Ś df \mathbf{c} 21.30 133.99 526.98 27.35 174967.21 SS Event x Group₃ Subj w event Group₃ Source Residual Event

(3x4) ANOVA Summary Table of Performance with Repeated Measures on Group₃

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APPENDIX E (continued)

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APPENDIX F

Raw Data Key

VO ₂ Max	Maximum oxygen consumption.
Max HR	HR upon completion of VO_2 max test.
Max LA	[LA] upon completion of VO_2 max test.
Event	Track event: 1=400m, 2=800m, 3=1500m.
Meet	Track meet: 1=Rochester Quad, 2=Ithaca Invite, 3=Moravian Invite, 4=Rochester Invite.
ACHGHRBP	Absolute change in HR from baseline to pre-race.
PCHGHRBP	Percent change in HR from baseline to pre-race.
ACHGHRPP	Absolute change in HR from pre-race to post-race.
PCHGHRPP	Percent change in HR from pre-race to post-race.
ACHGLBP	Absolute change in [LA] from baseline to pre-race.
PCHGLBP	Percent change in [LA] from baseline to pre-race.
ACHGLPP	Absolute change in [LA] from pre-race to post-race.
PCHGLPP	Percent change in [LA] from pre-race to post-race.
Performance	Time necessary to complete a race at given distance.
Avg.Vel.	Average velocity maintained over a given race distance.

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Raw Data

Subject	Age	Height	Weight	VO2 max	Max HR	Max LA	Event	Meet	Baseline HR
L	(years)	(cm)	(km)	(ml/kg/min)	(bpm)	(mmol/L)			(bpm)
1	19	172.7	69.5	70.48	193	13.87	3	1	60
1	19	172.7	69.5	70.48	193	13.87	3	2	72
1.	19	172.7	69.5	70.48	193	13.87	3	3	60
1	19	172.7	69.5	70.48	193	13.87	3	4	80
2	19	175.3	70.9	58.43	190	8.99	2	1	66
2	19	175.3	70.9	58.43	190	8.99	2	2	72
2	19	175.3	70.9	58.43	190	8.99	2	3	60
2	19	175.3	70.9	58.43	190	8.99	2	4	60
3	22	188	73.6	56.97	194	9.37	1	1	78
3	22	188	73.6	56.97	194	9.37	_ 1	2	72
3	22	188	73.6	56.97 .	194	9.37	1	3	78
3	22	188	73.6	56.97	194	9.37	1	/ 4	72
4	21	170	68.2	55.42	198	10.86	1	1	72
4	21	170	68.2	55.42	198	10.86	1	2	76
4	21	170	68.2	55.42	198	10:86	1	3	78
4	21	170	68.2	55.42	198	10.86	1	4	78
5	19.	175.3	68.2	60.46	184	10.25	2	1	54
5	19	175.3	68.2	60.46	184	10.25	2	2	66
5	19	175.3	68.2	60.46	184	10.25	2	3	66
5	19	175.3	68.2	60.46	184	10.25	2	4	78
6	19	180	73.2	62.66	197	14.01	2	1	60
6	19	180	73.2	62.66	197	14.01	2	2	78
6	19	180	73.2	62.66	197	14.01	2	3	78
6	19	180	73.2	62.66	197、	14.01	2	4	72
7	21	180	66.8	57.80	195	11.20	3	1	72
7	21	180	66.8	57.80	195	11.20	3	2	78
7	21	180	66.8	57.80	195	11.20	3	3	72
7	21	180	66.8	57.80	195	11.20	3	4	72.
8	20	175	73.6	57.71	207	13.71	2	1	60
8	20	175	73.6	57.71	207	13.71	2	2	78
8	20	175	73.6	57.71	207	13.71	2	3	72
8	20	175	73.6	57.71	207	13.71	2	4	84

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Raw Data

Subject	Event	Meet	Pre-race HR	ACHGHRBP	PCHGHRBP	Post-race HR	ACHGHRPP	
		(bpm)	(bpm)	(%)	(bpm)	(bpm)		
1	3	1	97	37	62	,192	95	
1.	3	2	104	32	44	184	80	
1	3	3	120	60	100	170	50	
1	3	4	145	65	81	195	50	
2	2	1	125	59	89	191	66	
2	2	2	95	23	32	186	91	
2	. 2	3	142	82	137	191	. 49	
. 2	2	4	140	80	133	190	50	
3 3	1	1	104	26	33	170	66	
3	1	2	102	30	42	186	84	
3	1	3	135	57	73	184	49	
3	1	4	135	63	88	174	39	
4	1	1	83	11	15	195	112	
4	1	2	106	30	39	205	99	
4	1	3	128	50	64	198	70	
4	1	4,	130	52	67	194	64	
5	2	1	98	44	81	190	⁻ 92	
5	2	2	138	72	_ 109	192	54	
5	2	3	113	47	71	183	70	
5	2	4	121	43	55	180	59	
6	2	1	135	75	125	196	61	
6	2	2	140	62	79	191	51	
6	2	3	140	62	79	187	47	
6	2	4	150	78	108	196	· 46	
7	3	1	150	78	108	194	44	
7	3	2	140	62	79	195	55	
7	3	3	155	83	115	195	40	
7	3	4	144	72	100	190	46	
8	2	1	135	75	125	191	56	
8	2	2	126	48	· 62	198	, 72	
8	2	3	135	63	88	199	64	
8	2	4	151	67	80	206	55	

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Raw Data

Subject	Event	Meet	PCHGHRPP	Baseline LA	Pre-race LA	ACHGLBP	PCHGLBP
· · · · · ·			. (%)	(mmol/L)	(mmoi/L)	(mmol/L)	. (%)
			· · · · ·	· · · · · · · · · · · · · · · · · · ·			
1	3	1	98	[~] 1.53	3.39	1.87	122
1	3	2	77	1.98	2.40	0.42	21
1	3	3	42	1.89	1.07	-0.82	-43
1	3	4	34	1.29	1.87	0.58	45
2	2	_1	53	1.60	1.91	0.30	19
2	2	2	96	2.30	1.39	-0.91	-40
2	2	3	35	2.90	1.45	-1.45	-50
2	2 _	4	36	1.49	1.27	-0.21	-14
3	1	1	63	0.97	2.08	1.11	114
3	1	2.	82	2.21	1.46	-0.74	-34
3	1	3	36	2.97	1.30	-1.67	-56
3	1	4	29	1.55	1.39	-0.16	-11
4	1	1	135	1.73	1.77	0.04	2
4	1	2	93	2.26	1.59	-0.67	-30
4	1	3	55	1.80	1.87	0.06	3
4	1	4	49	1.29	1.54	0.25	20 _
5	-2	1	94	1.65	1.73	0.08	5
5	2	2	39	1.63	1.39	-0.24	-15
5	2	3	62	1.51	1.43	-0.09	-6,
5	2	4	49	1.67	1.34	-0.33	-20
6	2	1	45	1.14	1.68	0.54	48
6	2	2	36	1.43	1.48	0.05	4
6	2	3	. 34	2.49	3.07	0.58	23
6	2	4	31	1.73	1.69	-0.04	-2
7	3	1	29	1.88	3.52	1.64	87 .
7	3	2	39	1.80	2.20	0.39	22
7	· 3	3	26	1.53	1.79	0.27	17
7	3	4	32	2.54	2.23	-0.30	-12
8	2	1	41	1.56	4.09	2.52	161
8	2	2	57	2.80	3.02	0.21	8
8	2	3	47	1.87	3.19	1.33	71
8	2	4	36	1.48	2.33	0.86	58

Raw Data

Subject	Event	Meet	Post-race LA	ACHGLPP	PCHGLPP	Performance	Avg.Vel.
			(mmol/L)	(mmol/L)	(%)	(secs)	(m/sec)
					-		
1	3	1	12.81	9.41	277	250	6
1	3	2	14.27	11.88	495	246.4	6.088
1	3	3	11.99	10.92	1018	247.9	6.051
1	3	4	15.14	13.28	711	249.9	6.002
2	2 .	1	13.87	11.96	628	125.89	6.355
2	2	2	14.07	12.68	914	125.8	6.359
2	2	3	14.35	12.90	889	123.24	6.491
2,	2 .	4	12.34	11.07	869	128.54	6.224
3	1	1	14.94	12.86	618	52.27	7.653
3	1	2	13.97	12.51	855	51.91	7.706
3	1	3	13.16	11.86	913	51.62	7.749
3	1	4	12.87	11.48	828	52.3	7.648
4	1	1	16.82	15.06	852	50.27	7.957
4	1	2	16.30	14.71	926	51.03	7.839
4,	1	3	15.59	13.72	735	50.05	7.992
4	1	4	14.83	13.29	863	50.45	7.929
5	2	<u> </u>	13.06	11.33	656	125.69	6.365
5	2	2	11.28	9.89	713	129.27	6.189
5	2	3	15.41	13.98	981	123.18	6.495
5	2	4	13.82	12.48	933	128.92	6.205
6	2	1	14.73	13.05	778	125.55	6.372
6	2	2	13.06	11.59	785	125.7	6.364
6	2	3	14.44	11.37	371	122.51	6.53
6	2	4	13.68	11.99	709	125.69	6.365
7	3	1	14.46	10.94	311	260.92	5.749
7	3	2	13.29	11.09	505	261.2	5.743
7	3	3	12.80	11.00	614	265.02	5.66
7	3	4	12.01	9.78	438	271.23	5.53
8	2	1	15.84	11.75	287	123.52	6.477
8	2	2	10.88	7.86	261	131.64	6.077
8	2	3	15.51	12.32	386	120.74	6.626
8	2	4	13.48	11.14	477	126.9	6.304

Raw Data

Subject	Baseline T (°C)	Pre-race T (°C)
1	33.89	34.78
2	35.06	34.89
3	35.11	34.94
4	35.61	35.89
5	35.56	35.50
6	34.83	35.00
7	34.72	34.50
8	35.28	35.44

REFERENCES

Andersen, K. L., Bolstad, A., & Sand, S. (1960). The blood lactate during recovery from sprint runs. <u>Acta Physiologica Scandinavica</u>, 48, 231-237.

Andzel, W. D. (1982). One mile run performance as a function of prior exercise. Journal of Sports Medicine, 22, 80-84.

Billat, V. L. (1996). Use of blood lactate measurements for prediction of exercise performance and for control of training: Recommendations for long distance running. Sports Medicine, 22 (3), 157-175.

Bond, V., Adams, R. G., Tearney, R. J., Gresham, K., & Ruff, W. (1991). Effects of active and passive recovery on lactate removal and subsequent isokinetic muscle function. Journal of Sports Medicine and Physical Fitness, 31 (3), 357-361.

Bonen, A. (2000). Lactate transporters (MCT proteins) in heart and skeletal muscles. <u>Medicine and Science in Sports and Exercise</u>, 32 (4), 778-789.

Bonen, A. & Belcastro, A. N. (1976). Comparison of self-selected recovery methods on lactic acid removal rates. <u>Medicine and Science in Sports, 8</u> (3), 176-178.

Bonen, A., Campbell, C. J., Kirby, R. L., & Belcastro, A. N. (1979). A multiple regression model for blood lactate removal in man. <u>Pflugers Archives, 380</u> (3), 205-210.

Borg, G. A. U. (1982). Psychological bases of physical exertion. <u>Medicine and</u> Science in Sports and Exercise, 14, 377-381.

Brooks, G. A. (1988). Blood lactic acid: Sports "bad boy" turns good. <u>Gatorade</u> <u>Sports Science Exchange, 1</u> (2).

Brooks, G. A. (1991). Current concepts in lactate exchange. <u>Medicine and</u> <u>Science in Sports and Exercise</u>, 23 (8), 895-906. Brooks, G. A. (2000). Intra- and extra-cellular lactate shuttles. <u>Medicine and</u> <u>Science in Sports and Exercise, 32</u> (4), 790-799.

Buono, M. J., & Roby, F. B. (1982). Acid-base, metabolic and ventilatory responses to repeated bouts of exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology, 53 (2), 436-439.

Colt, E. W. D., Wardlaw, S. L., & Frantz, A. G. (1981). The effect of running on ¹ plasma β-endorphin. <u>Life Sciences, 28,</u> 1637-1640.

Dassonville, J., Beillot, J., Lessard, Y., Jan, J., André, A. M., Le Pourcelet, C., Rochcongar, P., & Carré, F. (1998). Blood lactate concentrations during exercise: Effect of sampling site and exercise mode. <u>Journal of Sports Medicine and Physical Fitness, 38</u> (1), 39-46.

De Bruyn-Prevost, P. (1980). The effects of various warming up intensities and durations upon some physiological variable during an exercise corresponding to the wc₁₇₀. European Journal of Applied Physiology and Occupational Physiology, 43, 93-100.

De Bruyn-Prevost, P., & Lefebvre, F. (1980). The effects of various warming up intensities and durations during a short maximal anaerobic exercise. <u>European Journal of Applied Physiology and Occupational Physiology, 43,</u> 101-107.

Delp, M. D. & Laughlin, M. H. (1998). Regulation of skeletal muscle perfusion during exercise. <u>Acta Physiologica Scandinavica</u>, 162, 411-419.

De Vries, H. A. (1959). Effects of various warm-up procedures on 100-yard times of competitive swimmers. <u>Research Quarterly, 30</u> (1), 11-20.

Donovan, C. M. & Pagliassotti, M. J. (2000). Quantitative assessment of pathways ot lactate disposal in skeletal muscle fiber types. <u>Medicine and Science in</u> Sports and Exercise, 32 (4), 772-777.

Feliu, J., Ventura, J. L., Segura, R., Rodas, G., Riera, J., Estruch, A., Zamora, A., & Capdevila, L. (1999). Differences between lactate concentration of samples from ear lobe and the finger tip. Journal of Physiology and Biochemistry, 55 (4), 333-339.

Fox, E. L., Bowers, R. W., & Foss, M. L. (1989). <u>The physiological basis of</u> physical education and athletics (4th ed.). Dubuque, IA: Wm. C. Brown.

Fujitsuka, N., Yamamoto, T., Ohkuwa, T., & Miyamura, M. (1982). Peak blood lactate after short periods of maximal treadmill running. <u>European Journal of Applied</u> <u>Physiology and Occupational Physiology, 48,</u> 289-296.

Genovely, H., & Stamford, B. A. (1982). Effects of prolonged warm-up exercise above and below anaerobic threshold on maximal performance. <u>European Journal of</u> Applied Physiology and Occupational Physiology, 48, 323-330.

Gladden, B. L. (2000). Muscle as a consumer of lactate. <u>Medicine and Science in</u> Sports and Exercise, <u>32</u> (4), 764-771.

Grassi, B., Quaresima, V., Marconi, C., Ferrari, M., & Cerretelli, P. (1999). Blood lactate accumulation and muscle deoxygenation during incremental exercise. Journal of Applied Physiology, 87 (1), 348-355.

Grodjinovsky, A., & Magel, J. R. (1970). Effect of warm-up on running performance. <u>Research Quarterly, 41</u> (1), 116-119.

Gutin, B., Stewart, K., Lewis, S., & Kruper, J. (1976). Oxygen consumption in the first stages of strenuous work as a function of prior exercise. <u>Journal of Sports</u> <u>Medicine, 16,</u> 60-65.

Hautier, C. A., Wouassi, D., Arsac, L. M., Bitanga, E., Thiriet, P., & Lacour, J. R. (1994). Relationships between postcompetition blood lactate concentration and average running velocity over 100-m and 200-m races. <u>European Journal of Applied Physiology</u> and Occupational Physiology, 68, 508-513.

Holum, J. R. (1994). <u>Fundamentals of general, organic, and biological chemistry</u> (5th ed.). New York: John Wiley & Sons.

Houmard, J. A., Johns, R. A., Smith, L. L., Wells, J. M., Kobe, R. W., & McGoogan, S. A. (1991). The effect of warm-up on responses to intense exercise. International Journal of Sports Medicine, 12 (5), 480-483.

Hultman, E., & Sahlin, K. (1980). Acid-base balance during exercise. In R. S. Hutton & D. I. Miller (Eds.), <u>Exercise and Sport Sciences Reviews</u> (Vol. 8). USA: Franklin Institute.

Ingjer, F., & Strømme, S. B. (1979). Effects of active, passive or no warm-up on the physiological response to heavy exercise. <u>European Journal of Applied Physiology</u> <u>and Occupational Physiology</u>, 40, 273-282.

Jacobs, I. (1986). Blood lactate implications for training and sports performance. Sports Medicine, 3, 10-25.

Jacobs, I., Tesch, P. A., Bar-Or, O., Karlsson, J., & Dotan, R. (1983). Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. Journal of Applied Physiology, 55 (2), 365-367.

Karlsson, J., Bonde-Petersen, F., Henriksson, J., & Knuttgen, H. G. (1975). Effects of previous exercise with arms or legs on metabolism and performance in exhaustive exercise. Journal of Applied Physiology, 38 (5), 763-767.

Katz, A., & Sahlin, K. (1988). Regulation of lactic acid production during exercise. Journal of Applied Physiology, 65 (2), 509-518.

Klausen, K., Knuttgen, H. G., & Forster, H. V. (1972). Effect of pre-existing high blood lactate concentration on maximal exercise performance. <u>Scandinavian Journal of</u> <u>Clinical Laboratory Investigation, 30,</u> 415-419.

Lacour, J. R., Bouvat, E., & Barthelemy, J. C. (1990). Post-competition blood lactate concentrations as indicators of anaerobic energy expenditure during 400-m and 800-m races. <u>European Journal of Applied Physiology and Occupational Physiology, 61,</u> 172-176.

Lehninger, A. L., Nelson, D. L., & Cox, M. M. (1993). <u>Principles*of biochemistry</u> (2nd ed.). New York: Worth Publishers.

Martin, B. J., Robinson, S., Wiegman, D. L., & Aulick, L. H. (1975). Effect of warm-up on metabolic responses to strenuous exercise. <u>Medicine and Science in Sports</u> and Exercise, 7 (2), 146-149.

Mazzeo, R. S., Brooks, G. A., Shoeller, D. A., & Budinger, T. F. (1986). Disposal of blood [1-13C] lactate in humans during rest and exercise. Journal of Applied Physiology, 60 (1), 232-241.

McArdle, W. D., Katch, F. I., & Katch, V. L. (1991). <u>Exercise physiology:</u> <u>Energy, nutrition, and human performance (3rd ed.)</u>. Philadelphia: Lea & Febiger. McMurray, R. G., Forsythe, W. A., Mar, M. H., & Hardy, C. J. (1987). Exercise intensity-related responses of β-endorphin and catecholamines. <u>Medicine and Science in Sports and Exercise, 19</u> (6), 570-574.

Mitchell, J. B., & Huston, J. S. (1993). The effect of high- and low-intensity warm-up on the physiological responses to a standardized swim and tethered swimming performance. Journal of Sports Sciences, 11, 159-165.

Pagliassotti, M. J. & Donovan, C. M. (1990a). Role of cell type in net lactate removal by skeletal muscle. <u>American Journal of Physiology, 258, E635-E642</u>.

Pagliassotti, M. J. & Donovan, C. M. (1990b). Glycogenolysis from lactate in rabbit skeletal muscle fiber types. <u>American Journal of Physiology, 258</u>, R903-R911.

Powers, S. K., & Howley, E. T. (1997). <u>Exercise physiology: Theory and</u> application to fitness and performance (3rd ed.). Dubuque, IA: Brown & Benchmark.

Rahkila, P., Hakala, E., Alén, M., Salminen, K., & Laatikainen, T. (1988). ßendorphin and corticotropin release is dependent on a threshold intensity of running exercise in male endurance athletes. <u>Life Sciences, 43</u>, 551-558.

Reilly, T. & Woodbridge, V. (1999). Effects of moderate dietary manipulations on swim performance and on blood lactate-swimming velocity curves. <u>International</u> <u>Journal of Sports Medicine, 20</u> (2), 93-97.

Robergs, R. A., Costill, D. L., Fink, W. J., Williams, C., Pascoe, D. D., Chwalbinska-Moneta, J., & Davis, J. A. (1990). Effects of warm-up on blood gases, lactate and acid-base status during sprint swimming. <u>International Journal of Sports</u> <u>Medicine</u>, 11 (4), 273-278. Robergs, R. A., Pascoe, D. D., Costill, D. L., Fink, W. J., Chwalbinska-Moneta, J., Davis, J. A., & Hickner, R. (1991). Effects of warm-up on muscle glycogenolysis during intense exercise. <u>Medicine and Science in Sports and Exercise, 23</u> (1), 37-43.

Sahlin, K. (1986). Muscle fatigue and lactic acid accumulation. <u>Acta</u> Physiologica Scandinavica, 128 (Suppl. 556), 83-91.

Saltin, B., Rådegran, G., Koskolou, M. D., & Roach, R. C. (1998). Skeletal muscle blood flow in humans and its regulation during exercise. <u>Acta Physiologica</u> <u>Scandinavica, 162, 421-436</u>.

Sawka, M. N., Knowlton, R. G., Miles, D. S., & Critz, J. B. (1979). Postcompetition blood lactate concentrations in collegiate swimmers. <u>European Journal of</u> <u>Applied Physiology and Occupational Physiology, 41,</u> 93-99.

Schwarz, L., & Kindermann, W. (1992). Changes in β-endorphin levels in response to aerobic and anaerobic exercise. <u>Sports Medicine</u>, 13 (1), 25-36.

Sforzo, G. A. (1988). Opioids and exercise: An update. Sports Medicine, 7, 109-124.

Sherwood, L. (1993). <u>Human physiology: From cells to systems</u> (2nd ed.). New York: West Publishing.

Simoneau, J. A. & Bouchard, C. (1989). Human variation in skeletal muscle fiber-type proportion and enzyme activities. <u>American Journal of Physiology, 257, E567-E572</u>.

Staron, R. S. (1997). Human skeletal muscle fiber types: delineation, development, and distribution. <u>Canadian Journal of Applied Physiology, 22</u> (4), 307-327.

Stewart, I. B., & Sleivert, G. G. (1998). The effect of warm-up intensity on range of motion and anaerobic performance. Journal of Orthopedics and Sports Physical <u>Therapy, 27</u> (2), 154-161.

Stringer, W., Wasserman, K, Casaburi, R., Pórszász, J., Maehara, D, & French, W. (1994). Lactic acidosis as a facilitator of oxyhemoglobin dissociation during exercise. Journal of Applied Physiology, 76 (4), 1462-1467.

Svendenhag, J. & Sjödin, B. (1984). Maximal and submaximal oxygen uptakes and blood lactate levels in elite male middle- and long-distance runners. <u>International</u> Journal of Sports Medicine, 5, 255-261.

Taoutaou, Z., Granier, P., Mercier, B., Mercier, J., Ahmaidi, S., & Prefaut, C. (1996). Lactate kinetics during passive and partially active recovery in endurance and sprint athletes. <u>European Journal of Applied Physiology and Occupational Physiology</u>, <u>73</u> (5), 465-470.

Taylor, D. V., Boyajian, J. G., James, N., Woods, D., Chicz-Demet, A., Wilson, A. F., & Sandman, C. (1994). Journal of Applied Physiology, 77 (4), 1913-1918.

Wasserman, D. H., Connolly, C. C., & Pagliassotti, M. J. (1991). Regulation of hepatic lactate balance during exercise. <u>Medicine and Science in Sports and Exercise, 23</u> (8), 912-919.

Watt, E. W., & Hodgson, J. L. (1975). The effect of warm-up on total oxygen cost of a short treadmill run to exhaustion. <u>Ergonomics, 18</u> (4), 397-401.

Weltman, A., Stamford, B. A., & Fulco, C. (1979). Recovery from maximal effort exercise: lactate disappearance and subsequent performance. Journal of Applied Physiology, 47 (4), 677-682.

Yoshida, T. (1984). Effect of dietary modifications on lactate threshold and onset of blood lactate accumulation during incremental exercise. <u>European Journal of Applied</u> <u>Physiology and Occupational Physiology, 53</u> (3), 200-205.

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