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The effect of prior localized muscle fatigue on lactate production during submaximal exercise

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during submaximal exercise**

Morrison-Gibson, Molly A., M.S.

University of Nevada, Las Vegas, 1994

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The Effect of Prior Localized Muscle Fatigue on Lactate
Production during Submaximal Exercise

by

Molly A. Morrison-Gibson

A thesis submitted in partial fulfillment of the
requirements for the degree of

Master of Science

in

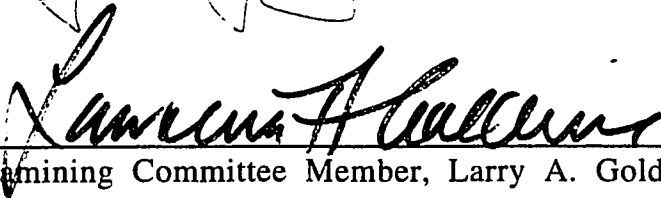
Exercise Physiology

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May 1994

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ABSTRACT

Muscle fatigue is a complex phenomena that involves both metabolic and neural factors. The present study examined the prolonged effects of localized muscle fatigue on lactate production during exercise. Five subjects, 3 females and 2 males, ages 25-29 ($x=27 \pm 1$) participated in the study. Subjects completed a max VO_2 test and two testing sessions, which consisted of cycling for 20 minutes at 80%-85% max VO_2 , once with prior fatigue and once without. Two-way repeated measures ANOVA showed gross lactate production was 20% higher in the non-fatigue trial than in the fatigue trial ($9.3 \pm .03$ vs. $7.6 \pm .04$, $p = .02$). Net lactate production was 34% higher in the non-fatigued trial than in the fatigue trial ($7.8 \pm .4$ vs. $5.2 \pm .4$, $p = .006$). RQ was higher in the fatigue trial than in the non-fatigue trial ($.97 \pm .03$ vs. $.89 \pm .03$, $p = .04$). In conclusion, lactate production is less in a fatigued state than a non-fatigue state. This change can be attributed to an alteration in muscle fiber recruitment patterns.

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CHAPTER 1

INTRODUCTION

Muscle fatigue, which is defined as the inability to maintain a given level of force production, has a negative effect on performance and physical work capacity. Although the exact mechanisms behind muscle fatigue are still unknown, both electrical and metabolic factors have been proposed. Muscle fatigue can occur at a number of steps between the brain and the actin-myosin crossbridges. Disruptions at any of these steps can impair the muscles ability to contract. There are two general types of fatigue, central and peripheral. Central fatigue can result from lack of motivation, impaired CNS transmission, or impaired motor neuron recruitment (Gibson & Edwards, 1985). Peripheral fatigue occurs at or distal to the neuromuscular junction. (Sahlin, 1992). There are two types of peripheral fatigue, high frequency fatigue, which is loss of force at high frequencies of neural stimulation and low frequency fatigue, which is a loss of force at low stimulation frequencies. The mechanisms behind high frequency fatigue are suggested to be impairment of propagation of an action potential, while low frequency fatigue is caused by an impairment of the excitation/contraction coupling (Gibson & Edwards, 1985). This study will concentrate on the effects of peripheral fatigue on

performance and will assume that central fatigue will not affect the results.

It has been established that metabolite changes occurring as a result of high intensity muscle contraction, such as ATP depletion and accumulation of H^+ , can inhibit the function of the contractile machinery. Changes in the levels of these metabolites immediately affect the muscle's ability to continue to perform a task at a given level of force. However, the metabolites are removed from the blood within minutes following a high intensity, short bout of exercise (Miller et al, 1987). The rapid removal of hindering metabolites allows a subject to perform the same task at a near maximal level within minutes after experiencing fatigue. Studies have been done on the immediate effects of fatigue on performance in both short bouts of high intensity exercise and long bouts of low intensity exercise but little research has been done on the interaction between the two, that is, the persistent effects of high intensity fatigue on the ability to perform an aerobic task have not been examined. This study is designed to determine the cross-over effects from fatigue during strength activities on lactate production during endurance exercise. The following assumptions will be made during the study: 1) blood lactate level is an indicator of glycolytic rate and changes occurring in energy producing pathways 2) the glycolytic rate will be the same in both a fatigued state and a non-fatigued state, and 3) starting levels of glycogen will be the same in both a fatigued and non-fatigued state. Therefore, the purpose of this study is to examine the effect of prior localized muscle fatigue on lactate production during high intensity aerobic exercise performance.

CHAPTER 2

LITERATURE REVIEW

The etiology of muscle fatigue has been the focus of research for many years. Although the exact cause of muscle fatigue is still unknown there are many factors that have been accepted as contributors to fatigue.

Muscle fatigue is defined as the inability to maintain a given level of force during sustained or repeated contractions (Gibson and Edwards, 1985). Fatigue involves processes related either to peripheral mechanisms or to central nervous command. Central fatigue is attributed to an impairment in the central nervous system (CNS). Causes of central fatigue include motivation or lack thereof, a reduction in the number of recruited motor neurons, and impaired transmission of neural signals down the spinal cord (Gibson and Edwards, 1985). Peripheral fatigue is defined as fatigue which occurs at or distal to the neuromuscular junction (Sahlin,1992). Merton's (1954) classic experiment was first to show a distinction between central and peripheral fatigue. He found no difference in muscle tension with a maximum voluntary contraction and a maximum contraction stimulated electrically of isolated thumb flexors. Electrical stimulation produced the same EMG activity by the muscle, but force production was decreased. Therefore, there was a separation of neural signals and force production in a fatigued state.

In addition, when a muscle was fatigued voluntarily, electrical stimulation could not restore muscle tension providing evidence of fatigue sites other than the CNS. Peripheral fatigue has also been differentiated into high frequency and low frequency fatigue. High frequency fatigue is characterized by a loss of force at high neural stimulation frequencies (>50 Hz). Gibson and Edwards (1985) suggest impaired neuromuscular transmission and/or propagation of muscle action potential at the neuromuscular junction as the mechanisms behind high frequency fatigue. Low frequency fatigue is a loss of force at low neural stimulation frequencies due to impaired excitation/contraction coupling (Gibson and Edwards, 1985). Force production is affected differently by high frequency and low frequency stimulation. Metzger (1987) found a difference in recovery times between fatigue induced by high and low stimulation frequencies. In this study, the rat diaphragm was electrically stimulated to fatigue at 5-Hz(low) and 75-Hz (high) frequencies. Metzger (1987) found that force production was restored following the low frequency stimulation in 1 min., while the fatigue induced at 75-Hz was still evident in force production until 10-15 minutes of recovery. It should be noted that volitional muscle contraction is neurally stimulated in the range of 5-30Hz.

Role of Calcium

Several steps are involved in the transmission of a signal from the brain to a muscle initiating muscle contraction (Figure 1) (Gibson and Edwards, 1985). A disruption in any of these steps will result in an impaired ability for a muscle to contract. The excitation-

contraction coupling process involves a series of chemical reactions and pathways that ultimately result in the splitting of adenosine triphosphate (ATP), cross-bridge cycling, and muscle contraction. The first step in excitation-contraction coupling is the propagation of an action potential over the muscle sarcolemma and down the t-tubules of the muscle membrane. This charge movement causes the release of calcium (Ca^{++}) from the sarcoplasmic reticulum (SR). Ca^{++} binds to troponin C which causes a conformational change in the troponin-tropomyosin complex. Exposure of the binding sites on actin for myosin allows the contractile proteins actin and myosin to couple generating muscular contraction and force (Vollestad and Sejersted, 1988).

Ca^{++} is the primary ion in muscle contraction. Under normal conditions, saturating amounts of Ca^{++} are released from the sarcoplasmic reticulum and muscle contraction occurs. In a fatigued state, less Ca^{++} is available to bind all troponin sites (Fitts and Metzger, 1988). Therefore alterations in Ca^{++} storage and release may be related to muscle fatigue. Blinks et al (1978) showed there was a reduction in the amount of Ca^{++} released from the SR with sustained contractile activity. The slower release rate of Ca^{++} from the SR attenuates the excitation/contraction coupling rate, resulting in an increased twitch duration and a reduced rate of tension development (Fitts et al, 1982; Metzger and Fitts, 1987). Extracellular Ca^{++} can also affect Ca^{++} release from the SR. Low extracellular Ca^{++} causes a reduction in the t-tubular charge movement, hence a diminished signal for Ca^{++} release from the SR (Pizarro et al, 1987). Ca^{++} release from the SR may also be affected

by an altered concentration gradient between the SR and intracellular fluid (Fitts, 1988). Edwards et al (1977) suggested that the amount of Ca^{++} stored in the SR decreases during exercise because of reduced reuptake of intracellular Ca^{++} . This would ultimately result in reduced Ca^{++} release and may be due to slower Ca^{++} pump activity. It has also been suggested that Ca^{++} binding proteins are involved in fatigue. Increased binding of the binding protein parvalbumin has been found during contractile activity (Fitts and Metzger, 1988). Gibson and Edwards (1985) proposed that troponin may lose its sensitivity to Ca^{++} thus resulting in muscle fatigue in the same manner that reduced levels of intracellular Ca^{++} affect performance.

Role of metabolites

Metabolic agents have also been shown to contribute to muscle fatigue. At a high intensity, (>90% VO_{2max}), exercise exceeds maximal aerobic power and requires a high level of anaerobic work (Fitts and Metzger, 1988). Sahlin (1992) found significant increases of metabolites of muscle contraction at intensities >90% VO_{2max} . Because high intensity resistance exercise involves the anaerobic energy producing pathway, there are several changes in the concentration of specific metabolites. Adenosine triphosphate (ATP) and phosphocreatine (PC) levels decrease while lactate, the H^+ ion, adenosine diphosphate (ADP) and inorganic phosphate (Pi) levels all increase. Each of these metabolites has been nominated as a fatigue inducing agent (Vollestad and Sejersted, 1988). High intensity exercise produces high turnover rates of ATP, leaving relatively low

levels of ADP and Pi (Sahlin, 1992). At a certain point in high intensity exercise, ATP utilization cannot be equaled by ATP formation resulting in energy deficiency and reduced force production by the muscle (Sahlin, 1992). This decrease in ATP formation, although small, increases intracellular ADP and Pi which are potential fatiguing agents (Sahlin, 1992). Sahlin (1992) proposed that in a fatigued muscle, the rate of ADP rephosphorylation is slowed, causing an increase in contraction time and a decrease in contraction amplitude. Fitts and Metzger (1988) suggest Pi contribute to fatigue by directly inhibiting hydrolysis of ATP by ATPase. Low tissue levels of ATP have also been suggested as a fatiguing agent (Fitts and Metzger, 1987). Karlsson and Saltin (1970) found that ATP was depleted following 2 minutes of exercise in a high, moderate and low intensity trial, however, fatigue only occurred with the highest workload demonstrating that low levels of tissue ATP do not cause muscle fatigue. Phosphocreatine (PC) stores also decrease with muscle activity (Fitts and Holloszy, 1976). PC participates in the process known as the PC-ATP shuttle which is responsible for moving ATP from the mitochondria to the cross-bridges (Simon et al, 1985). There is a possibility that low levels of PC slow the rate of rephosphorylation of ADP to ATP by disrupting the shuttle system, leading to low levels of ATP at the cross-bridges and producing muscle fatigue (Fitts and Metzger, 1988). Dawson (1976) and Fitts and Metzger (1976) show that although muscle PC declines with continued muscle contraction, the time course of the decline in tension is different from the time course of the depletion of muscle PC stores, making a relationship unlikely. H⁺ ion, which

results from lactic acid dissociating into lactate and H^+ , can affect muscle contraction in several ways. H^+ can: 1) directly inhibit actomyosin ATPase and ATP hydrolysis 2) inhibit PFK and hence the rate of glycolysis 3) competitively bind to troponin C inhibiting actin-myosin crossbridge interaction and 4) inhibit SR-ATPase which reduces Ca^{++} reuptake and ultimately Ca^{++} release (Nakamura and Schwartz, 1971). Fabiato and Fabiato (1978) using skinned muscle fibers showed that acidosis decreases the force output of skeletal muscle. High levels of H^+ also increased the threshold of Ca^{++} needed for contraction as well as causing higher amounts of intracellular Ca^{++} to be needed in order to produce a given tension (Fabiato and Fabiato, 1978). Sahlin suggests that acidosis impairs the rephosphorylation of ADP thus causing a decreased capacity to generate ATP. Despite a relationship between fatigue and acidosis, fatigue can not solely be linked to high levels of H^+ . Subjects with phosphorylase deficiency are unable to produce lactic acid, yet still experience similar patterns of muscle fatigue as normal subjects (Mills and Edwards, 1984). High levels of extracellular potassium ions (K^+) have also been linked to muscle fatigue. High frequency stimulation of the muscle causes an increase of extracellular K^+ (Vollestad and Sejersted, 1988). Metzger and Fitts (1987) found that the accumulation of extracellular K^+ around the sarcolemma causes a disruption in sarcolemma action potentials, but these disruptions were not enough to account for the large decreases in muscle force output. Vollestad and Sejersted (1988) found that increased K^+ levels were removed with adequate blood flow and did not contribute to muscle fatigue.

Fiber type recruitment patterns

Skeletal muscle is composed of essentially three fiber types, distinguishable by characteristics such as oxidative capacity, glycolytic capacity, ATPase activity and contraction times (Armstrong, 1988). Fibers with high oxidative capacity, low ATPase activity, slow contraction times and relatively low force production are considered slow twitch or Type I muscle fibers (Armstrong, 1988). Fast twitch or Type II muscle fibers have either a high or low oxidative capacity characterized by high ATPase activity, fast contraction times and high force production (Green, 1986). Type II muscle fibers are divided into fast oxidative/glycolytic or Type IIa and fast glycolytic or Type IIb. The average person possesses about 47-53% slow twitch fibers and individual muscles are composed with fibers that reflect its function, such as the back muscles which are slow twitch in order to maintain posture (Holloszy, 1988). An endurance runner may possess up to 75% slow twitch fibers in their leg muscles and a sprinter's leg muscles may be composed of 75% fast twitch fibers (Tesch et al, 1984).

Muscle fiber recruitment patterns depend on the specific activity and the intensity of exercise involved. Involvement of specific fibers in activity is based on the metabolic demands of the exercise and is reflected in the glycogen loss patterns among fibers. For light exercise (30 to 40% VO_{2max}), which requires low force production, Type I muscle fibers are recruited. Moderate intensity exercise (60-70% VO_{2max}) employs mainly Type I fibers and Type

Ia fibers, while exercise intensities above 80% VO_{2max} requires the involvement of all three fiber types (Saltin and Gollnick, 1988). From glycogen loss patterns, it can be assumed that fast twitch muscle fibers are less resistant to fatigue than slow twitch muscle fibers at high intensity exercise (Green, 1986). During high intensity or maximal exercise, Type IIb fibers will fatigue first causing a shift to Type Ia and Type I fibers to maintain force output by the muscle (Armstrong, 1988). Naeije and Zorne (1982) demonstrated that as a muscle becomes fatigued, there is a decrease in firing frequency by the motor units involved illustrating a shift from Type II muscle fibers to Type I fibers.

Recovery from high intensity exercise

Sahlin and Ren (1989) examined recovery of the contractile function of muscle in relation to metabolite changes using the knee extension exercise. Following fatiguing exercise, recovery of generation capacity (MVC) and endurance (time to fatigue) was measured. They found that maximum force production capability of the muscle is restored more rapidly than endurance following a fatiguing isometric exercise. The high H^+ concentration present did not affect force production but may possibly have affected endurance capacity by impairing the rephosphorylation of ADP (Stalin and Ren, 1989). A study by Stull and Clarke (1971) examined the patterns of recovery following isotonic and isometric hand gripping exercises. One trial consisted of maintaining a maximal static contraction, while trial two involved rhythmic maximal contractions. Their results showed recovery of force

production to be more rapid following isotonic exercises compared with isometric exercise. However, force production was recovered within 4 minutes for both isotonic and isometric conditions. Miller et al (1987) suggest three phases of recovery from fatiguing exercise. The first phase of recovery involves restoration of muscle membrane potential and impulse propagation which occurs within 6 minutes following the fatiguing exercise. The second phase is recovery of the MVC which is complete within 20 minutes and correlates with restoration of pH and high energy phosphates (ATP, PC). The final phase of recovery is a restoration of neuromuscular efficiency. This phase does not occur for over 60 minutes possibly indicating an impairment of the excitation-contraction coupling process (Miller et al, 1987).

Role of glycogen

Depletion of glycogen stores has been shown to be a contributing factor in muscle fatigue. Although the exact mechanisms regulating the breakdown of glycogen, or glycogenolysis are unresolved, muscle phosphorylase, Ca^{++} , epinephrine and cyclic AMP mediate the breakdown of glycogen. Phosphorylase exists in two forms, *a* and *b*. Phosphorylase *a* is the active form, stimulating glycogenolysis, and phosphorylase *b* is the inactive form (Conlee et al, 1979). Glycogenolysis is activated when a muscle contracts and Ca^{++} released from the sarcoplasmic reticulum increases phosphorylase *a* activity by activating phosphorylase *b* kinase (Richter et al, 1982). Epinephrine also stimulates glycogenolysis by increasing cyclic AMP thus initiating the reaction converting

phosphorylase *b* to *a* (Richter et al, 1982). The major cause of muscle fatigue during endurance exercise at intensities of 65-85% $\text{VO}_{2\text{max}}$ is glycogen depletion (Fitts, 1988; Hultman and Spriet, 1988). This is evident by the fact that adequate levels of free fatty acids and blood glucose are present following exhaustive long term exercise (Fitts, 1988). However, there may be other factors contributing to fatigue from moderate intensity exercise such as a disruption in the SR membrane. Fitts et al, (1982) examined the functional capacity of isolated SR membranes, which are involved in the regulation of intracellular Ca^{++} . Following an exhaustive swim by rats, there was a significant decrease in the amount of SR protein isolated in the slow twitch soleus muscle, and a decrease in Ca^{++} uptake, without a concomitant decrease in the SR ATPase activity (Fitts et al, 1982). These results suggest damage to the SR membrane in addition to glycogen depletion as contributors to muscle fatigue during long bouts of low to moderate exercise.

Glycogen depletion is not considered a contributing factor of fatigue in high intensity, short bouts of exercise. Tesch et al (1986) concluded from a study on metabolic changes during intense and prolonged, heavy resistance exercise that glycogen depletion did not contribute to muscle fatigue. At exhaustion, muscle biopsies revealed relatively high levels of glycogen which were not significantly different from resting levels. This can be attributed to the fact that high intensity exercise relies more on ATP-PC stores for energy. In addition, recovery of force production is rapid following short bouts of high intensity fatiguing exercise and because glycogen resynthesis is slow following exercise that depletes glycogen stores,

glycogen depletion is not the cause of fatigue in high intensity exercise (Piehl, 1974). Research by Roberts et al (1991) supports the finding that heavy resistance exercise does not significantly deplete glycogen stores.

Role of lactate

Lactate is an end-product of glycolysis. The amount of lactic acid formed is a direct result of the levels of pyruvate, [NAD]/[NADH] in the cytoplasm and the enzymatic activity of lactate dehydrogenase (LDH). When pyruvate production is greater than conversion to acetyl CoA in the TCA cycle, there is a net increase of lactate. Low levels of lactate are present in the blood at rest, measuring 1-2mM (Gollnick, 1986). Lactate levels in the blood during exercise depend on both the intensity and type of exercise. Exercise lactate levels range from 3-5mM in prolonged moderate intensity exercise to as high as 20mM during maximal exercise or with repeated bouts of dynamic exercise (Hermansen and Stensvold, 1972). In low intensity exercise (40-60% VO_{2max}), lactate appearance in the blood reaches a steady state level and concentration does not significantly exceed resting levels (Gollnick et al, 1986). As exercise intensity increases, there is a similar rise in lactate production. At exercise intensities of 60-80% VO_{2max} , lactate begins to accumulate in the blood depending on the fitness level of the individual (Armstrong, 1988). The point at which lactate begins to accumulate in the blood is termed "lactate threshold or anaerobic threshold." Lactate concentration in the blood can be expressed as the difference between lactate production and lactate clearance

(Wasserman et al, 1986) If lactate formation exceeds lactate clearance, there will be a net increase in blood lactate.

There is controversy surrounding the mechanisms that cause the lactate threshold. Wasserman et al (1986) proposed two mechanisms by which blood lactate increases: 1) glycolytic rate increases so rapidly that pyruvate is not utilized fast enough by the mitochondria and builds up in the cytoplasm causing a relative increase in lactate concentration and 2) the mitochondrial membrane shuttle which is responsible for reoxidizing NAD becomes rate limited. Holloszy (1988) concluded that low muscle oxygen and recruitment of fast twitch or Type II muscle fibers also contribute to the accumulation of lactate. Type II muscle fibers have a high glycolytic capacity and their recruitment during high intensity exercise may produce higher levels of lactate (Armstrong, 1988). A restriction of oxygen available to the contraction muscle results in an increase of lactate production because oxidative phosphorylation will be depressed (Stainsby, 1986). Skinner and McLellan (1980) proposed that the LDH found in Type II fibers has a higher affinity for attaching to pyruvate, increasing lactate formation.

The major fate of lactate is direct oxidation (Brooks, 1986). At rest, approximately 50% of lactate is oxidized, while during sustained exercise at 50% of VO_2 max, 90% of the lactate is removed by oxidation. Lactate is also taken up by the liver for gluconeogenesis. There is evidence that the exercising muscle itself can take up lactate, but this depends on the training state of the muscle and glycogen concentration (Gollnick et al, 1986).

Performance is adversely affected by muscle fatigue. The reduced capacity to produce force inhibits performance on both high intensity, short bouts of exercise and low intensity, prolonged bouts of exercise. Although the mechanisms of fatigue may change with different intensities and durations of exercise, research so far has failed to examine the interaction between the fatigue resulting from resistance exercise and subsequent performance during a prolonged submaximal exercise bout. Pendergast et al (1983) examined the effect of high levels of lactate on both aerobic and anaerobic capacity. High levels of lactate were induced by running on a treadmill, after which subjects performed a max VO₂ test and a submaximal exercise trial. The results indicated that max VO₂ and the efficiency of aerobic work were not affected by preceding high levels of lactate, however, the endurance capacity for both the aerobic work and anaerobic work trials was reduced (Pendergast et al, 1983). These results agree with research showing that high levels of lactate negatively affect force output by the muscle and endurance during exercise bouts (Nakamura & Schwartz, 1971; Fabiato & Fabiato, 1978). However, there is a need for research examining the persistent affects of preceding anaerobic, high intensity resistance exercise on submaximal aerobic exercise when acidosis is no longer present in the blood, and other metabolic agents that contribute to fatigue have been removed from the muscle. It will be of interest to examine the residual effects of fatigue from resistance exercise on the physiological response of lactate production during a subsequent prolonged moderate to high intensity bout of exercise.

CHAPTER 3

METHODS

Five subjects, 3 females and 2 males, ages 25-29 ($X = 27$, $S.E. = 1$) volunteered to participate in the study, after giving informed consent (Table 1). Height and weight were recorded for each subject. Harpenden calipers were used to measure four skinfolds (abdomen, ilium, thigh and tricep) and percent body fat was calculated using the Jackson and Pollack sum of four formula (Golding et al, 1989).

Table 1: Subject characteristics

SUBJECT	SEX	AGE	HEIGHT (cm)	WEIGHT (kg)	BODYFAT %
1	M	29	184	77	14.7
2	M	25	176	65	9.1
3	F	27	160	60	18.4
4	F	28	167	100	24.5
5	F	28	173	74	19.6
MEAN		27	172	75	17.3
S.E.		1	37	21	2.9

Maximal oxygen consumption (VO_{2max}) was determined in each subject using a Schwinn BioDyne cycle ergometer. Subjects

completed two testing sessions, each on a separate day. Each session consisted of cycling for 20 minutes at an intensity of 80% $\text{VO}_{2\text{max}}$, once with localized muscle fatigue of the quadriceps and once without. Lactate was measured every 5 minutes during exercise and at 5 and 10 minutes during recovery. Heart rate and oxygen consumption (VO_2) were measured to monitor exercise intensity.

Max VO_2 test

The subjects were fitted with a Vantage heart rate monitor (Polar Inc., Stanford CT). To measure VO_2 , subjects were fitted with a headpiece that held a Hans Rudolph non-rebreathing valve and mouthpiece. The test was performed on a Schwinn BioDyne cycle ergometer and VO_2 was measured by a Vista Metabolic measurement system (Vacumetrics Inc., Ventura, CA.) using Turbofit software (version 2.2A). Each subject pedaled for approximately 2 minutes without resistance to allow for a short warm-up. The test protocol consisted of 1 minute stages with increasing workloads of 150 kp until volitional fatigue. VO_2 and heart rate were measured every 20 seconds.

Non-fatigue trial

Resting lactate levels were determined by taking a blood sample from the finger. A semi-automatic lancet was used to puncture the skin and blood was collected in a Microtainer Brand tube with EDTA (Becton Dickinson & Co., Rutherford, NJ.). The blood was then analyzed using a YSI Model 23L Lactate Analyzer (Yellow

Springs Instruments, Yellow Springs, OH.) Next, subjects were fitted with the heart rate monitor and a two-way, non-rebreathing valve to measure VO_2 . The workload during the 20 minute cycle exercise was predetermined to be between 80%-85% of VO_{2max} (Table 2). After steady state was reached, changes in workload were made if the subject was not in the range of 80%-90% of VO_{2max} . Blood samples were taken every 5 minutes during exercise and 5 and 10 minutes during recovery. Blood samples were stored on ice and analyzed for lactate concentration using a YSI model 23 lactate analyzer. Duplicate samples were analyzed and calibration was checked after every 4th sample.

Fatigue trial

Each subject's resting lactate level was measured using the same technique as the non-fatigue trial. The subject then performed an isometric maximum voluntary contraction (MVC) of the quadriceps muscle group. The MVC was done on a strength table in the sitting position with the knee at approximately a 110 degree angle. Both legs were done at the same time and the upper leg was stabilized during the contraction. The MVC was measured by a cable tensiometer attached to the middle of a padded bar. Each subject was told to perform a maximal contraction and hold it for 3 seconds. Three attempts were made and the highest value was recorded as the MVC. The subjects then performed isokinetic leg extensions at 70% of MVC on a Life Circuit machine until voluntary fatigue. At voluntary fatigue, the isometric MVC was immediately measured. If the fatigue MVC was 50%-60% of the original MVC, the subject was

considered to be fatigued (Table 3). If the fatigue MVC was higher than 60% of the original MVC, the subject was instructed to continue the leg extension exercise. Following the fatiguing exercise, active recovery was performed by cycling with no resistance on the cycle ergometer. When recovery blood lactate reached levels within 1 mM of resting blood lactate, the subject then performed the same cycling protocol as in the non-fatigue trial. Blood lactate was measured every 5 minutes during exercise and 5 and 10 minutes post-exercise and analyzed for lactate content.

Statistical analysis

Mean blood lactate levels between groups and within subjects were analyzed by two-way repeated measures analysis of variance (ANOVA). Mean respiratory quotient (RQ) values between groups were also analyzed by two-way repeated measures ANOVA. A dependent t-test was performed on individual means for net lactate production.

CHAPTER 4

RESULTS

Fatigued MVC was 57% of the non-fatigued MVC, demonstrating that the leg extension protocol successfully induced fatigue (Table 2). Subjects cycled at an average intensity of 82% $\text{VO}_{2\text{max}}$ in the non-fatigue trial and an average intensity of 83% $\text{VO}_{2\text{max}}$ in the fatigue trial, indicating that work levels were equal between exercise trials (Table 3). Gross lactate production was 22% higher in the non-fatigue trial than the fatigue trial (9.3 ± 0.6 vs. 7.6 ± 0.4 , $p < 0.05$) (, Table 4, Figure 2). Net lactate production was 50% higher in the non-fatigue trial than the fatigue trial (7.8 ± 0.4 vs 5.2 ± 0.4 , $p < 0.01$) (Table 5, Figure 3). Individual values for mean lactate production are shown in Figure 4. Mean respiratory quotient (RQ) was higher in the fatigue trial than in the non-fatigue trial ($.97 \pm .03$ vs. $.89 \pm .03$, $p < 0.05$) (Table 6, Figure 5).

CHAPTER 5

DISCUSSION

Muscle fatigue is a complex phenomenon which may result from alterations in the muscle itself or in neural input to the muscle. A generalized state of fatigue can be attributed to depletion of muscle glycogen stores or the presence of metabolites which impair the excitation/contraction coupling process (Vollestad and Sejersted, 1988). Glycogen repletion or metabolite removal would be expected to reverse the perception of fatigue or promote recovery. However, some athletes experience characteristics of chronic fatigue or overtraining, a condition which is reflected in a decrement in exercise performance. Although VO_{2max} was unchanged, Costill (1985) found that performance time on a standard task was increased and the task required a higher percentage of VO_{2max} when subjects were overtrained. Subjective feelings of heaviness in the legs and muscle soreness were also reported (Costill, 1985). In the present study, it was hypothesized that localized muscle fatigue, without depletion of muscle glycogen stores, could serve as a model of overtraining. To test this hypothesis, subjects first performed maximal leg extension exercises to fatigue, then exercised on a cycle ergometer at 80% VO_{2max} for 20 minutes. The underlying assumption was that an increase in relative intensity of the cycling

exercise induced by fatigue would be reflected in an increase in blood lactate levels.

Contrary to what was expected, blood lactate levels were significantly decreased during the 20 minute cycling session following a fatiguing bout of leg extension resistance exercise. The rate at which muscle utilizes glycogen is a function of the amount of glycogen available initially and the relative intensity of the workload (Richter and Galbo, 1986; Coyle et al, 1985). Tesch et al (1986) examined glycogen levels of the vastus lateralis following an exercise regimen consisting of 5 sets each of front squats, leg extensions, back squats, and leg presses to muscle failure. Results showed that glycogen levels were not significantly different from pre-exercise levels and did not seem to be a cause for impaired muscle function (Tesch et al, 1986; Roberts, 1991). Since the type of exercise performed to induce local fatigue was most likely not sufficient to reduce muscle glycogen levels, it can be assumed that glycogen levels were the same in both conditions prior to the cycling exercise (Piehl, 1974; Roberts et al, 1991). Thus, differences in lactate production would not be a result of a differential rate of glycolysis due to different starting levels. The fact that lactate levels were actually lower in the fatigued state argues against an increased dependence on anaerobic glycolysis for energy production in these conditions. An alternate explanation for the reduced rate of lactate formation is that there was a shift in the pattern of motor unit recruitment. Type IIB fibers which are highly glycolytic, are recruited for high intensity exercise ($>85\%VO_{2max}$). Increased lactate levels generally reflect their involvement in exercise (Armstrong, 1988; Holloszy, 1988). All

muscle fibers are recruited to maintain force production during short bouts of maximal exercise, such as the leg extension exercise employed in this study (Armstrong, 1988). Type IIb muscle fibers are less resistant to fatigue and are exhausted first in heavy exercise (Green, 1986; Burke et al, 1973). Therefore, the reduction in force output which occurs with resistance exercise is due in part to the withdrawal of the fatigued Type IIb fibers. When Type IIb muscle fibers can no longer contribute to force production, there must be a shift to Type I and IIa fibers to maintain force output at a given level (Naeije and Zorn, 1982). Type I, or slow twitch fibers are highly oxidative and are recruited for low intensities of exercise (<60% $\text{VO}_{2\text{max}}$). Type IIa fibers have both glycolytic and oxidative capacities and are recruited, along with Type I fibers, for moderate intensity exercise (60%-70% $\text{VO}_{2\text{max}}$) (Saltin and Gollnick, 1988). Because of the involvement of Type I and Type IIa fibers, low to moderate intensity exercise does not result in high lactate levels (Armstrong, 1988). The results of the present study therefore suggest that the resistance exercise selectively fatigued Type IIb fibers which are slower to recover than other fiber types. If Type IIb muscle fibers are unavailable for recruitment during the 20 minute, high intensity cycling, a higher percentage of Type I and IIa fibers are recruited to maintain force output and a shift in motor unit recruitment pattern occurs.

Recovery of muscular force production following high intensity fatiguing exercise, both dynamically and statically, has been found to be rapid (Sahlin and Ren, 1989; Stull and Clark, 1971). Miller et al (1987) proposed 3 phases of recovery following both static and

dynamic exercise of the adductor pollicis muscle in which the last phase involved regaining resting excitation-contraction coupling potential. Miller found the excitation-contraction coupling to be impaired up to 60 minutes post-exercise. In the present study, lactate levels returned to resting levels within 35 minutes following the fatiguing leg extension exercise in all subjects, suggesting metabolite clearance from the muscle and blood. However, neuromuscular efficiency may not have been restored by this time (Miller et al, 1987; Gibson and Edwards, 1985). The contractile function of the fatigued Type IIb fibers may have still been impaired, rendering them unavailable for recruitment during the subsequent cycling exercise. In the present study, subjects worked at the same absolute workload in both the fatigued and non-fatigued state, which therefore required the same force output. An impairment in Type IIb fiber activation would necessitate Type I and IIa fibers providing the energy for the cycling.

The results of this study also showed a significantly higher RQ in the fatigued state compared with the non-fatigued state, providing evidence for a shift in motor unit recruitment patterns. A lower blood lactate concentration at the same absolute workload, and a higher RQ suggests an increase in carbohydrate oxidation. Glycogen degradation, results in the formation of pyruvate, which is either oxidized in the TCA cycle or reduced to lactate (Saltin and Gollnick, 1988). The muscle fiber types involved in a particular exercise task play a major role in determining the fate of degraded carbohydrates. Type IIb muscle fibers have a much lower oxidative capacity than Type I and IIa fibers. Therefore involvement of Type I and IIa

fibers results in a greater degree of oxidative energy production to provide the same level of force output (Saltin and Gollnick, 1988). Hence, the recruitment of Type I and IIa fibers during the cycling exercise with prior fatigue, due to the slow recovery of the Type IIb fibers, is consistent with the lower lactate levels and higher RQ value found in this study.

CHAPTER 6

CONCLUSION

The purpose of this study was to examine the effect of prior localized leg fatigue on lactate production during submaximal cycling exercise. A significant decrease in blood lactate levels was found during a 20 minute cycling session at 80% VO_{2max} following localized leg fatigue. These results are suggestive of an alteration in muscle fiber recruitment patterns following a fatiguing bout of resistance exercise. The resistance exercise may have caused a prolonged impairment of the excitation/contraction coupling mechanism in Type IIb fibers, making them less available for recruitment during a subsequent aerobic exercise session. Type I and IIa fibers would then be responsible for force production during the ensuing cycling exercise. Specific physiological changes occur with a shift to recruitment of Type I and IIa fibers. These changes include an increased oxidation of carbohydrates which results in lower blood lactate levels, assuming the same rate of glycolysis, and a higher RQ in the prior fatigued state. Because of the apparent shift in fiber recruitment pattern to meet with the demands of the endurance task after the prior resistance exercise, it is not clear that this protocol is a suitable model of overtraining. More work will be required before this can be determined.

TABLE 2: MVC (lbs) prior to and following the resistance exercise.

SUBJECT	MVC	MVC- FATIGUED	% of MVC
1	198	123	62%
2	143	72	50%
3	98	52	53%
4	116	71	61%
5	124	76	61%
MEAN	136	79	57%
S.E.	61	12	3

TABLE 3: Oxygen consumption (VO₂) (ml/kg/min.).

SUBJECT	MAX VO ₂	FATIGUE VO ₂	NON- FATIGUE VO ₂
1	40	34	35
2	46	33	35
3	41	38	35
4	31	25	26
5	42	37	34
MEAN	40	33	33
S.E.	13	2	2

TABLE 4: ANOVA table for gross lactate production.

SOURCE	DF	SS	MS	F	P
Group	1	47.35	47.35	11.56	.0273
Time	5	61.92	12.38	8.80	.0002
Group*Time	5	4.34	.87	.72	.6150

TABLE 5: ANOVA table for net lactate production

SOURCE	DF	SS	MS	F	P
Group	1	103.75	103.75	26.75	.0066
Time	5	60.97	12.19	8.30	.0002
Group*Time	5	5.93	1.19	.90	.5092

TABLE 6: ANOVA table for RQ.

SOURCE	DF	SS	MS	F	P
Group	1	.062	.062	8.68	.0421
Time	3	.186	.062	104.70	.0001
Group*Time	3	.004	.001	1.46	.2757

Possible fatigue mechanisms

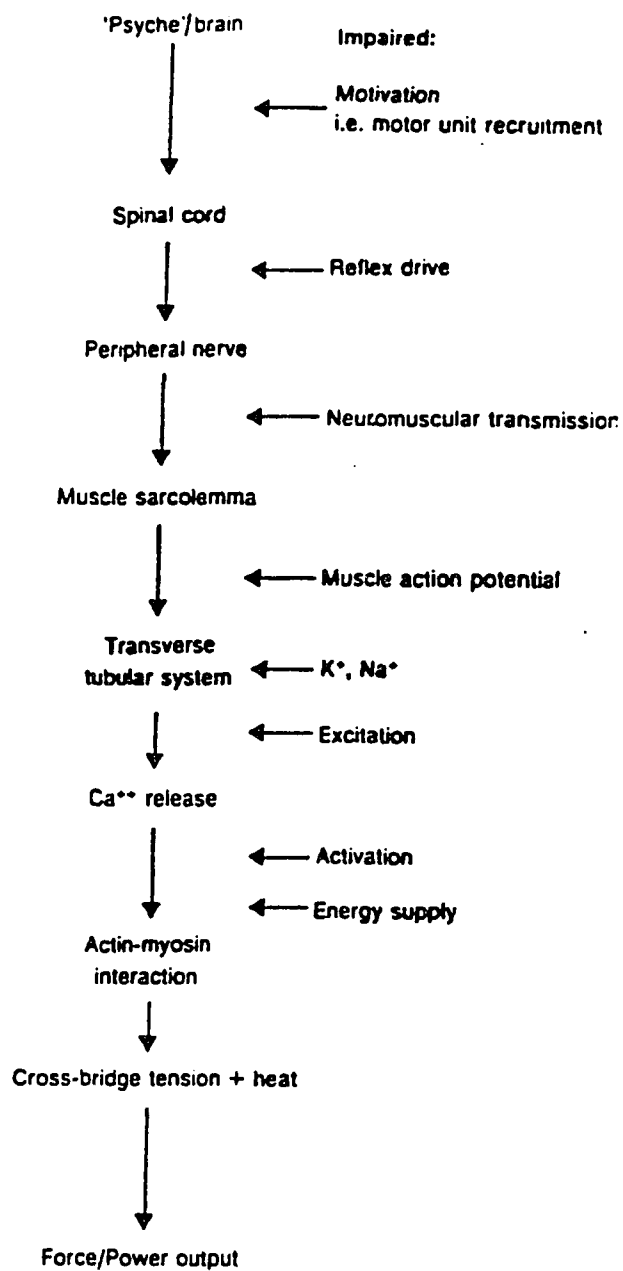


Figure 1: Current summary of command chain for muscle contraction (Armstrong, 1988)

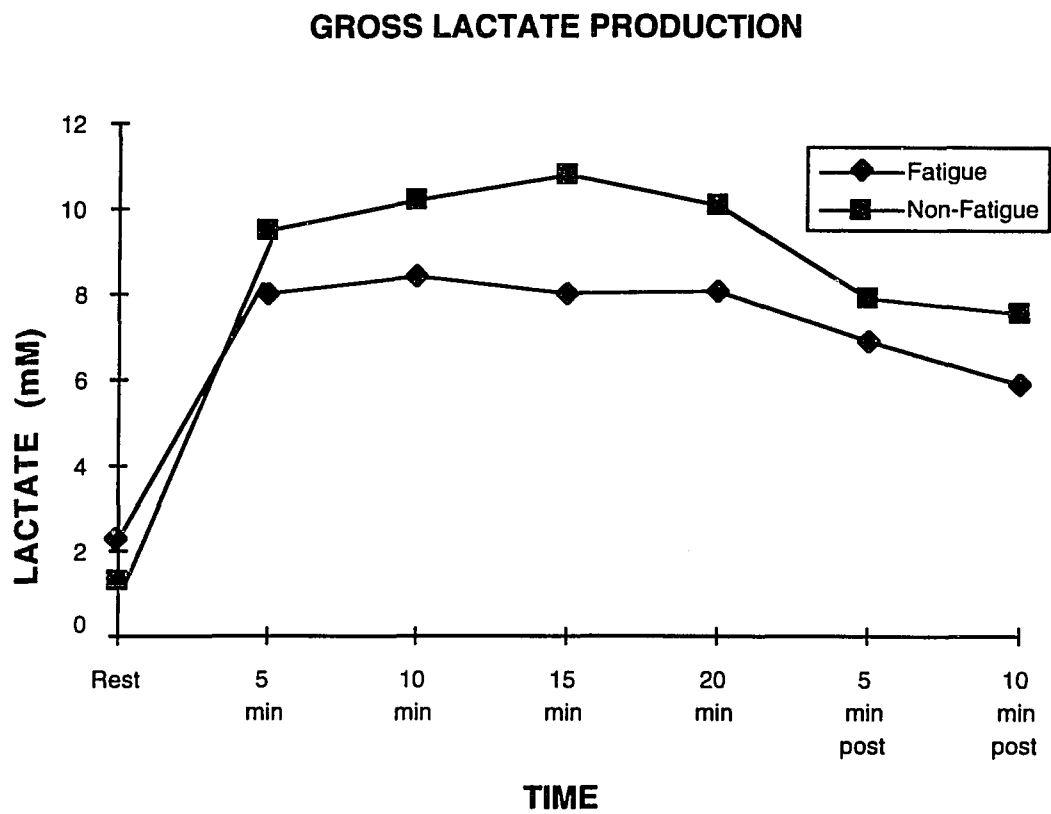


Figure 2: Gross lactate production in fatigue and non-fatigue trials.

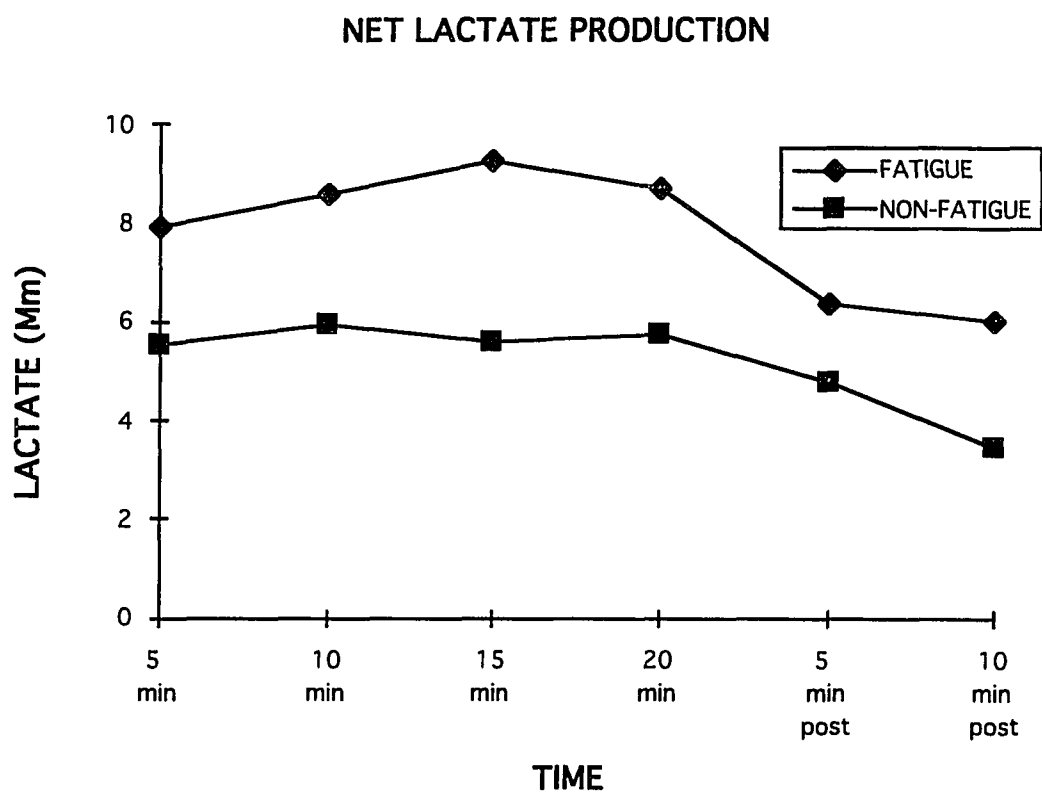


Figure 3: Net lactate production in fatigue and non-fatigue trials.

MEAN LACTATE PRODUCTION FOR EACH SUBJECT

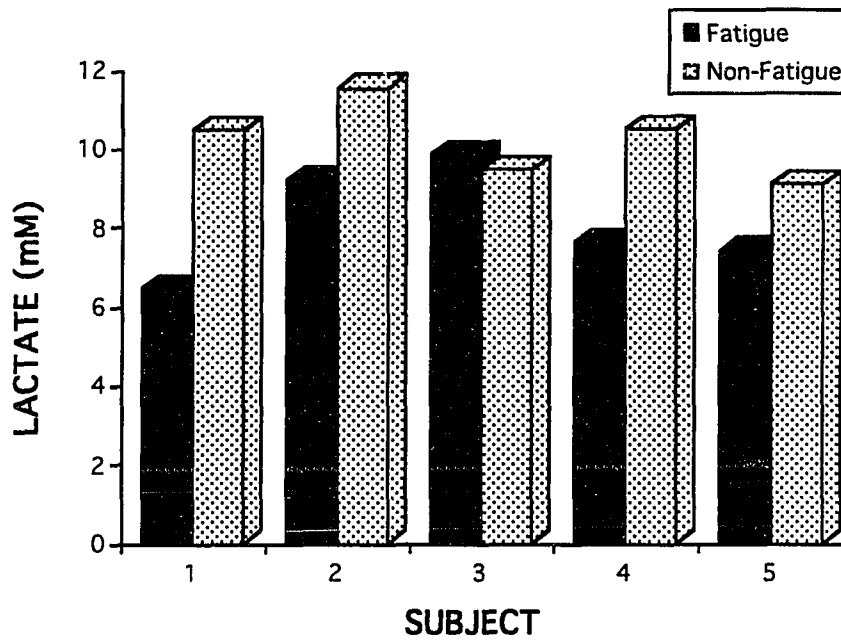


Figure 4: Mean net lactate production for each subject. Fatigued was significantly lower than non-fatigued ($p = .04$).

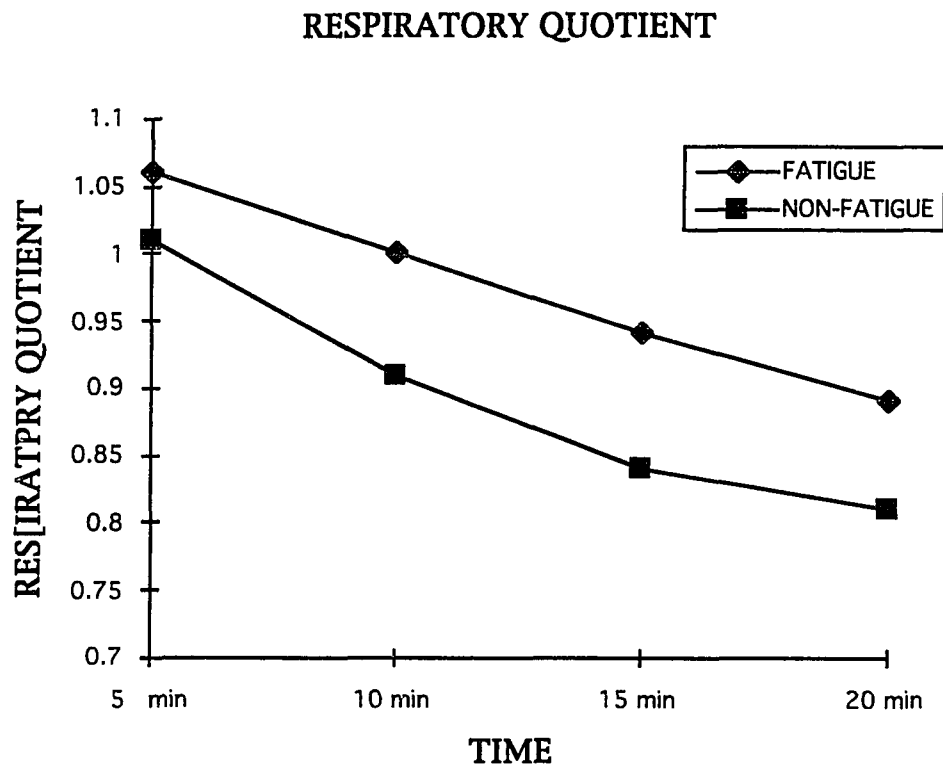


Figure 5: Respiratory Quotient for fatigue and non-fatigue trials.

APPENDIX III
TRIAL RECORDING FORMS

SUBJECT CHARACTERISTICS

Name_____Age_____

Height_____cm Weight_____kg

Sum of 4 skinfold:

Tricep_____

Abdomen_____

Iliac_____

Thigh_____

Total_____

MAX VO2 TEST

1 Min_____ Workload_____KGM _____%

2 Min_____ Workload_____KGM _____%

3 Min_____ Workload_____KGM _____%

4 Min_____ Workload_____KGM _____%

5 Min_____ Workload_____KGM _____%

6 Min_____ Workload_____KGM _____%

7 Min_____ Workload_____KGM _____%

8 Min_____ Workload_____KGM _____%

9 Min_____ Workload_____KGM _____%

TRIAL 2- date_____ time_____

TRIAL 3- date_____ time_____

The Effect of Localized Muscle Fatigue on Lactate Production during Submaximal Exercise

consist of the aerobic exercise on the cycle ergometer without the prior leg-extension exercise. The same exercise intensity and protocol for lactate measurement will be followed.

Risks and Benefits

Because this test involves puncturing the fingertip, you may experience some bruising. You may also experience localized muscle soreness from the knee extension exercise and the max VO₂ test. Proper preparation, precautions and supervision will minimize help minimize the risks involved. Be assured that major complications are minimal.

Right to Withdraw

You are free to refuse to participate in this study, or to withdraw at any time. Your decision will not adversely affect your status at UNLV or cause a loss of benefits to which you might otherwise be entitled.

Confidentiality

Your name will remain confidential from any publication or reporting of results. The form being signed will remain the property of the experimenter and the UNLV exercise physiology laboratory.

The Effect of Localized Muscle Fatigue on Lactate
Production during Submaximal Exercise

Subjects Statement of Consent

I have read the above description of this research study, and I understand it. I have been informed of the risks and benefits involved, and all of my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered. I understand that I will receive a copy of this form.

I understand that I am free to withdraw this consent and discontinue participation in this study at any time without prejudice.

I voluntarily consent to my participation in the described research study.

printed name of subject

signature of subject

date

APPENDIX II

PARTICIPANT IDENTIFICATION

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)*
A Self-administered Questionnaire for Adults

PAR Q & YOU

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and check the YES or NO opposite the question if it applies to you.

YES NO

- 1. Has your doctor ever said you have heart trouble?
- 2. Do you frequently have pains in your heart and chest?
- 3. Do you often feel faint or have spells of severe dizziness?
- 4. Has a doctor ever said your blood pressure was too high?
- 5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?
- 6. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
- 7. Are you over age 65 and not accustomed to vigorous exercise?

If You Answered

YES to one or more questions

If you have not recently done so, consult with your personal physician by telephone or in person BEFORE increasing your physical activity and/or taking a fitness test. Tell him what questions you answered YES on PAR-Q, or show him your copy.

programs

After medical evaluation, seek advice from your physician as to your suitability for:

- unrestricted physical activity, probably on a gradually increasing basis.
- restricted or supervised activity to meet your specific needs, at least on an initial basis. Check in your community for special programs or services.

NO to all questions

If you answered PAR-Q accurately, you have reasonable assurance of your present suitability for:

- A GRADUATED EXERCISE PROGRAM - A gradual increase in proper exercise promotes good fitness development while minimizing or eliminating discomfort.
- AN EXERCISE TEST - Simple tests of fitness (such as the Canadian Home Fitness Test) or more complex types may be undertaken if you so desire.

postpone

If you have a temporary minor illness, such as a common cold.

* Developed by the British Columbia Ministry of Health. Conceptualized and critiqued by the Multidisciplinary Advisory Board on Exercise (MABE). Translation, reproduction and use in its entirety is encouraged. Modifications by written permission only. Not to be used for commercial advertising in order to solicit business from the public.

Reference: PAR-Q Validation Report, British Columbia Ministry of Health, May, 1978.

* Produced by the British Columbia Ministry of Health and the Department of National Health & Welfare.

APPENDIX III
TRIAL RECORDING FORMS

SUBJECT CHARACTERISTICS

Name_____Age_____

Height_____cm Weight_____kg

Sum of 4 skinfold:

Tricep_____

Abdomen_____

Iliac_____

Thigh_____

Total_____

MAX VO2 TEST

1 Min_____ Workload_____KGM _____%

2 Min_____ Workload_____KGM _____%

3 Min_____ Workload_____KGM _____%

4 Min_____ Workload_____KGM _____%

5 Min_____ Workload_____KGM _____%

6 Min_____ Workload_____KGM _____%

7 Min_____ Workload_____KGM _____%

8 Min_____ Workload_____KGM _____%

9 Min_____ Workload_____KGM _____%

TRIAL 2- date_____ time_____

TRIAL 3- date_____ time_____

FATIGUE TRIAL

NAME _____

Resting Lactate level _____ Pre-exercise lactate _____

80% of Max VO₂ _____ KGM

MVC _____

70% MVC _____

Post-fatigue MVC _____ % of MVC _____

CYCLING

TIME	HEART RATE	VO ₂	LACTATE LEVEL
5 MIN			
10 MIN			
15 MIN			
20 MIN			
5 POST			
10 POST			

NON-FATIGUE TRIAL

NAME _____

TIME	HEART RATE	VO2	LACTATE LEVEL
5 MIN			
10 MIN			
15 MIN			
20 MIN			
5 POST			
10 POST			

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