

ABSTRACT

The Effects of Active and Passive Recovery on Blood Lactate in Collegiate Female Tennis Players

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Purpose: To examine the effects of active and passive recovery on lactate in females and to determine if a relationship exists between power and blood lactate concentration. Methods: Nine (9) female athletes performed two Wingate Power Tests. One test ended with passive recovery, the other ended with active recovery. Lactate was drawn during the recovery periods. From the Wingate Test, power indices were obtained. A t-test and an ANOVA were performed to evaluate differences in active and passive recovery and Pearson's correlations were used to examine relationships between power and lactate values. Results: Active recovery significantly ($p < 0.03$) increased lactate removal compared to passive recovery. There were no significant correlations between lactate and power measures. Conclusions: Active recovery increases the rate of lactate removal in females; however, lactate measures are not correlated to the power measures from the Wingate Test.

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Tennis Players

by

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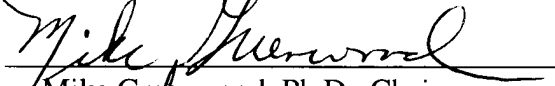
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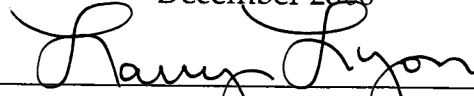

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LIST OF ABBREVIATIONS

AMP absolute mean power	MCT monocarboxylate transporter
ANOVA analysis of variance	N₂ nitrogen
APP absolute peak power	O₂ oxygen
AR active recovery	PDH pyruvate dehydrogenase
AT anaerobic threshold	pH concentration of protons/hydrogen ions
ATP adenosine triphosphate	PR passive recovery
ATPase adenosine triphosphatase	RMP relative mean power
CO₂ carbon dioxide	RPP relative peak power
FI fatigue index	STOF slow twitch oxidative fibers
H⁺ hydrogen ion	VCO₂ carbon dioxide production
H₂O hydrogen dioxide, water	V_E pulmonary ventilation
La lactate	VO₂ oxygen consumption
LDH lactate dehydrogenase	VO_{2max} maximal oxygen consumption
LRR lactate rate of removal	W watt

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CHAPTER ONE

Introduction

In athletics, advancements in technology and a greater understanding of biological systems have been key components in improving performance. Lactate concentration is one of the many blood markers that researchers have investigated. An understanding of the physiology of lactate has enabled physiologists and coaches to use this parameter as an indicator of glycolytic stress (Bishop & Martino, 1993). As the precision of the analysis of blood lactate increases, portable lactate analyzers provide rapid results from a finger-prick.

Lactate is a metabolic intermediate produced from the conversion of pyruvate through the catalytic action of lactate dehydrogenase (LDH) (Katz & Sahlin, 1988; Spriet, Howlett, & Heigenhauser, 2000). The rate of lactate production in skeletal muscle mainly depends upon the flux of substrate through the glycolytic pathway (Connett, Honig, Gayeski, & Brooks, 1990). At rest, there is normally a continuous production of lactate. Resting levels of blood lactate are approximately 1 millimole in untrained subjects; however, in elite athletes, resting lactate levels can be as much as 4mM (Donovan & Pagliassotti, 2000). During low intensity exercise (below 40% VO₂ Max) lactate levels show little fluctuation from baseline (Gollnick, Bayly, & Hodgson, 1986). At higher power outputs, with a greater demand for ATP, the rate of glycolysis increases (Gollnick et al., 1986). If the rate of pyruvate production exceeds the ability of pyruvate dehydrogenase (PDH) to metabolize the substrate, lactate is produced, increasing blood lactate levels (Spriet et al., 2000).

Several studies have suggested that lactate accumulation is associated with muscular fatigue and hinders performance (Ahmaidi et al., 1996; Bond, Adams, Tearney, Gresham & Ruff 1991; Bonen & Belcastro, 1977; Fitts & Holloszy, 1976; Froese & Houston, 1987; Gollnick et al., 1986; Karlsson & Saltin, 1970). Fatigue results from two main processes: a) the accumulation of substances, such as lactate, pyruvate, hydrogen ions and carbon dioxide, and b) the depletion of substances, such as glucose, glycogen, and O₂ necessary for activity (Brooks, Fahey & White, 1996; Häkkinen & Myllylä, 1990). Normal pH of skeletal muscle is approximately 7.0 at rest (Gollnick et al., 1986). With the production of lactate, as demonstrated in high intensity exercise, there is also a production of hydrogen ions (Pilegaard et al., 1999). This increase in hydrogen ion concentration interferes with anaerobic metabolism by disrupting the activities of key enzymes; it is also associated with attenuation in ATP production, lipolysis, and muscle tension (Belcastro & Bonen, 1975; Fitts & Holloszy, 1976; Gollnick et al., 1986; Monedero & Donne, 2000; Sahlin & Henriksson, 1984; Wasserman, Beaver, & Whipp, 1986). Acidosis, associated with lactate accumulation, has been reported to have detrimental effects on the contractile process within the musculature (Fitts & Holloszy, 1976). Hydrogen ions displace calcium from troponin, which causes interference in muscle contraction (Gollnick et al., 1986). It is the production of these hydrogen ions and, therefore, the decrease in pH that causes the effects associated with fatigue (Gollnick et al., 1986; Robergs, 2001; Wasserman et al., 1986).

Lactate is removed from the blood through a combination of methods. Lactate is metabolized by the heart, brain and liver through oxidation (Bonen, Campbell, Kirby, & Belcastro, 1979; Ide, Schmalbruch, Quistorff, Horn, & Secher, 2000; Katz, 1986). However, the primary mode of lactate removal is via oxidation in slow-twitch, oxidative

fibers (STOF) (Brooks & Gaesser, 1980; Gladden, 2000; Mazzeo, Brooks, Schoeller, & Budinger, 1986; Pagliassotti & Donovan, 1990a; Pagliassotti & Donovan, 1990b), and the rate of uptake is directly proportional to the rate of blood flow (Brooks, 1991). Monocarboxylate transporter 1 (MCT1), which has been associated with lactate removal, is found predominately in STOF (Bonen, 2000; Brooks, 2000), indicating its importance in the removal of lactate. The work/rest ratio during recovery periods also plays an important role in lactate removal (Baechle & Earle, 2000). For longer rest periods, with a lower lactate concentration and stroke volume the buffer capacity and aerobic power production are minimal (Baechle & Earle, 2000). On the other hand, if shorter rest periods are employed, the opposite adaptations are seen, but at the expense of optimal speed (Baechle & Earle, 2000).

Studies have shown that lactate removal is significantly increased through the application of active recovery compared to passive recovery (Ahmaidi et al., 1996; Bangsbo, Graham, Johansen & Saltin, 1994; Bond, Adams, Tearney, Gresham & Ruff, 1991; Francaux, Jacqmin, Michotte de Welle, & Sturbois, 1995; Gupta, Foswami, Sadhukhan, & Mathur, 1996; Mondero & Donne, 2000). Differences in intramuscular lactate clearance between active and passive recovery are primarily due to a difference in the metabolism within the target muscle (Bangsbo et al., 1994; Katz & Sahlin, 1988). While there are a number of studies that examine the removal of lactate during active recovery in males, there are few studies that examine the removal of lactate within female subjects. During the literature search only one study was found that examined mixed-gender subjects (4 female and 3 male) and recovery measures on lactate (Hermansen & Stensvold, 1972) and no studies could be located that exclusively examined female subjects and the removal of lactate via recovery measures. With the lack of literature on

lactate response to recovery in female subjects, it would be beneficial to examine this population. In a study conducted by Simoneau et al. (1985), researchers found that male subjects exhibited higher glycolytic enzymatic activity than female subjects. Furthermore in a study conducted by Kent-Braun, Ng, Doyle and Towse (2002), female subjects appeared to rely less on anaerobic pathways for the supply of ATP during muscular contractions compared to male subjects. Finally, in a study conducted by Jacobs, Tesch, Bar-Or, Karlsson, & Dotan (1983), male subjects produced higher lactate concentrations when compared to female subjects after supramaximal exercise. It appears that female subjects respond differently in lactate production. Is there a difference in lactate removal in this population? Therefore the primary purpose of this study is to determine the effects of active and passive recovery on lactate dynamics in female subjects.

The Wingate Anaerobic Test, introduced in 1974, has been used in various laboratories to help evaluate physiological responses to supramaximal exercise (Bar-Or, 1987; Froese & Houston, 1987). The test consists of a 30-second cycle ergometer test at maximal speed against a constant force. The National Strength and Conditioning Association states that the work/rest ratio's for the high energy phosphate system, the anaerobic glycolytic system, and aerobic system are 1:12 to 1:20, 1:3 to 1:5, and 1:1 to 1:3 respectively (Baechle & Earle, 2000). The Wingate anaerobic test taxes the high energy phosphate system, simulating sprinting, jumping or heavy lifting (Baechle & Earle, 2000). From this test, three parameters of power can be obtained: (a) peak power-the highest mechanical power, (b) mean power-the average mechanical power throughout the 30 second period, and (c) fatigue index-the degree to which power decreases during the test (Bar-Or, 1987; Jacobs et al., 1982). These three measurements are positively

correlated with the presence of fast-twitch, high glycolytic muscle fibers (Bar-Or, 1987; Froese & Houston, 1987; Weinstein, Bediz, Dotan, & Falk, 1998). Also, lactate production is correlated with the presence of fast-twitch muscle fibers (Bar-Or, 1987; Connett, Gayeski, & Honig, 1984; Fitts & Holloszy, 1976; Gollnick et al., 1986; Sahlin & Henriksson, 1984; Stainsby, 1986). Therefore, another purpose of this study is to determine if there is a correlation between power measures obtained from the Wingate Test and lactate production.

Research Questions

As mentioned previously there is little research on the effects of active recovery on blood lactate measures in female athletes, despite published literature on the differences between male and female subjects with lactate production (Jacobs et al., 1983). The following questions were investigated:

1. Is there a difference in lactate removal between active and passive recovery in female collegiate tennis players?
2. Will significant correlations be seen between power measures (peak power, mean power, and fatigue index) and lactate production?

Significance of the Study

This research is significant because it is comparing active and passive recovery methods using a female sample. There has been little research on this issue in the female population despite differences in physiological responses to exercise exhibited by male and female subjects.

Hypotheses

The following hypotheses are based upon research which utilized solely male subjects or both male and female subjects (Ahmaidi et al., 1996; Bangsbo et al., 1994; Bond et al., 1991; Francaux et al., 1995; Gupta et al., 1996; Mondero & Donne, 2000). It is hypothesized that:

- H₁ There will be a statistically significant increase in plasma lactate removal during active recovery compared to passive recovery in female collegiate tennis players.
- H₂ There will be a significant positive correlation between power measurements and plasma lactate production.

Independent and Dependent Variables

The independent variable in this study is the method of recovery, active or passive. The dependent variables in this study are plasma blood lactate, peak power, mean power, and fatigue index.

Definition of Terms

The following terms are used throughout this study to describe aspects of lactate removal, exercise recovery measures, and power production.

- Active recovery – exercise sustained at sub-maximal intensity (Ahmaidi et al., 1996; Baechle & Earle, 2000; Bangsbo et al., 1994; Mondero et al., 2000).
- Exercise induced acidosis – condition in which there is an increase in H⁺ concentration (decrease in pH) as a result of exercise (Robergs, 2001).
- Fatigue index – rate of fatigue, the degree of power drop-off during the test (Bar-Or, 1987).

- Lactate dehydrogenase – enzyme that converts pyruvate into lactate by the addition of hydrogen from NADH_2 (McArdale, Katch & Katch, 2000; Spriet et al., 2000).
- Lactate oxidation – process by which lactate is metabolized by surrounding tissues through the donation of electrons (Brooks, 1986; McArdale et al., 2000).
- Lactate shuttle – mechanism by which lactate is moved throughout the interstitium and vasculature; enables glycogenolysis in one cell to supply another with lactate for oxidation (Brooks, 2000; McArdale et al., 2000).
- Lactate threshold – point at which the production of lactate exceeds its removal rate (Antonutto & Di Prampero, 1995; Moquin & Mazzeo, 2000).
- Mean power – the average power maintained over the 30 second test (Bar-Or, 1987).
- Monocarboxylate transporters – transporters that facilitate the exchange of lactate between fast-twitch and slow-twitch muscle fibers (Bonen, 2000; Brooks, 2000).
- Passive recovery – inactive rest (Ahmaidi et al., 1996; Baechle & Earle, 2000, Bangsbo et al., 1994; Mondero et al., 2000).
- Peak power – the highest 5 second mechanical power produced during the 30 second test (Bar-Or, 1987).
- Pyruvate dehydrogenase – enzyme that catalyzes the decarboxylation of pyruvate to form acetyl-CoA (Howlett et al., 1998; McArdale et al., 2000).

Limitations

The following are conditions which are beyond the researcher's control which may or may not influence the outcome of the hypotheses.

- The research design does not include male subjects as a control or comparison.
- The study included a small sample size (n=9).
- The conclusions of the study may only apply to collegiate aged female athletes.
- Subjects were recruited and not randomly selected.
- First-aid measures were not monitored post test by researchers, as subjects were instructed to seek further medical attention from the athletic training staff and team physician.

Delimitations

The following are parameters set to provide consistency within the study and to narrow the scope of the study.

- Female subjects from 18 to 22 years of age, were recruited from the Baylor University Varsity Athletic Women's Tennis Program. This provided information for a homogenous group of subjects.
- To ensure test accuracy, the equipment and environment were the same for both tests. Each subject performed all testing using the same Monark™ cycle ergometer. The cycle ergometer was calibrated prior to testing sessions.
- Subjects were instructed not to perform additional exercise prior to testing. In addition, subjects were instructed not to eat a meal 12 hours before testing.

- One researcher and the same Accusport™ lactate analyzer obtained all blood samples. The Accusport™ lactate analyzer was calibrated prior to testing of subjects.
- Thorough explanation and demonstration of the research protocol was done prior to testing.

Assumptions

The following statements are assumptions made by the researcher in order to proceed with the study. They describe what is assumed of the subjects of the study and the reliability and reproducibility of testing parameters.

- It was assumed that over a week's period, an individual's rate of lactate removal and production does not change without training (Gollnick et al., 1986; Tomlin & Wenger, 2001).
- It was also assumed that the Accusport™ portable lactate analyzer accurately measures blood lactate from the reagent strips (Boldt, Kumle, Suttner, & Haisch 2001; Fell, Rayfield, Gulbin, & Gaffney, 1998; Yam, Chua, Razvi, & Arulkumaran, 1998).
- It was assumed that each subject followed verbal and written instructions and completed the survey honestly.
- Finally, the following assumptions were made concerning lactate dynamics: 1) lactate removal occurs via oxidation to CO₂ and H₂O and/or resynthesis to glycogen, and 2) blood lactate samples are proportional to whole body lactate (Berthoin et al., 2002; Brooks, 1991; Gollnick et al., 1986; Oosthuysen & Carter, 1999; Yam et al., 1998).

CHAPTER TWO

Review of Literature

Introduction

The following review will discuss pertinent metabolic aspects of lactate disposal and varying methods of lactate recovery. The review covers the metabolism of lactate through oxidation and glyconeogenesis. It explains the importance of metabolic transporters and fiber type in lactate metabolism. It also reviews the current literature on lactate removal through active recovery and subsequent muscular performance.

Lactate Metabolism

In a study by Mazzeo et al. (1986), the magnitude and extent of blood lactate disposal and oxidation was evaluated at rest and at two different exercise intensities using a non-radioactive tracer ($1\text{-}^{13}\text{C}$) lactate. The study included six healthy male subjects all accustomed to cycle ergometer exercise. Prior to the experiment $\text{VO}_{2\text{ max}}$ was determined for each subject in order to set workloads. The exercise bouts were two separate levels of intensity defined as: 1) easy exercise, exercise which causes a relatively small change in lactate concentration compared with resting, and 2) heavy exercise, exercise which causes a high lactate concentration. The rate of O_2 consumption (VO_2), rate of CO_2 production (VCO_2) and the respiratory exchange ratio ($\text{R}=\text{VCO}_2/\text{VO}_2$) were monitored throughout testing. To assess the amount of enriched CO_2 produced samples of expired air were collected in vacutainers. To assess the amount of enriched lactate, lactate was separated from other blood metabolites. Dunn's multiple comparison was used to assess the three lactate testing conditions. A linear regression demonstrated the relationship

between 1) lactate disposal and VO_2 and 2) lactate disposal and blood lactate concentration. The results of the study demonstrated that during both heavy and easy exercise, lactate concentrations rose significantly compared to rest, with a five-fold increase during heavy exercise. They also found that lactate was produced continuously, even at rest. In addition, lactate oxidation increased directly with an increase in metabolic rate. From this information the authors concluded that there was a direct linear relationship between blood lactate disposal and the metabolic rate through the oxidation of lactate. Similarly, Brooks (2000) found that at 75% of lactate disposal was through oxidation during steady state exercise and recovery from exercise and that lactate production is evident at rest. Further, Miller et al. (2002), found that increased blood lactate during moderate exercise not only increases lactate oxidation but also spared blood glucose and decreased glucose production in healthy active male subjects.

Glyconeogenesis is another method of lactate disposal. A rabbit study by Donovan and Pagliassotti (2000) assessed the pathways of lactate disposal in various skeletal muscle fiber types. Three different muscle preparations were identified for testing purposes: glycolytic (Type IIb fibers), oxidative (Type I fibers) and mixed (Type IIa fibers). All muscle preparations were initially perfused with 1 mM of lactate to mimic resting levels. This initial perfusion caused a net lactate release in all muscles preparations. However, the net release in the oxidative preparation was the lowest followed by the mixed preparation, with the glycolytic preparation contributing the highest net lactate release. As lactate concentrations were increased all muscle preparations demonstrated a transition from net lactate release to net lactate consumption. For the oxidative preparation this transition occurred at 1-2 mM of lactate, while the transition for the glycolytic preparation did not occur until 4 mM. To assess the disposal

pathways of the different muscle preparations, radioactive tracers of ^{14}C -Lactate and $[^3\text{H}]$ glucose were perfused into the preparations. It was determined that the oxidation of lactate to carbon dioxide (CO_2) was a significant pathway for all preparations representing 28%, 51%, and 39% for the glycolytic, oxidative, and mixed preparations respectively. Tracer analysis was also employed to estimate glyconeogenic rates. Of the three fiber types only Type II fibers showed glyconeogenesis as a significant pathway of disposal, and found it to be a dominant pathway in Type IIb fibers. These findings demonstrate the selectivity fiber type may have for different modes of lactate disposal.

Åstrand, Hultman, Juhlin-Dannfelt & Reynolds (1986) also evaluated glyconeogenesis as a main avenue of lactate removal. The study included seven healthy volunteers (5 men and 2 women). The experimental exercise consisted of heavy dynamic exercise using both arm and leg muscles on a cycle ergometer until exhaustion. Blood samples were obtained from the brachial artery and the femoral vein at rest and 60 min post exercise. Muscle biopsies were also obtained from the vastus lateralis muscle. Gas samples were analyzed for O_2 , CO_2 , and N_2 by mass spectrometry. Blood samples taken were analyzed for their acid-base status, hematocrit, glucose concentration and lactate concentration. The results found that 50% of the lactate formed was synthesized into glycogen, observed predominately in fast-twitch muscle. In addition there was a 10% consumption of lactate observed through the liver.

There are still differing opinions on whether glyconeogenesis occurs within the recovering muscle tissue or whether lactate is transported to other tissues for disposal. Bangsbo et al. (1994) suggested that most of lactate released was not available for resynthesis, contrary to the findings of Åstrand et al. (1986).

There has been a recent focus in the literature about the role of lactate transporters. Bonen (2000) reviewed the role of these important proteins. There have been seven monocarboxylate transporters (MCT1-MCT7) identified. MCT1, cloned in hamster ovarian cells in 1994, has been identified in rat studies to cause a significant increase in lactate uptake, and is also found to be a stereo selective, proton coupled lactate transporter. MCT2 has similar kinetic characteristics for lactate transport as seen with MCT1, however the kinetics of the two differ in their ability to transport pyruvate. It should be noted that MCT2 has not been detected in humans. This suggests that MCT2 may be species specific. MCT3 has only been found in the retinal epithelium of chickens and rats, containing amino acid sequences similar to that of MCT1 and MCT2. MCT4 was found more abundantly in fast-twitch oxidative fibers and fast-twitch glycolytic fibers than slow-twitch fibers. MCT5 and MCT7 have only been found in the heart and MCT6 has been identified in both the heart and skeletal muscle. The significance of MCT5, 6 and 7 still remains unclear. Studies have also shown that there is a significant correlation between oxidative fibers and MCT1 expression. This suggests that MCT1 is the primary transporter in those types of fibers. Also, with an increase in MCT1 expression, there is an increase with lactate movement along the sarcolemma. The transport of lactate across cellular membranes may be a key factor in lactate removal in slow twitch muscle fibers (Bonen, 2000; Brooks, 2000).

In a study by Jacobs et al. (1983), researchers sought to examine the differences in lactate production after supramaximal exercise between male and female subjects. The study included 15 male and 7 female physical education students. Each subject performed two supramaximal exercise bouts of 10 and 30 s on a cycle ergometer. Muscle biopsies were obtained to analyze lactate concentration. The male subjects had

significantly higher lactate concentrations and higher power production compared to the female subjects.

Lactate Recovery Methods

There are numerous studies that focus on the effects of active recovery on lactate removal. Bangsbo et al. (1994), sought to determine the net exchange of metabolites during active and passive recovery. The study included six healthy male subjects, ages ranging from 22-26 yrs. Prior to the experiment subjects practiced the exercise on four separate occasions. A catheter was placed in the femoral artery and femoral veins. All subjects performed a warm up on a cycle ergometer at a work rate of 10 watts. During the testing portion, subjects performed two exhaustive cycle bouts; one was followed by 10 minutes of active recovery and the other by passive recovery. The recovery modes were randomly assigned. The protocol was repeated after an hour of recovery with the opposite leg. Blood flow was measured by thermodilution of the femoral vein. Leg blood flow was measured at 0.5, 1.3, 2.4, 3.2, 5.2, 7.2, and 9.8 minutes of recovery. Blood samples were taken at 0.8, 1.8, 2.9, 5.5, 7.8, and 10.2 minutes of recovery. Lactate was analyzed with the use of a fluorometric assay. Net lactate exchange by the thigh was calculated by multiplying blood flow by femoral via blood concentration differences. Lactate metabolism within the muscle was calculated as the difference between net lactate decrease and net lactate release.

Mean blood flow for passive recovery was found to be lower than active recovery. Immediate post exercise lactate concentration was similar for both modes of recovery. However, after 10 minutes of recovery, active recovery had a significantly reduced arterial lactate concentration. The decline in the net lactate release was similar for both modes of recovery; however, there was a small net lactate efflux for passive recovery,

whereas the net release for passive recovery was nil. Muscle glycogen was not significantly altered during either active or passive recovery. It was concluded that low intensity exercise reduced muscle lactate more rapidly than passive recovery. Furthermore, the high release of lactate and pronounced uptake of glucose during both active and passive recovery indicate that lactate is not significant in the synthesis of glucose through intramuscular glyconeogenesis.

Stamford et al. (1981) evaluated the effects of active recovery above and below anaerobic threshold on lactate removal. The study included six healthy male subjects, with a mean age of 27. Each subject performed a VO_2 max test on a Collins electric bicycle ergometer. Each subject performed 3 maximal cycle ergometer tests on a Monarch bicycle on separate occasions. Each test was followed by either passive recovery, active recovery at 40% VO_2 max, or active recovery at 70% VO_2 max. Blood samples were drawn at rest and during recovery. Their results indicated that active recovery at 40% VO_2 max was significantly more efficient at the removal of lactate.

In a related study by Dodd et al. (1984), the researchers evaluated the effects of active recovery at 35%, 65%, and a combination of 35% and 65% VO_2 max on lactate removal. This study included seven male subjects with a mean age of 29. The cycling experiment consisted of three 50 sec maximal bouts on a cycle ergometer. Following the exercise portion, the subjects performed one of the following recovery periods for 40 min: 1) passive recovery, 2) active recovery at 35% VO_2 max, 3) active recovery at 65% VO_2 max, and 4) active recovery at 65% VO_2 max for 7 min immediately followed active recovery at 35% VO_2 max for 33 min. Each subject performed all recovery periods. Blood samples were obtained throughout the recovery periods. Cycling at 35% VO_2 max

and the combination of 65% and 35 % VO_2 significantly increased the rate of lactate removal when compared to cycling at 65% VO_2 max alone.

Francaux et al. (1995), investigated lactate metabolism during active and passive recovery. Five healthy male subjects volunteered for the study. Each subject was required to perform six tests. The exercise portion consisted of 1 minute on a cycle ergometer at a power output of 385 W at 110 rpm. The recovery portion of the study consisted of passive recovery and active recovery at 5 different intensities: 60, 90, 120, 150, and 180 watts for 1 hour. Blood samples were taken at the ear lobe. The rate of lactate disappearance increased from passive recovery to moderate exercise recovery (60 watts) and decreased from moderate exercise recovery to intense exercise recovery.

Researchers have also tested muscle massage as another method of lactate removal. In a study by Gupta et al. (1996), researchers sought to determine the effects of short-term massage on lactate removal. The study included ten male subjects. All subjects performed a VO_2 max to determine the workload setting. The exercise portion was performed on a cycle ergometer at 150% VO_2 max for 60 sec. Tests were followed by active recovery, passive recovery, or short-term massage. Blood samples were drawn during each recovery period and analyzed. Active recovery was found to be the most effective means of lactate removal, with short-term massage not significantly different from passive recovery.

During athletic competition, recovery after exercise is a self-selected event. To evaluate the effectiveness of self-selected recovery, Belcastro and Bonen (1975) compared this mode of recovery to controlled active recovery and passive recovery. This experiment included seven male subjects. All tests were performed on a bicycle ergometer at 90% VO_2 max followed by 30 min of recovery. There were five controlled

recovery periods: passive recovery and active recovery at 30 %, 45% 60% and 80% of VO_2 max. Additionally there were two uncontrolled recovery periods, in which intensity was determined by each individual subject. Blood samples were taken during each recovery period. Self-selected recovery and active recovery at 30% and 45% VO_2 max were found to be the most effective rates for removing blood lactate.

Research in the area of lactate recovery has also focused the differences between sprint and endurance athletes. Taoutaou et al. (1996) focused their research on lactate kinetics during active and passive recovery in endurance and sprint athletes. The study included seven endurance and seven sprint trained male athletes. All subjects performed a VO_2 max test prior to the exercise portion. The exercise portion was performed on a Monarch cycle ergometer beginning with a 3 min warm-up at 60 W. During the exercise bout subjects were instructed to pedal at 60 rpm as the intensity increased by levels of 30 W every minute until exhaustion. Blood samples were drawn via catheter from a superficial forearm vein at intervals during recovery. Results from blood analysis showed that during the first minute of recovery the endurance trained subjects produced higher concentrations of lactate when compared to the sprint trained subjects. Furthermore, peak lactate was higher for the endurance-trained subjects during passive recovery. However, the rate of removal of lactate was the same for both groups during passive recovery. During active recovery, endurance trained athletes were able to significantly remove more lactate when compared to their sprint trained counterparts.

Karlsson and Saltin (1970) evaluated the relationship between muscular fatigue and lactate concentration. Three healthy male subjects performed six experiments (three exhaustive and three non-exhaustive) over a period of two months. Subjects performed heavy exercise to exhaustion at three different workloads (high, medium, and low) on a

Krogh cycle ergometer. Blood lactate was determined through enzymatic analysis. There were no significant differences in lactate concentration at the three different workloads. The researchers concluded exhaustive exercise produced greater amounts of lactate compared to non-exhaustive exercise.

Ahmaidi et al. (1996) explored the effects of active recovery on plasma lactate concentration and subsequent anaerobic power output following repeated exercise bouts. The study included ten healthy male subjects ages 24-29 years. All subjects performed incremental aerobic exercise and two force velocity tests. During aerobic testing, respiratory variables and gas exchanges were measured with a breath-by-breath metabolic system. The force velocity test consisted of repeated bouts of intensive exercise against increasing breaking forces which were separated by a 5 min. recovery period. Blood samples were taken via catheter placed in a superficial forearm vein and analyzed for plasma lactate.

During the force velocity tests that were interspersed with passive recovery there was a significant positive correlation between the increase in power output and the increase in lactate measured. Lactate was calculated to be significantly lower from the force velocity test with active recovery compared to the force velocity test with passive recovery. For lower forces (2 kg and 4 kg), the mean power output values for active recovery were not significantly different from passive recovery. However, the mean power output, at 6 kg of force was significantly different between active and passive recovery.

The researchers concluded that the force velocity test with repeated rest periods of active recovery lowered plasma lactate concentrations. Furthermore, with this decrease in plasma lactate concentration, there was a corresponding increase in the power output at

participants' breaking forces. Similarly, Spierer, Goldsmith, Baran, Hryniewicz and Katz (2004) found that active recovery at 28% of VO_2 max increased total work achieved during repeated 30 s Wingate anaerobic tests compared to passive recovery.

However there have been conflicting reports on subsequent performance after active recovery. Bond et al. (1991) examined the effects of active and passive recovery on lactate removal and subsequent muscle function. The study included five healthy, active, male subjects. Each subject took part in the random experimental sessions. Each session was divided into four periods. The first period consisted of establishing the baseline lactate level via catheter placed in the cubital vein of the forearm. Lactate was assayed in the triplicate using the enzymatic chemical reaction method. The second period consisted of 60 seconds of supramaximal exercise (150% VO_2 max on a cycle ergometer). Five minutes after supramaximal exercise, a blood sample was taken and analyzed for lactate concentration. The third period consisted of another active recovery for 20 minutes at 30% VO_2 max or passive recovery (sitting on the ergometer). The fourth period consisted of an isokinetic muscle test, which examined peak torque, work output and fatigue in the quadracep extensor muscle. For data analysis, a repeated measures of analysis of variance was used. For significant results, a multiple t-test for post hoc analysis was applied to locate the source of the significant difference. Analysis of data indicated the rate of lactate removal during active recovery was significantly different ($p < 0.05$) compared to passive recovery. From these results the researchers concluded that while there was an increase in lactate removal from the blood, this lower concentration of lactated had no effect on subsequent muscle function. Further, in a study by Dupont, Blondel, and Berthoin (2003), the researchers found that after a 30s

supramaximal exercise bout passive recovery allowed their subjects to perform longer compared to active recovery.

In order to determine an optimal intensity and duration for active recovery for subjects with varying levels of fitness, Gmada et al. (2005) compared seven trained and seven untrained healthy subjects. Subjects performed three supramaximal intermittent exercise followed by either: passive recovery (PR), active recovery corresponding to the first ventilatory threshold minus 20% (VT1), active recovery corresponding to the second ventilatory threshold minus 20% (VT2), or a combined active recovery which consisted of 7 min at VT2 followed by 13 min at VT1 (CR). Peak lactate was observed significantly earlier with VT2 and CR compared to VT1 and PR. CR showed significant faster lactate clearance compared to PR, VT1, or VT2.

To examine the extent fiber type plays in lactate removal, Bonen et al. (1979) investigated the role of muscle fiber composition in the removal of blood lactate. Ten physically active women (exercised 3 days per week) took part in this study. Each subject was to perform an exercise bout on a cycle ergometer at 90% VO_2 max for 6 minutes. This exercise bout was followed by 20 minutes of passive recovery, and on another occasion by 20 minutes of active recovery at 30% VO_2 max. Blood samples were taken at the forearm vein at 0 (immediately post exercise), 5, 10, 15, and 20 minutes during each recovery period. Muscle samples were taken at rest via needle biopsy from the vastus lateralis of the right and left legs several weeks prior and several weeks after exercise portion. Fast twitch and slow twitch muscle fibers were identified by staining for myosin ATPase density. Active recovery was found to be significantly faster than passive recovery for the rate of lactate removal. Furthermore, a significant correlation was found between the La removal rate and the % of slow twitch fibers, and the lactate

concentration. From these findings, the researchers concluded that active recovery and slow twitch fiber content both play roles in the removal of lactic acid.

Summary

To summarize, lactate is primarily metabolized through oxidation which occurs primarily in slow-twitch muscle fibers (Bonen et al., 1979; Donovan & Pagliassotti, 2000; Mazzeo et al., 1986). Further, the concept of O₂ deficit may be inaccurate due to the continuous production of lactate at rest (Brooks, 2000; Mazzeo et al., 1986). Lactate produced from fast twitch fibers may also be metabolized in the liver through glyconeogenesis (Åstrand et al., 1986; Donovan & Pagliassotti, 2000). The simultaneous production and utilization of lactate demonstrates that it is an important energy source in working muscles. Intracellular and extracellular lactate transporters may also play a role in the removal of lactate through oxidation (Bonen, 2000; Brooks, 2000). Finally, there are an abundance of studies that have shown that active recovery increases the rate of lactate removal (Ahmaidi et al., 1996; Bangsbo et a., 1994; Bonen et al., 1979; Bond et al., 1991; Dodd et al., 1984; Francaux et al., 1995; Gupta et al., 1996; Stamford et al., 1981); however, none of these studies have evaluated a female sample.

CHAPTER THREE

Methods

Research Design

A repeated measures design was used in this research study. The testing protocol was identical for both testing sessions with the exception of either active or passive recovery post testing.

Description of Subjects

Nine collegiate female tennis players were recruited as subjects from the Baylor University varsity athletic team. This study was approved by the University Internal Review Board for humans as subjects prior to any data collection procedures. All subjects were given an informed consent letter to sign (Appendix A). They were also asked to fill out a fitness readiness questionnaire (Appendix B) before testing to identify subjects contraindicated for inclusion to this research to reduce possible health risks.

Subjects were instructed to maintain their normal diet on the day before the test. Additionally, subjects were asked not to exercise on the day of the test, to refrain from consuming caffeine and alcohol at least 12 hours before the test and to refrain from eating at least 2 hours before the test. The consumption of alcohol and caffeine may cause dehydration. In women, low levels of dehydration have been shown to shift lactate threshold, decreasing the time to exhaustion (Moquin & Mazzeo 2000).

Data Collection

The following measures were taken before testing: resting heart rate (bpm), resting blood pressure (mmHg), height (in), weight (kg), and resting blood lactate (mM). These procedures were done utilizing a heart rate monitor, a sphygmomanometer, a tape measure, a scale and, a finger-stick protocol, respectively.

The AccusportTM portable lactate analyzer measures lactate by enzymatic determination and reflective photometry (wavelength 660 nm) from whole blood and plasma. The instrument has been tested using guidelines of the European Committee for Clinical Laboratory Standards and has cleared Federal Drug Administration for use in sports medicine. The AccusportTM portable lactate analyzer is calibrated by using coding information on the lot of test strips. One of these code strips is taped to each vial of test strips. This code strip contains the calibrating information for the current lot of test strips on a bar code. To calibrate the analyzer it must be turned on and the flap closed over the measuring area. The code strip is then inserted into the slot at the base of the analyzer. The code strip is inserted into the slot as far as it can go and then quickly removed. The strip will then calibrate the analyzer for the current lot of test strips based on the information on the code strip. Calibration of the portable lactate analyzer was done on each testing day. Measuring time is approximately 60 seconds from whole blood samples ranging 0.8 mmol/l – 22 mmol/l.

Exercise testing consisted of an anaerobic power test, the Wingate Power Test, on a MonarkTM cycle ergometer. The Wingate Power Test involved pedaling on a cycle ergometer at a maximal level of exertion for 30 seconds. The test is designed to measure anaerobic power output for primarily the lower extremity muscle groups (i.e., quadriceps). All subjects were encouraged to perform the testing portion of the

experiment at their maximal physical limits. Heart rate was monitored throughout the test. The following three measurements were obtained from the Wingate Test:

- a. Peak 5-second power (absolute and relative): the highest 5-second power score during the entire 30-second test reflects the ability of the muscle to break down ATP from primarily two sources: stored ATP and stored creatine phosphate.
- b. Mean 30-second power (absolute and relative): the average power output of the entire test, reflects ATP production via anaerobic glycolysis.
- c. Fatigue index: the difference between the highest 5-second power score and the lowest 5-second power score, divided by the highest 5-second power score; a high score (>45%) indicates relatively low muscular endurance, a low score (<30%) indicates the ability of the musculature to resist fatigue.

Safety and Confidentiality

Because this study involved exertions at maximal levels and invasive procedures, each subject encountered possible risks. At high levels of exertion, subjects were likely to experience discomfort and pain associated with fatigue. In addition, blood lactate collection requires lancing a finger for blood samples. There were a total of five finger sticks, using the index, middle and ring fingers. This was likely to cause discomfort for subjects and improper administration of this protocol could have led to infection. Subjects were instructed to seek medical attention from the athletic training staff and team physician if first-aid was needed post testing session.

The following precautionary measures were taken to ensure the safety of each subject and investigators:

- a. Review of medical history and fitness readiness questionnaires of the subjects.
- b. Subjects read and signed the attached informed consent letter.

- c. Testing procedures were completely explained prior to testing.
- d. Testing could be terminated by the subject or the investigator at any time during the experiment.
- e. The testing was monitored by an investigator with CPR certification and certification by the American College of Sports Medicine as an Exercise Test Technologist.
- f. Emergency numbers were posted at the testing site.
- g. Biohazard precautions included: all investigators were required to wear latex gloves during testing and changed for each subject, sterile lancets were utilized for finger sticks and utilized only once then disposed, alcohol prep of area to be sampled, disposal of all sharp and biohazard materials in proper containers.
- h. Subjects were debriefed on their results and given proper procedures for recovery and adequate hydration.
- i. All information and material pertaining to each subject was coded and locked in a file for confidentiality.
- j. After the completion of the study each subject was able to gain access to their own results (Appendix D).

Procedures

Prior to both testing sessions, subjects were familiarized with the entire exercise and recovery protocols by instruction and demonstration. Prior to testing each subject warmed up on the bicycle ergometer for 3-5 minutes at an intensity to achieve a heart rate between 150-160 bpm. The cycling was interspersed with 4-5 seconds of all out sprints to help the subject get a feel for the actual test.

After the warm up, the subject rested for at least 2 minutes but not for more than 5 minutes.

On cue, the subject was instructed to pedal as fast as possible. Simultaneously, the ergometer resistance was increased to a predetermined resistance within 1-3 seconds. The resistance is equal to 0.075 times body mass (kg). Revolutions and power data were both calculated with the SMI OptoSensor 2000TM and SMI PowerTM software (Jacobs, Johnson, Mahoney, Carter & Somarriba, 2004; Jacobs, Mahoney & Johnson, 2003).

Each subject was required to perform two different Wingate Tests (one active recovery and one passive recovery) with identical resistance loads 3-7 days apart, and at a similar time of the day. The method of recovery following the first exercise test was determined randomly. The second exercise test concluded with the opposing recovery method. For the passive recovery test, each subject remained seated on the cycle ergometer after the exercise portion of testing. For the active recovery test, each subject rode the cycle ergometer at 30% testing resistance at 60 rpm. Each recovery phase lasted 20 minutes. Blood lactate was measured four times for each recovery phase (at 0, 3, 5 and 20 minutes post exercise) (Margarucci & Mansouri, 2000). Each individual test took approximately 30-35 minutes. Finally, each subject was debriefed, given information pertaining to results, recovery, hydration, etc.

Calculations

The following calculations were utilized in this study.

Percent lactate recovery:

$$\% \text{ Recovery} = [(La_{\text{peak}} - La_{\text{final}}) / (La_{\text{peak}} - La_{\text{rest}})] \times 100$$

Lactate rate of removal (LRR):

$$\text{LRR} = (La_{\text{peak}} - La_{\text{final}}) / La_{\text{peak}}$$

Absolute peak 5-second power (APP):

$$\text{APP (watts)} = \text{load (kg)} \times \text{peak revolutions} \times 11.765$$

Relative peak 5-second power (RPP):

$$\text{RPP (watts/kg)} = \text{APP} / \text{body wt (kg)}$$

Absolute mean 30-second power (AMP):

$$\text{AMP (watts)} = \text{load (kg)} \times \text{average revolutions} \times 11.765$$

Relative mean 30-second power (RMP):

$$\text{RMP (watts/kg)} = \text{AMP} / \text{body wt (kg)}$$

Fatigue index (FI):

$$\text{FI (\%)} = (\text{highest peak 5-second power} - \text{lowest peak 5-second power}) / \text{highest peak 5-second power}$$

Statistical Analysis

All statistical calculations were performed using SPSS[®] and Microsoft[®] Excel. Two t-tests were performed to compare values from the two test days, active and passive. To examine relations between lactate and power variables, data was evaluated using the Pearson Product Moment Correlation Coefficient. In order to examine lactate characteristics over time (during the recovery phases), a factorial, 2x4 (recovery type x time) ANOVA was performed, adjusted using the Greenhouse-Geisser correction. The level of statistical significance was set at $p < 0.05$.

CHAPTER FOUR

Results

Subject Demographics

Nine female athletes were initially recruited for this study; however one subject was unable to complete the active recovery portion of the experiment. This subject complained of dizziness after the Wingate Test and was immediately removed from the protocol. This subject's heart rate and blood pressure were immediately monitored and were determined to be within "normal" levels. Her immediate post exercise heart rate was 73 bpm and her blood pressure was 110/58 mmHg. The team athletic trainer and team physician were notified of this of subject's vitals and complaints. The subject was instructed to see the team physician immediately as a precautionary measure. This subject was eliminated from all statistical calculations, reducing the usable sample size to eight subjects. Table 1 presents demographic data for subjects.

TABLE 1

Physical Characteristics of Subjects

Demographic Data	Mean Values (SD)
Age (yr)	19.5 (0.5)
Body mass (kg)	65.2 (9.5)
Height (in)	69.4 (1.3)

Pre-recovery Comparisons

For the analysis of lactate removal to be valid, certain measurements prior to the recovery phases must show no differences. Resting lactate measured before the exercise test showed no significant differences comparing subjects' two test days (active and passive recovery) $t(7) = .355, p = .733$ (Figure 1). Relative peak power and relative mean power during the Wingate tests for both test portions were also not statistically different, $t(7) = .401, p = .700$ and $t(7) = .410, p = .694$, respectively, as shown in Figure 2. Fatigue Index, for the duration of each exercise session, revealed no significant differences, $t(7) = -1.469, p = .185$, (see Figure 3). Finally, there was not a statistically significant difference for immediate post-test lactate comparing the two sessions, $t(7) = 1.452, p = .190$, (Figure 1).

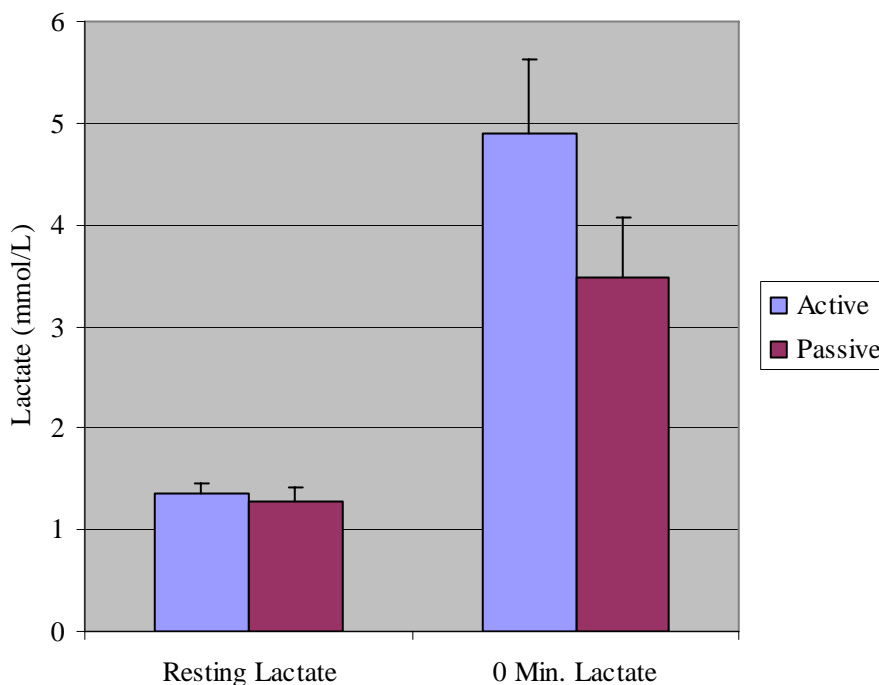


Figure 1 – Mean values for resting lactate and immediate post-test lactate for active and passive recovery.

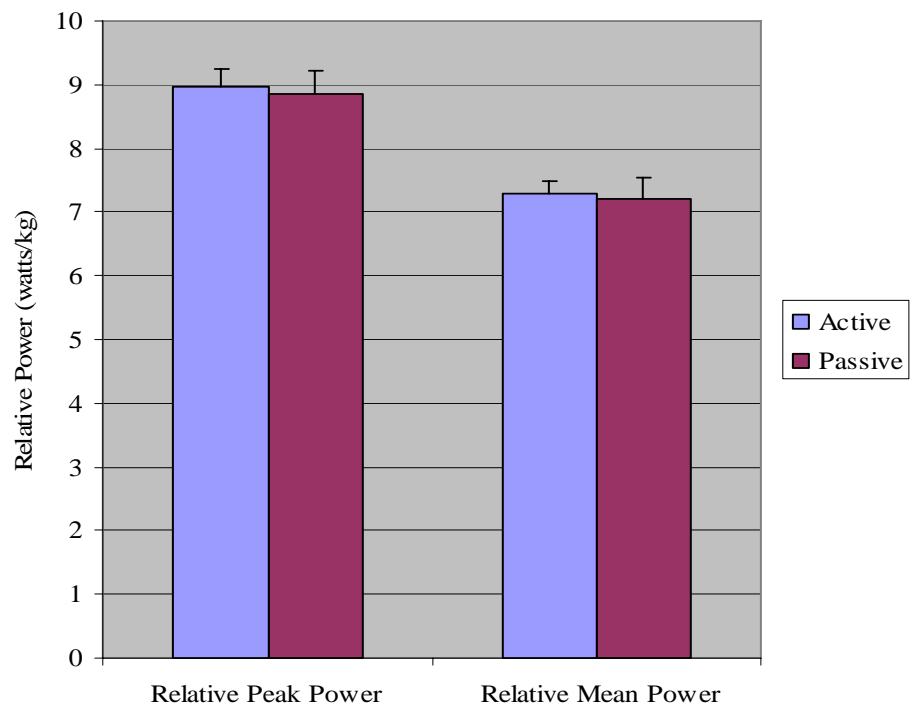


Figure 2 – Mean values for relative peak and mean power for active and passive recovery tests.

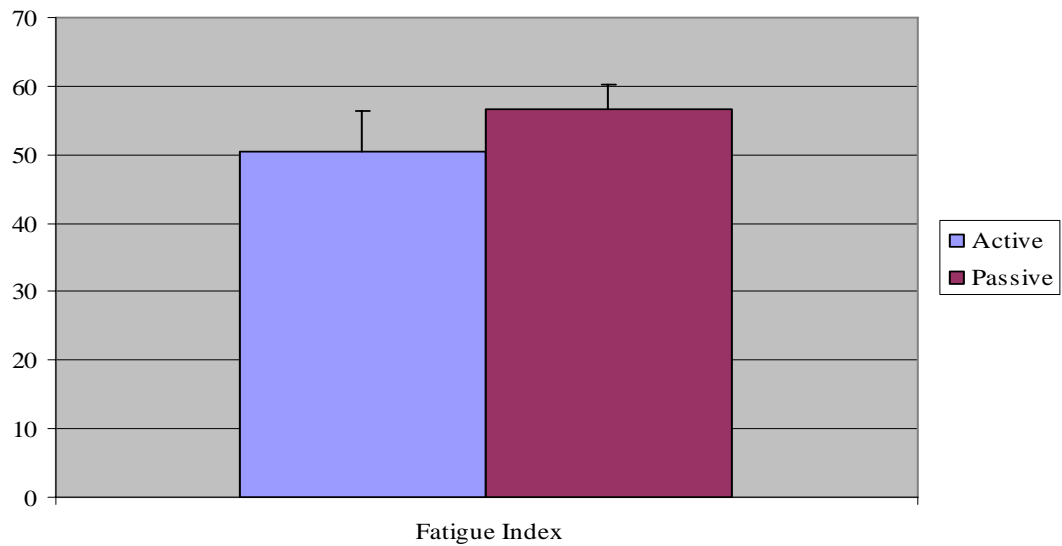


Figure 3 – Mean values for fatigue index for active and passive recovery.

Timeline Recovery Characteristics

Examination of lactate values over time were analyzed using a 2 x 4 (Recovery Type x Time) ANOVA with repeated measures on both factors. Due to the potential for violating the assumption of sphericity, probability levels and degrees of freedom were adjusted using the Greenhouse-Geisser epsilon correction. There was a significant main effect for time, $F(1.41, 9.85) = 12.49, p = .003, \eta^2 = .64$, but not for recovery type, $F(7, 1) = .045, p = .839, \eta^2 = .01$. Also, the recovery type by time interaction was not significant, $F(2.09, 14.60) = 2.62, p = .105, \eta^2 = .27$. Follow-up pairwise analysis using the least significant difference procedure was used to examine time difference, and showed significant differences between the lactate at 0 min and at 3 and 5 minutes, $p = .014$ and $p = .034$, respectively. Also, there were significant differences between the lactate at 20 minutes and at 3 and 5 minutes, $p = .000$ and $p = .002$, respectively (Figure 4). All other comparisons were not significant (see Figure 4). Table 2 presents means values for lactate recovery at 0, 3, 5, and 20 mins.

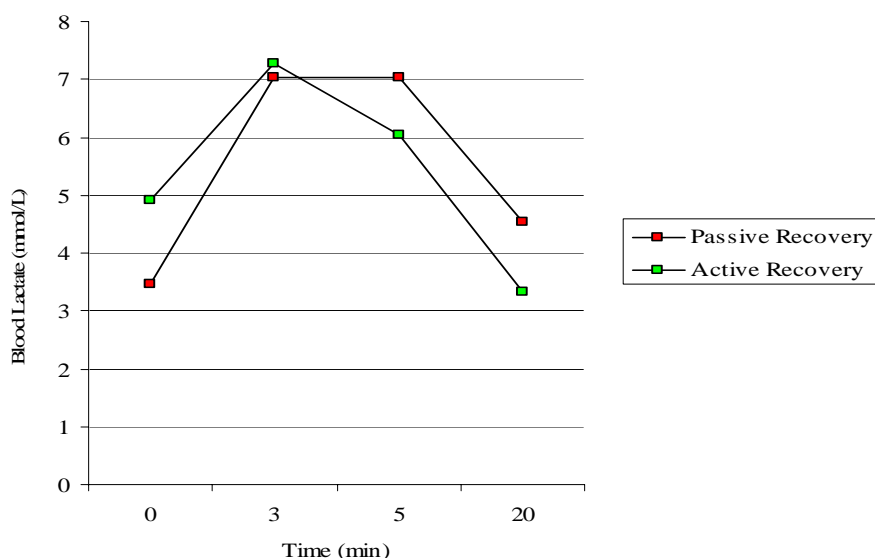


Figure 4 – Mean lactate by time for active recovery.

TABLE 2

Comparison of Mean Lactate Values for Timeline Recovery

Time	Active Recovery (n=8) Mean (SD) (mmol/L)	Passive Recovery (n=8) Mean (SD) (mmol/L)
0 minutes	4.9 (2.1)	3.5 (1.7)
3 minutes	7.3 (3.9)	7.0 (2.5)
5 minutes	6.0 (2.6)	7.0 (3.3)
20 minutes	3.3 (1.5)	4.5 (1.9)

Removal Comparisons

All but one of the subjects exhibited a decline in lactate during the passive recovery session. For this subject, peak lactate was recorded at 20 min post-exercise and for all other subjects peak lactate was obtained at either 3 or 5 minutes during recovery. The percent of lactate recovery between active and passive recovery was significantly different, $t(7) = 2.733$, $p = .029$ (see Figure 5). Further, comparing active and passive recovery conditions, there was a significant difference for rate of removal, $t(7) = 3.005$, $p = .020$ (see Figure 6).

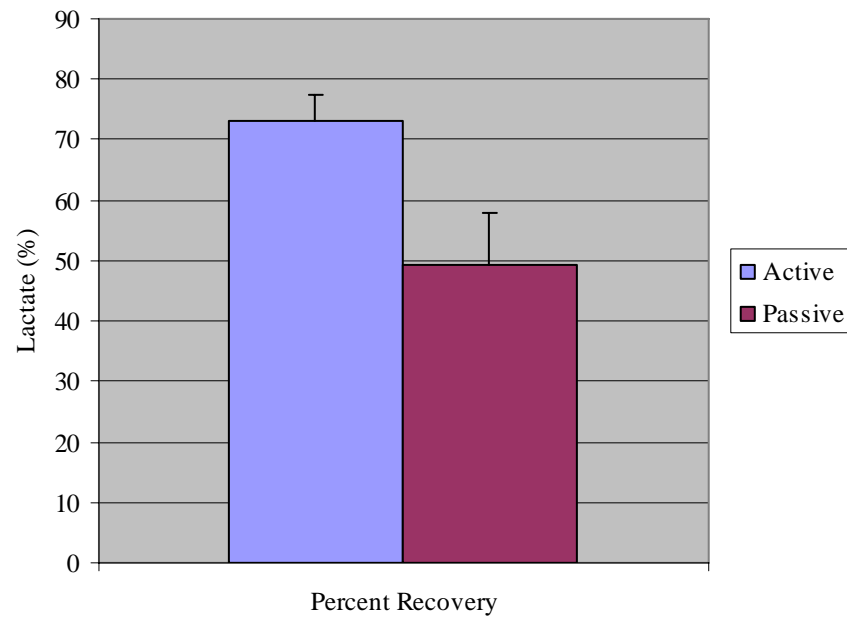


Figure 5 – Mean values for percent recovery for active and passive recovery.

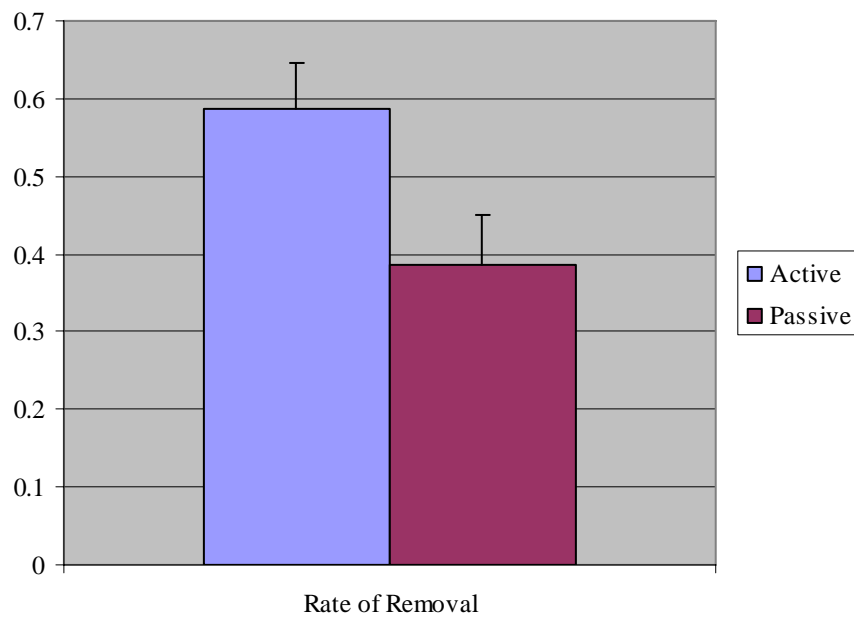


Figure 6 – Mean values for rate of removal for active and passive recovery. (Significant difference between active and passive recovery for rate of removal $t(7) = 3.005$, $p = .020$).

Power and Lactate Correlations

Correlations between passive recovery peak lactate and either absolute or relative peak power from the preceding Wingate Test did not reach statistical significance (Table 3). The correlation between passive recovery peak lactate and fatigue index was likewise not significant (Table 3). Also, correlations between active recovery percent of lactate removal and both absolute and relative peak power were not statistically significant (Table 3, Figure 7). The correlations between active recovery rate of lactate removal and both absolute and relative peak power were not statistically significant. (Table 3, Figure 8). Finally, correlations between either active recovery percent of removal or active recovery rate of removal and fatigue index from the preceding Wingate Test were not significant (Table 3).

TABLE 3

Correlations between Power and Fatigue Index and Lactate Values

Lactate Measure	Statistical Measurement	Absolute Peak Power	Relative Peak Power	Fatigue Index
Passive Rec. Peak Lactate	Pearson Correlation	.305	-.063	.034
	Sig. (2-tailed)	.462	.881	.937
	N	8	8	8
Active Rec. % Rec.	Pearson Correlation	-.652	-.646	.285
	Sig. (2-tailed)	.080	.083	.494
	N	8	8	8
Active Rec. Rate of Lactate Removal	Pearson Correlation	-.589	-.590	.143
	Sig. (2-tailed)	.124	.124	.736
	N	8	8	8

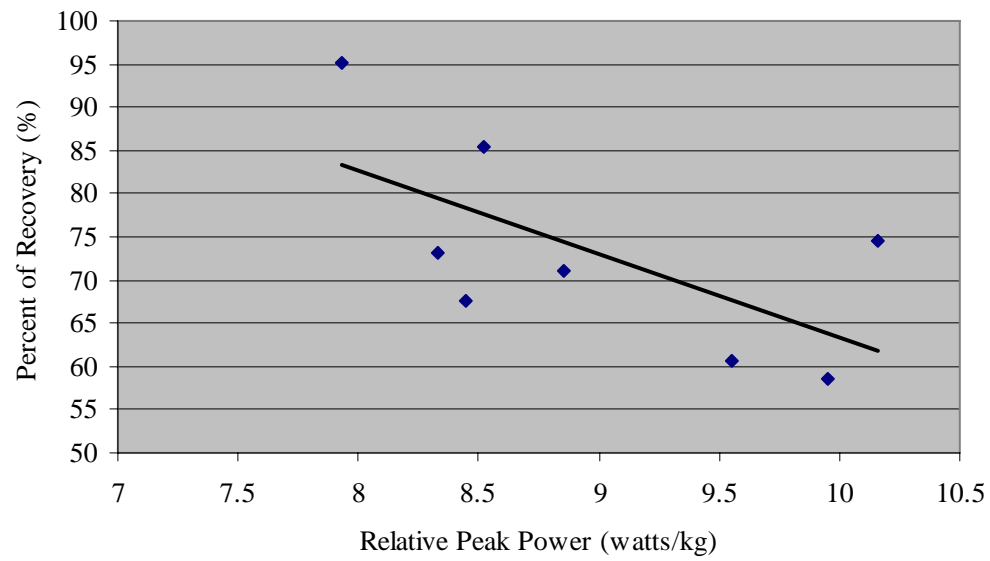


Figure 7 – Scatter plot depicting relative peak power vs. percent lactate recovery for active recovery test.

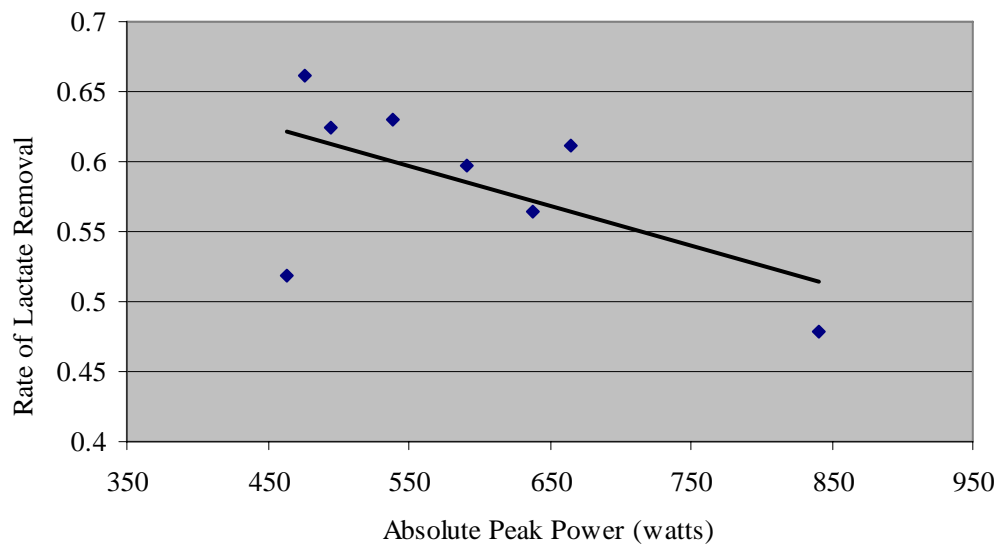


Figure 8 – Scatter plot depicting absolute peak power vs. rate of lactate removal for active recovery test.

CHAPTER FIVE

Discussion

It was important that data pertaining to both exercise sessions were identical to make accurate assumptions about recovery comparisons and correlations. This appeared to be the case as blood lactate measures prior to the Wingate Test were nearly identical and not statistically different. Three measures: relative peak power, relative mean power, and fatigue index were examined to determine if subjects exhibited different intensities when performing the two tests on different days. Results showed no differences between power or fatigue comparing the two test days. Finally, prior to the administration of the recovery protocols, blood lactate was again drawn to determine if any difference existed between the two test days. Based upon the present findings it is reasonable to assume that all subjects began the test with the same level of lactate in their blood, that each subject performed the same amount of work during each exercise session, and that after each exercise session that same amount of lactate was present.

The primary purpose of this study was to examine whether active and passive recovery leads to differential lactate removal in a female sample. Three different measures were employed to determine if there were any such differences: a) time characteristics of lactate values, b) rate of lactate removal and c) percent of lactate removal.

Examining the timeline characteristics of lactate, this study demonstrated that there was a significant effect for time, combining the two modes of recovery for 0, 3, 5, and 20 minutes. However, there was not a significant effect for recovery type, combining

all time data points for each recovery type. Furthermore, results of ANOVA did not detect a significant interaction as might be expected. These results deviate from the published literature on lactate recovery over time. In the 1994 study by Bangsbo et al., there was a two-fold higher net metabolism of lactate after the initial 3 minutes of active recovery. Furthermore, in the 1979 study by Weltman, Stamford & Fulco, the researchers found significant interactions between time and recovery conditions. At five and ten minutes, three modes of active recovery ($AR > AT$, $AR < AT$, $AR > AT + O_2$) resulted in a significantly lower blood lactate than passive recovery. Also, there were significant differences between 15 and 20 min for all recovery methods. Two explanations for the differences between this study and past studies could be the result of a lower number of involved subjects and resultantly poor statistical power (Froese & Houston, 1987; Jacobs et al., 1983; & Weinstein et al., 1998).

In contrast to the findings for lactate values over time, results showed a significant difference for the rate of lactate removal between active and passive recovery (.585 and .386 respectively). In this regard, results agree with previous literature. In a study by Belcastro & Bonen (1975), self selected recoveries were found to be a more effective method of active recovery at 32% VO_2 max produced an optimal rate of 3.2%/min lactate removal. Furthermore, as shown in a tracer study by Francoux et al. (1995), lactate rates increase from passive recovery to moderate exercise recovery and decrease from moderate exercise recovery to intense exercise recovery.

Like the results for rate of lactate removal, results showed a significant difference between active and passive recovery. Percent of lactate removal, while closely related to the rate of lactate removal, measures the amount of lactate decreased over the 20 min recovery periods. The active recovery resistance of 7.5% of each subject's body mass

produced a 73.3% removal of lactate. Passive recovery produced a 49.4% decrease in the amount of lactate produced over the 20 min period. These findings are similar to those of previous studies, all of which define active recovery at varying degrees below maximal exercise (Bonen & Belcastro, 1977; Bonen et al., 1979; Francini, Takito, Nakamura, Matsushigue, & Kiss, 2003; Gladden, 2000; & Watson & Hanley, 1986).

In accordance with the previous literature, this study demonstrates that active recovery is a more effective means of lactate removal, compared to passive recovery in female subjects (Bond et al., 1991; Gupta et al., 1996; Monedero & Donne, 2000; Watson & Hanley, 1986). While timeline recovery characteristics did not demonstrate the expected difference between the two recovery types, examining the data as a whole would support this conclusion. As shown in Figure 4, the active the active recovery session began with a mean lactate of 4.9 mol/L, peaked at 3 min with a lactate of 7.2 mol/L, and finished with 3.3 mol/L of lactate. Conversely, passive recovery began with 3.5 mol/L of lactate, peaked with 7.0 mol/L of lactate, and finished with 4.5 mol/L of lactate. Examining this data shows that active recovery began with a higher initial blood lactate and ended with a lower concluding blood lactate. Examining the large size effect associated with the recovery type by time interaction ($\eta_p^2=.27$), it is likely that low statistical power resulted in the failure to demonstrate the expected pattern of results.

A second purpose of this study was to examine the correlation between power output and lactate values. To determine correlations between power and lactate measures, correlations between the following variables were computed: a) peak power (absolute and relative) and passive recovery peak lactate, b) fatigue index and passive recovery peak lactate, c) peak power (absolute and relative) and active recovery, percent of lactate removal, d) fatigue index and active recovery, percent of lactate removal, e)

peak power (absolute and relative) and active recovery, rate of lactate removal, and f) fatigue index and active recovery, rate of lactate removal. While there was a negative association between active recovery, percent of removal and peak power, all correlations were determined to be not significant. Again, this is likely the result of low statistical power. These findings are inconsistent with the current literature on the relationship between power, fiber type and lactate dynamics. Performance factors (blood lactate, peak power and power decrease) during the Wingate test are positively correlated to percent of fast-twitch fibers (Bar-Or, 1987; Froese & Houston, 1987; Weinstein et al., 1998). Furthermore, in a study conducted by, Sahlin and Henriksson (1984), researchers found a positive correlation between fast-twitch muscle fibers and blood lactate. Positive correlations have been reported between running velocities during 100 to 400m sprints and peak lactate (Fujitsuka, Yamamoto, Ohkuwa, Santo, & Miyamura, 1982; Ohkuwa, Kato & Katsumata, 1984). In the Wingate study, by Froese and Houston (1987), researchers focused on the relationship between power measures, obtained during the test, lactate production, and muscle fiber composition. In this study researchers utilized a larger resistance setting for the Wingate test when compared to the present study. These researchers found significant correlation between power measures, post-test lactate concentration, and the percentage of fast-twitch fibers in their male subjects (n=12). However, the researchers did not find a significant correlation in their female subjects (n=18). The findings in this study along with information provided in the Froese and Houston study (1987) allude to a number of issues regarding women subjects. First, women tend to have a larger proportion of body fat and a lower muscle mass than men (Brooks et al., 1996). Furthermore, women subjects tend to have a slower rate of isometric force development than men (Bell & Jacobs, 1986; Komi & Karlsson, 1979).

In addition glycolytic enzymatic activity in female (n=38) subjects has been determined to be lower than their male (n=37) counterparts (Simoneau et al., 1985) for hexokinase, phosphofructokinase, LDH, and malate dehydrogenase. Finally, Jacobs et al. (1983), in a study of 15 male and 7 female participants, found that the female subjects produced significantly ($p=0.005$) lower levels of lactate compared to males, 47.4 and 73.9 mmol*kg respectively, after a 30-s Wingate Test.

Conditioning for tennis should focus on developing both the high energy phosphate system and the anaerobic glycolytic system (Baechle & Earle, 2000). Training for these systems increases the concentrations of myosin ATPase, creatine kinase, and myokinase (Baechle & Earle, 2000). Work rest ratios for tennis players have been suggested to be 2-4 s of rest for every second of work (Kovacs, 2004). The work rest ratio in this study design is 1:40 (work:rest) which corresponds to the high energy phosphate system (Baechle & Earle, 2000).

Conclusions

In conclusion, results show that active recovery is a more efficient means of removing blood lactate in collegiate female tennis player when compared to passive recovery. However, this study did not show a relationship between power indices and lactate production. The present findings, however, are limited by low statistical power resulting from a small sample size.

Recommendations for Further Study

The procedures found within this study have various applications in sports performance. For the coach and exercise physiologist, improvements must be made in the following areas to improve upon the current literature on athletic performance:

- Studies should further examine the differences between male and female subjects and the glycolytic pathway as it relates to fatigue.
- Attention should focus on the feasibility of incorporating recovery methods during athletic competition and practice.
- Relationships between lactate values and quantifiable athletic performance outcomes (i.e. sprint time) should be determined to allow training measures to be produced.

These suggestions for future research could provide valuable information to the coach and athlete who seek to monitor an athletic performance during practice and competition.

APPENDICES

APPENDIX A

Informed Consent

The Effects of Active and Passive Recovery on Blood Lactate Research Study:

Informed Consent

You are invited to participate in a study comparing percent (%) recovery through blood lactate analysis between active and passive recovery. I/we hope to attain information pertaining to physical functioning. You were selected as a possible participant in this study because of your level of fitness.

If you decide to participate, you will be asked to perform two tests on the bicycle ergometer. This test will measure your physiological responses (i.e., heart rate, lactic acid production, and power output) at maximal effort for 30 seconds, followed by 20 minutes of recovery.

Because you will be asked to work to a maximal level of exertion, some risks and discomforts are possible. At higher work intensities some discomfort may occur. However, this is a natural response and discomforts of the human body do occur during high work rates. Part of the test (lactic acid determination) will involve an invasive procedure to collect blood. This procedure is a simple finger stick before testing and during recovery. There will be a total of 10 finger sticks for each subject (5 per test). All sanitary and biohazard precautions will be taken to ensure a safe condition for you, as a subject. At higher work rates, possible risks involve heart arrhythmia. In a worse case scenario, the subject can lose consciousness and possibly die. The investigators will utilize pre-test questionnaires and during test administration, will take extreme measures to ensure the safety of each participant.

Any information obtained in this study in which you can be identified will remain confidential. The data obtained from this study will be presented at a professional meeting and/or published in a research journal. Information obtained in this study will be numerically coded to ensure confidentiality of each participant.

As a benefit of your participation in this study, you will gain valuable information about how your body reacts to maximal exertion and the level at which you recover from these work intensities.

Participation in this study is entirely voluntary. Your decision whether or not to participate will not affect your current or future relations with Baylor University. If you decide to participate, you may withdraw from the study at any time without affecting your status as a subject, or student.

If you have any questions or concerns about this study or any problems that result from participation, you may consult with Larry W. Coffey II or Dr. Frank B. Wyatt. Mr. Coffey can be reached at (254) 710-4466 and Dr. Wyatt can be reached at (254) 710-3504. Mail correspondence to Mr. Larry W. Coffey II, P.O. Box 97074 Department of Student Activities, Baylor University, Waco, TX 76798-7074, or Dr. Frank B. Wyatt, P.O. Box 97313, Department of HHP, Baylor University, Waco, TX 76798-7313.

If you have questions regarding your rights as a participant in this research, you may also contact the Baylor University Research Committee for Protection of Human Subjects in Research, Dr. Ben Pierce, Chair, Baylor University, Waco, TX 76798. Dr Pierce may also be reached at (254) 710-4288.

By signing this form I am acknowledging that I am 18 years or older. I have also read and understand this form. I have been given a copy of this form to keep for my own records. I have agreed to participate in this research.

Name (Printed)

Signature

Date

APPENDIX B

Fitness Questionnaire

Physical Activity Readiness
Questionnaire - PAR-Q
(revised 1994)

PAR – Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
<input type="checkbox"/>	<input type="checkbox"/>	1. Has your doctor ever said that you have a heart condition <u>and</u> that you should only do physical activity recommended by a doctor?
<input type="checkbox"/>	<input type="checkbox"/>	2. Do you feel pain in your chest when you do physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	3. In the past month, have you had chest pain when you were not doing physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
<input type="checkbox"/>	<input type="checkbox"/>	5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
<input type="checkbox"/>	<input type="checkbox"/>	7. Do you know of any other reason why you should not do physical activity?

**If
you
answered**

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want - as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active - begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal - this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively.

DELAY BECOMING MUCH MORE ACTIVE:

- If you are not feeling well because of temporary illness such as a cold or a fever - wait until you feel better; or
- If you are or may be pregnant - talk to your doctor before you start becoming more active.

Please note: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

You are encouraged to copy the PAR-Q but only if you use the entire form

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction.

NAME _____

SIGNATURE _____

SIGNATURE OF PARENT _____

or GUARDIAN (for participants under the age of majority)

DATE _____

WITNESS _____

continued on other side...

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APPENDIX C
Recovery Data Sheet

Active Recovery Data Sheet
Resting Data

Name: _____ Date: _____
 Body Mass (kg): _____ Height (in): _____
 Age (yrs): _____

Wingate Power Test Data

Resting Lactate (mM): _____
 Cycle ergometer resistance (kg): _____ (body mass x 0.075)
 Absolute Peak Power:(watts)_____
 Relative Peak Power:(watts/kg) _____
 Absolute Mean Power: (watts)_____
 Relative Mean Power: (watts/kg)_____
 Fatigue Index: _____

Recovery Data

Active Recovery Pedaling Resistance 150 rpms at 2 kgm
 Immediate Blood Lactate (mM): _____
 3-min Blood Lactate (mM): _____
 5-min Blood Lactate (mM): _____
 20-min Blood Lactate (mM): _____
 % Lactate Recovery: _____

APPENDIX D

Subject Raw Data

Subject 1

Body Mass (kg): _____59.42_____ Height (in): _____70_____

Age (yrs):_____19_____

Active Recovery Data

Resting Lactate (mM): _____1.4_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.5_____ (body mass x 0.075)

Absolute Peak Power:(watts)___495___

Relative Peak Power:(watts/kg) ___8.33___

Absolute Mean Power: (watts)___420___

Relative Mean Power: (watts/kg)___7.06___

Fatigue Index: ___63.8___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____6.3_____

3-min Blood Lactate (mM): _____9.6_____

5-min Blood Lactate (mM): _____4.7_____

20-min Blood Lactate (mM): _____3.6_____

% Lactate Recovery: ___73.17_____

Passive Recovery Data

Resting Lactate (mM): _____1.3_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.5_____ (body mass x 0.075)

Absolute Peak Power:(watts)___535___

Relative Peak Power:(watts/kg) ___9.0___

Absolute Mean Power: (watts)___443___

Relative Mean Power: (watts/kg)___7.45___

Fatigue Index: _____62.5___

Immediate Blood Lactate (mM): _____3.8_____

3-min Blood Lactate (mM): _____4.5_____

5-min Blood Lactate (mM): _____5.0_____

20-min Blood Lactate (mM): _____5.2_____

% Lactate Recovery: _____0_____

Subject 2

Body Mass (kg): _____52.3_____ Height (in): _____68_____

Age (yrs): _____20_____

Active Recovery Data

Resting Lactate (mM): _____1.4_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.0_____ (body mass x 0.075)

Absolute Peak Power:(watts)___463___

Relative Peak Power:(watts/kg) ___8.85___

Absolute Mean Power: (watts)___380___

Relative Mean Power: (watts/kg)___7.26___

Fatigue Index: ___30.6___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____2.2_____

3-min Blood Lactate (mM): _____5.2_____

5-min Blood Lactate (mM): _____4.1_____

20-min Blood Lactate (mM): _____2.5_____

% Lactate Recovery: ___71.05___

Passive Recovery Data

Resting Lactate (mM): _____1.9_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.0_____ (body mass x 0.075)

Absolute Peak Power:(watts)___439___

Relative Peak Power:(watts/kg) ___8.4___

Absolute Mean Power: (watts)___358___

Relative Mean Power: (watts/kg)___6.85___

Fatigue Index: ___50.1___

Immediate Blood Lactate (mM): _____0.8_____

3-min Blood Lactate (mM): _____4.9_____

5-min Blood Lactate (mM): _____4.3_____

20-min Blood Lactate (mM): _____3.3_____

% Lactate Recovery: _____53.3_____

Subject 3

Body Mass (kg): _____60_____ Height (in): _____68_____

Age (yrs): _____20_____

Active Recovery Data

Resting Lactate (mM): _____1.8_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.5_____ (body mass x 0.075)

Absolute Peak Power:(watts)___476___

Relative Peak Power:(watts/kg) ___7.93___

Absolute Mean Power: (watts)___416___

Relative Mean Power: (watts/kg)___6.94___

Fatigue Index: ___66.9___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____5.9_____

3-min Blood Lactate (mM): _____4.7_____

5-min Blood Lactate (mM): _____5.2_____

20-min Blood Lactate (mM): _____2.0_____

% Lactate Recovery: ___95.12___

Passive Recovery Data

Resting Lactate (mM): _____1.2_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.5_____ (body mass x 0.075)

Absolute Peak Power:(watts)___455___

Relative Peak Power:(watts/kg) ___7.59___

Absolute Mean Power: (watts)___392___

Relative Mean Power: (watts/kg)___6.53___

Fatigue Index: ___76.9___

Immediate Blood Lactate (mM): _____3.4_____

3-min Blood Lactate (mM): _____7.6_____

5-min Blood Lactate (mM): _____5.8_____

20-min Blood Lactate (mM): _____4.6_____

% Lactate Recovery: ___46.88___

Subject 4

Body Mass (kg): _____63.2_____ Height (in): _____69_____

Age (yrs): _____19_____

Active Recovery Data

Resting Lactate (mM): _____1.7_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.75_____ (body mass x 0.075)

Absolute Peak Power:(watts)___539___

Relative Peak Power:(watts/kg) ___8.52___

Absolute Mean Power: (watts)___441___

Relative Mean Power: (watts/kg)___6.98___

Fatigue Index: ___62___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____6.5_____

3-min Blood Lactate (mM): _____3.3_____

5-min Blood Lactate (mM): _____4.3_____

20-min Blood Lactate (mM): _____2.4_____

% Lactate Recovery: ___85.42_____

Passive Recovery Data

Resting Lactate (mM): _____1.0_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.75_____ (body mass x 0.075)

Absolute Peak Power:(watts)___584___

Relative Peak Power:(watts/kg) ___9.24___

Absolute Mean Power: (watts)___460___

Relative Mean Power: (watts/kg)___7.27___

Fatigue Index: ___60.5___

Immediate Blood Lactate (mM): _____1.2_____

3-min Blood Lactate (mM): _____5.3_____

5-min Blood Lactate (mM): _____5.9_____

20-min Blood Lactate (mM): _____2.2_____

% Lactate Recovery: ___88.1_____

Subject 5

Body Mass (kg): _____84.5_____Height (in): _____69_____

Age (yrs):_____19_____

Active Recovery Data

Resting Lactate (mM): _____1.3_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____5.25_____ (body mass x 0.075)

Absolute Peak Power:(watts)___841___

Relative Peak Power:(watts/kg) ___9.95___

Absolute Mean Power: (watts)___604___

Relative Mean Power: (watts/kg)___7.14___

Fatigue Index: ___69.6___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____3.9_____

3-min Blood Lactate (mM): _____7.1_____

5-min Blood Lactate (mM): _____4.4_____

20-min Blood Lactate (mM): _____3.7_____

% Lactate Recovery: ___58.62_____

Passive Recovery Data

Resting Lactate (mM): _____0.8_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____5.25_____ (body mass x 0.075)

Absolute Peak Power:(watts)___860___

Relative Peak Power:(watts/kg) ___10.17___

Absolute Mean Power: (watts)___638___

Relative Mean Power: (watts/kg)___7.55___

Fatigue Index: ___52.6___

Immediate Blood Lactate (mM): _____5.0_____

3-min Blood Lactate (mM): _____10.2_____

5-min Blood Lactate (mM): _____10.4_____

20-min Blood Lactate (mM): _____6.5_____

% Lactate Recovery: ___40.63_____

Subject 6

Body Mass (kg): _____70_____ Height (in): _____70_____

Age (yrs): _____20_____

Active Recovery Data

Resting Lactate (mM): _____0.9_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____5.25_____ (body mass x 0.075)

Absolute Peak Power:(watts)___591___

Relative Peak Power:(watts/kg) ___8.45___

Absolute Mean Power: (watts)___523___

Relative Mean Power: (watts/kg)___7.47___

Fatigue Index: ___35.2___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____7.7_____

3-min Blood Lactate (mM): _____7.3_____

5-min Blood Lactate (mM): _____7.1_____

20-min Blood Lactate (mM): _____3.1_____

% Lactate Recovery: ___67.65___

Passive Recovery Data

Resting Lactate (mM): _____1.1_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____5.25_____ (body mass x 0.075)

Absolute Peak Power:(watts)___581___

Relative Peak Power:(watts/kg) ___8.30___

Absolute Mean Power: (watts)___481___

Relative Mean Power: (watts/kg)___6.87___

Fatigue Index: ___48.1___

Immediate Blood Lactate (mM): _____3.4_____

3-min Blood Lactate (mM): _____5.2_____

5-min Blood Lactate (mM): _____4.5_____

20-min Blood Lactate (mM): _____2.7_____

% Lactate Recovery: _____60.98_____

Subject 7

Body Mass (kg): _____65.45_____ Height (in): _____69_____

Age (yrs): _____19_____

Active Recovery Data

Resting Lactate (mM): _____1.2_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.5_____ (body mass x 0.075)

Absolute Peak Power:(watts)___665___

Relative Peak Power:(watts/kg) ___10.16___

Absolute Mean Power: (watts)___559___

Relative Mean Power: (watts/kg)___8.55___

Fatigue Index: ___31.5___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____2.1_____

3-min Blood Lactate (mM): _____5.7_____

5-min Blood Lactate (mM): _____6.7_____

20-min Blood Lactate (mM): _____2.6_____

% Lactate Recovery: ___74.55___

Passive Recovery Data

Resting Lactate (mM): _____1.3_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.5_____ (body mass x 0.075)

Absolute Peak Power:(watts)___676___

Relative Peak Power:(watts/kg) ___10.32___

Absolute Mean Power: (watts)___595___

Relative Mean Power: (watts/kg)___9.08___

Fatigue Index: ___45.9___

Immediate Blood Lactate (mM): _____4.8_____

3-min Blood Lactate (mM): _____7.3_____

5-min Blood Lactate (mM): _____6.6_____

20-min Blood Lactate (mM): _____4.0_____

% Lactate Recovery: _____55_____

Subject 8

Body Mass (kg): _____66.82_____ Height (in): _____70_____

Age (yrs): _____20_____

Active Recovery Data

Resting Lactate (mM): _____1.1_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____5.0_____ (body mass x 0.075)

Absolute Peak Power:(watts)___638___

Relative Peak Power:(watts/kg) ___9.55___

Absolute Mean Power: (watts)___466___

Relative Mean Power: (watts/kg)___6.97___

Fatigue Index: ___43.7___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____4.6_____

3-min Blood Lactate (mM): _____15.6_____

5-min Blood Lactate (mM): _____11.8_____

20-min Blood Lactate (mM): _____6.8_____

% Lactate Recovery: ___60.7___

Passive Recovery Data

Resting Lactate (mM): _____1.7_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____5.0_____ (body mass x 0.075)

Absolute Peak Power:(watts)___524___

Relative Peak Power:(watts/kg) ___7.84___

Absolute Mean Power: (watts)___411___

Relative Mean Power: (watts/kg)___6.15___

Fatigue Index: ___56.3___

Immediate Blood Lactate (mM): _____5.4_____

3-min Blood Lactate (mM): _____11.2_____

5-min Blood Lactate (mM): _____13.7_____

20-min Blood Lactate (mM): _____7.7_____

% Lactate Recovery: ___50___

Subject 9

Body Mass (kg): _____52.7_____Height (in): _____66_____

Age (yrs):_____18_____

Active Recovery Data

Resting Lactate (mM): _____1.7_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.0_____ (body mass x 0.075)

Absolute Peak Power:(watts)___568___

Relative Peak Power:(watts/kg) ___10.77___

Absolute Mean Power: (watts)___438___

Relative Mean Power: (watts/kg)___8.31___

Fatigue Index: ___59.9___

Pedaling Resistance 150 rpms at 2 kgm

Immediate Blood Lactate (mM): _____4.5_____

3-min Blood Lactate (mM): _____

Subject was unable to complete the active recovery test

5-min Blood Lactate (mM): _____

Post test HR: 73bpm BP: 110/58

20-min Blood Lactate (mM): _____4.9_____

Athletic Trainer was notified

% Lactate Recovery: ___73.17_____

Passive Recovery Data

Resting Lactate (mM): _____1.5_____

Wingate Power Test Data

Cycle ergometer resistance (kg): _____4.0_____ (body mass x 0.075)

Absolute Peak Power:(watts)___525___

Relative Peak Power:(watts/kg) ___9.98___

Absolute Mean Power: (watts)___418___

Relative Mean Power: (watts/kg)___7.95___

Fatigue Index: ___45.6___

Immediate Blood Lactate (mM): _____6.9_____

3-min Blood Lactate (mM): _____10.7_____

5-min Blood Lactate (mM): _____11.2_____

20-min Blood Lactate (mM): _____8.5_____

% Lactate Recovery: ___27.84_____

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