THE EFFECT OF RECOVERY STRATEGIES ON HIGH-INTENSITY EXERCISE PERFORMANCE AND LACTATE CLEARANCE

by

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B.H.K., The University of British Columbia, 2004

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in

The Faculty of Graduate Studies

(Human Kinetics)

THE UNIVERSITY OF BRITISH COLUMBIA

(Vancouver)

March 2008

Abstract

PURPOSE: To compare the effects of recovery intensity on performance of a bicycle sprint task and blood La^- clearance. **METHODS**: On three separate days twelve trained male subjects (27.4 ± 3.9 yrs) performed three supramaximal exercise (SE) bouts at 120% of maximum aerobic power (MAP) for 60% of the time to exhaustion (TTE). Bouts were separated by 5 min of passive recovery (PR), active recovery (AR) or combined active recovery (CAR). The third bout was followed by a 14 min recovery. Recovery intensities were: PR (rest), AR at 50% of the workload difference between the individual anaerobic threshold (IAT) and the individual ventilatory threshold (IVT) below the IVT ($IVT_{-50\%\Delta T}$), or CAR at the IAT workload for 5 min and at the $IVT_{-50\%\Delta T}$ workload for 9 min. Five 10 s sprints were performed 2 min post-recovery. Blood lactate (La^-) concentration, power parameters (Peak Power (PP), Mean Power (MP), Fatigue Index (FI), and Total Work (TW)), Heart Rate (HR), and Oxygen Uptake ($\dot{V}O_2$) were compared using repeated-measures ANOVA. Pairwise comparisons and dependent T-tests were performed to analyze differences. **RESULTS**: Mean La^- values for AR and CAR were lower than PR (9.7 ± 3.5, 9.5 ± 3.5, 11.7 ± 3.6, respectively, $p \leq 0.05$). La^- was significantly lower with CAR versus PR at the 3rd, 6th, 9th, and 14th min of recovery ($p \leq 0.05$). AR versus PR La^- was lower at the 6th, 9th, and 14th min of recovery ($p \leq 0.05$). Mean MP was greater in the AR group compared to the PR group (800.1 ± 114.5 vs 782.2 ± 111.7 W, $p \leq 0.05$). TW during AR was greater than PR ($p \leq 0.05$) but not CAR ($p > 0.05$, 40003.3 ± 5110.2, 39108.3 ± 4852.9, 39335.8 ± 5022.6 J, respectively). **CONCLUSIONS**: AR and CAR both demonstrated improved La^- clearance when compared to PR, but differences in La^- clearance did not determine performance on the sprint task. AR resulted in more TW than PR and greater maintenance of power over the sprints.

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CHAPTER I: INTRODUCTION

1.1 Introduction

Historically lactate[a] (La^-) and its accumulation during high-intensity exercise has been identified as a causative factor in fatigue development (Hill & Kupalov, 1929; Karlsson, Bonde-Petersen, Henriksson, & Knuttgen, 1975; Klausen, Knuttgen, & Forster, 1972; Stamford, Weltman, Moffatt, & Sady, 1981; Yates, Gladden, & Cresanta, 1983). More recently, the role of La^- as a causative agent in fatigue development has been questioned, but results remain inconclusive (Fitts, 2003; Gladden, 2004). It appears that the relationship of La^- accumulation to fatigue is part of a complex interactive process. Nonetheless, the association between La^- accumulation and the onset of fatigue during high-intensity exercise remains irrefutable. Though, it must be stressed that the chronological associations of the two events does not necessarily indicate causation. It has been suggested that an increased rate of La^- clearance may lead to faster recovery and/or postpone fatigue during repetitive exercise (Gisolfi, Robinson, & Turrell, 1966; Lindinger, McKelvie, & Heigenhauser, 1995). This has large implications for performance athletics, where the ability to recover quickly for subsequent high-intensity exercise bouts is crucial to success. This is especially true for sports where athletes may compete more than once in a day (e.g. track, cycling, swimming, ref. Dodd, Powers, Callender, & Brooks, 1984) or perform repetitive high-intensity bouts within one competition (e.g. hockey, football, soccer, basketball). The unequivocal findings that active recovery (AR, i.e. low-intensity aerobic exercise performed after high-intensity exercise) increases the rate of La^- clearance (Ahmaidi et al., 1996; Belcastro & Bonen, 1975; Davies, Knibbs, & Musgrove, 1970; Dupont, Moalla, Guinhouya, Ahmaidi, & Berthoin, 2004; Gupta, Goswami, Sadhukhan, & Mathur, 1996; Hermansen & Stensvold, 1972; Jervell, 1928; Newman, Dill, Edwards, & Webster, 1937; Rämmal & Ström, 1949; Siebers & McMurray, 1981; Stamford et al., 1981; Taoutaou et al., 1996; Weltman, Stamford, & Fulco, 1979), has lead to the belief that AR is beneficial to subsequent exercise performance. However, studies investigating the effect of active versus passive recovery (PR, i.e. no activity) on subsequent performance have been inconclusive, with some investigations showing benefits (Ahmaidi et al., 1996; Signorile, Ingalls, & Tremblay, 1993; Spierer, Goldsmith, Baran, Hryniewicz, & Katz, 2004; Thiriet et al., 1993) and others not (Dupont et al., 2004; Franchini, Yuri Takito, Yuzo Nakamura, Ayumi Matsushigue, & Peduti Dal'Molin Kiss, 2003; Siebers & McMurray, 1981; Weltman & Regan, 1983). Furthermore, the

[a] The term La^- will be used rather than lactic acid throughout this paper since at a physiological muscular pH range (~6.20-7.00) lactic acid is more than 99% dissociated to La^- and a proton due to its pKa value (pH=3.87, ref. Gladden, 2004; Robergs, Ghiasvand, & Parker, 2004)

optimal method(s) by which to perform AR remains uncertain since previous research has used a wide array of protocols. It has been suggested that recovery (and exercise) intensities should be expressed relative to individual thresholds rather than maximum oxygen consumption ($\dot{V}O_{2max}$, ref. Baldari, Videira, Madeira, Sergio, & Guidetti, 2004; McLellan & Skinner, 1982), since the metabolic outcome is dependent on thresholds. There is some evidence to support the use of a combined active recovery (CAR) of varying intensity to improve the clearance rate of La^- in comparison to AR at one intensity (Gmada et al., 2005). However, further research is needed to congeal this assertion. Additionally, there is currently no research available as to whether or not the increased rate of La^- clearance with a CAR translates to improved performance or what the effects of CAR are on performance independent of the effect on La^- clearance.

1.2 Rationale

In 1981, Stamford et al. suggested that there may not be one optimal exercise intensity at which to clear La^-. Rather than use a continuous submaximal constant load for AR (as had previously been used in most research protocols), they suggested using a progressively decreasing load starting above the anaerobic threshold (AT) and finishing below the AT. The author is aware of two studies that have used this suggestion and employed a CAR. Dodd et al. (1984) compared CAR (i.e. moderate-to-high-intensity followed by low-intensity exercise) to both passive and two traditional continuous constant load recoveries at moderate-to-high- and low-intensity, respectively. Their results showed that the CAR and low-intensity AR had the fastest La^- clearance rates, but that there were no statistically significant differences between those two recovery modalities. Recently Gmada et al. (2005) showed that CAR resulted in a faster La^- clearance rate when compared with constant load moderate-to-high-intensity, low-intensity active, and passive recovery (the effect was more pronounced in trained subjects compared to untrained). Together, these investigations demonstrate that CAR is at least equal to or better than AR with respect to blood La^- clearance rate. However, neither of these studies examined the effect of the recovery periods on subsequent performance.

1.3 Purpose

The purpose of this investigation was two-fold: to examine the effects of two active recoveries, relative to individual thresholds (i.e. CAR and AR), and PR in trained male cyclists on the subsequent performance of five 10 s bicycle sprints after a work bout of three supramaximal exercise (SE) intervals. Secondly, to examine the effects of the three recoveries on blood La^- clearance rate.

1.4 Significance of the Study

The results of investigations examining the effects of different recovery methods on performance have implications for the design and implementation of training programs and competition strategies. Specifically, this investigation provides insight into the use of different recovery intensities and their effects on subsequent performance of high-intensity exercise as well as blood La^- clearance. The results help to clarify what the effects of various recovery strategies are on performance and whether or not the use of active recoveries provide a benefit to high-intensity athletic performance and training.

1.5 Hypotheses

The following hypotheses were suggested:

a. **Blood Lactate Alternate Hypothesis (H_1)**: Δ CAR blood La^- > Δ AR blood La^- > Δ PR blood La^-

   The change in blood La^- in the CAR trial will be greater than the change in the AR trial which will in turn be greater than in the change PR trial. This hypothesis has been suggested because it has been previously shown that CAR is more effective than (Gmada et al., 2005) or as effective as (Dodd et al., 1984) AR at one sole intensity with respect to La^- clearance. It has been theorized that the faster La^- clearance rate with CAR is a result of the increased blood flow from the higher exercise intensity (Gmada et al., 2005). Previously it had been suggested that a CAR may be more effective at removing La^- since the decreasing exercise intensity would be commensurate with the decreasing La^- concentration (Stamford et al., 1981). The pairing of exercise intensity and La^- concentration is predicted to be beneficial because the rate La^- clearance is proportional to its concentration (Jorfeldt, 1970). Furthermore, it is hypothesized that AR will clear La^- faster than PR, since there is an abundance of literature that already supports this hypothesis (Baldari, Videira, Madeira, Sergio, & Guidetti, 2005; Franchini et al., 2003; McAinch et al., 2004; Spierer et al., 2004).

b. **Peak Power (PP) Output H_1**: $CAR_{PP} = AR_{PP} = PR_{PP}$

   No differences in PP output on the sprint task are hypothesized between recovery trials. This hypothesis has been suggested because it has previously been shown that AR modalities did not affect the development of PP in subsequent high-intensity exercise tasks with similar recovery durations (Ainsworth et al., 1993; Spierer et al., 2004; Weltman, Stamford, Moffatt, & Katch, 1977). Peak power (sometimes called anaerobic power) is mainly dependent on the phosphocreatine (PCr) system and free ATP, and

therefore is not expected to change significantly with the recovery durations in this investigation, since the prolonged 14 min recovery should provide ample time for replenishment of PCr levels.

c. **Mean Power (MP) Output H_1**: $CAR_{MP} > AR_{MP} > PR_{MP}$

The MP output on the sprint task will be statistically significantly greater in the CAR trial than the MP in the AR trial, which will be greater than the MP output in the PR trial. It is hypothesized that AR will result in a greater MP output than PR recovery since it has been previously shown that AR can improve subsequent performance with respect to MP output (Spierer et al., 2004; Thiriet et al., 1993). Combined active recovery is hypothesized to result in the greatest MP output since it has been shown that CAR can clear La^- faster than the other two recovery modes being tested (Gmada et al., 2005). High La^- and proton transport, as would occur during AR, has been suggested to prevent fatigue because of the beneficial effects of La^- anions in providing oxidizable substrate and gluconeogenic precursors (Thomas et al., 2005). The greater clearance of La^- is therefore not predicted to prevent fatigue by eliminating La^-, an agent in fatigue, but rather by utilizing La^- as fuel source to prevent fatigue. In the case of this investigation, it is being suggested that the fastest rates of La^- transport (i.e. clearance) will occur in the CAR trial and thus in this trial subjects will have the greatest resistance to fatigue. That is, their anaerobic capacity (or MP output) will be maintained.

d. **Fatigue Index (FI) H_1**: $PR_{FI} > AR_{FI} > CAR_{FI}$

Fatigue index will be greater in the PR trial than FI in the AR trial, which will be greater than FI in the CAR trial. Fatigue index is the percent power decrease over the course of a sprint and therefore will have the opposite results of MP.

e. **Total Work (TW) H_1**: $CAR_{TW} > AR_{TW} > PR_{TW}$

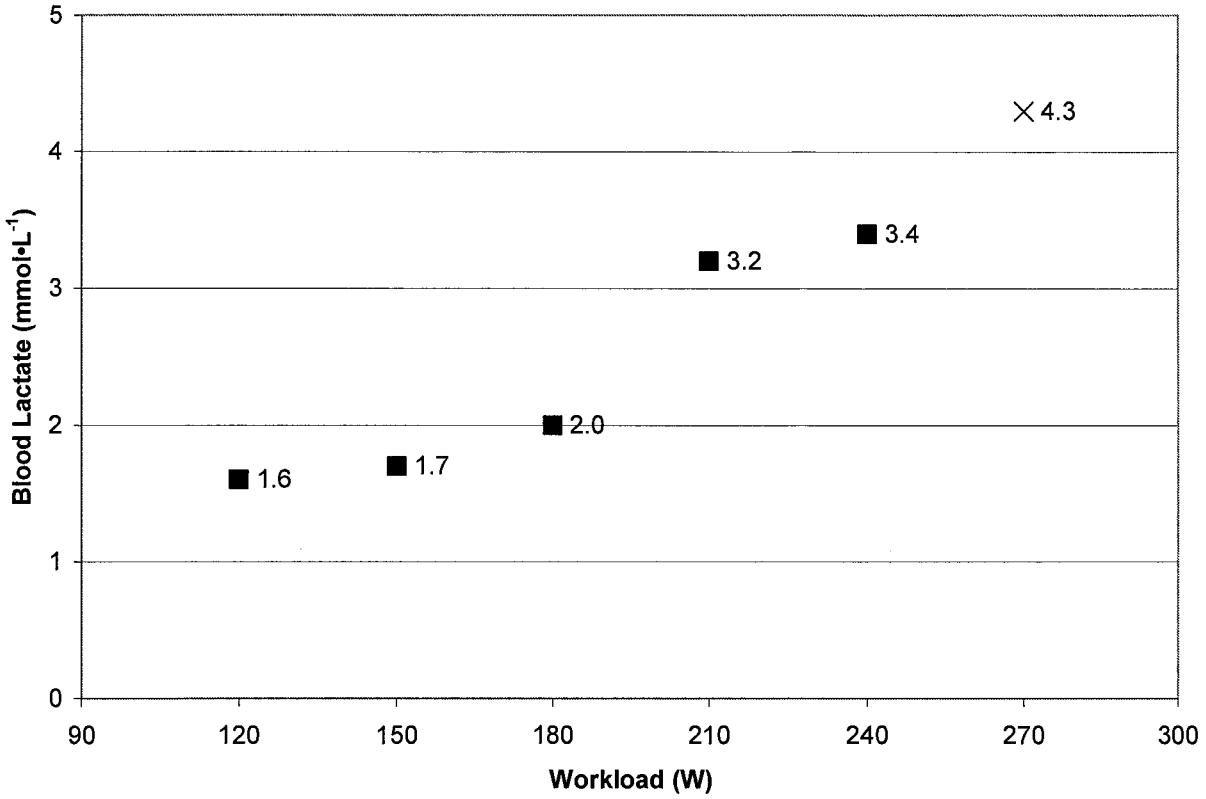
Total work on the sprint task will be statistically significantly greater in the CAR trial than TW in the AR trial, which will be greater than TW in the PR trial. This hypothesis is suggested because TW is the product of MP and time over the five sprints, and thus follows the above hypothesis of MP output.

1.6 Definitions

- Maximum Oxygen Consumption ($\dot{V}O_{2max}$) – a measure of cardiorespiratory fitness. $\dot{V}O_{2max}$ is highest amount of O_2 the body is able to consume and the product of maximal cardiac output ($L \cdot min^{-1}$) and arterial-venous difference (mL $O_2 \cdot L^{-1}$, ref. ACSM, 2006).

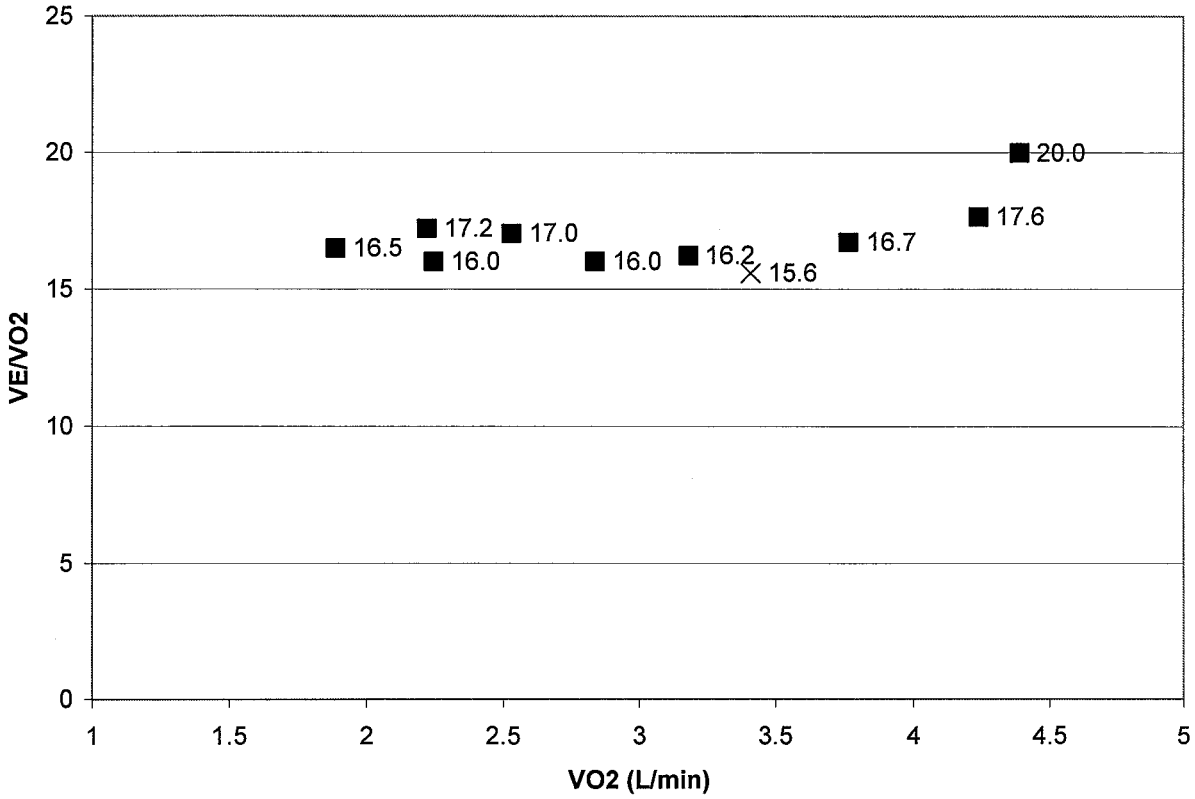
- Active Recovery (AR) – moderate- to low-intensity exercise performed after high-intensity exercise to promote a faster return to pre-exercise conditions.
- Combined Active Recovery (CAR) – moderate- to high-intensity exercise followed by moderate- to low-intensity exercise performed after high-intensity exercise to promote a faster return to pre-exercise conditions.
- Passive Recovery (PR) – resting post high-intensity exercise to promote a faster return to pre-exercise conditions.
- Individual Anaerobic Threshold (IAT) – a specific form of anaerobic threshold defined as the highest sustainable workload without an accumulation of La^- (i.e. maximal La^- steady state, ref. Baldari & Guidetti, 2000; Stegmann, Kindermann, & Schnabel, 1981). IAT is operationally defined as the workload corresponding to the second La^- increase of at least 0.5 $mmol \cdot L^{-1}$ from the previous value (Baldari & Guidetti, 2000, see Figure 1 for visual representation).
- Individual Ventilatory Threshold (IVT) – a specific form of ventilatory threshold also known as the point of optimum ventilatory efficiency (Hollmann, 2001). IVT is the lowest value of the ventilatory equivalent ($\dot{V}_E/\dot{V}O_2$), when $\dot{V}_E/\dot{V}O_2$ is plotted as a function of $\dot{V}O_2$ (Baldari & Guidetti, 2001, see Figure 2 for visual representation).

Figure 1. Example of Determination of Individual Anaerobic Threshold in Subject 11



IAT is the workload corresponding to the second La^- increase of at least 0.5 $mmol \cdot L^{-1}$ from the previous value and is represented by the ×

Figure 2. Example of Determination of Individual Ventilatory Threshold in Subject 2



IVT corresponds to the lowest $\dot{V}_E/\dot{V}O_2$ and is represented by the ×

1.7 Delimitations

This study will be delimited by:

a. A sample of university-aged (18-35) subjects from University of British Columbia students and others from the Vancouver area.

b. Setting the criteria for trained subjects as participating in competitive cycling with a heavy anaerobic component, PP and MP outputs greater than or equal to 11 and 9 $W \cdot kg^{-1}$ on the Wingate Anaerobic Test (WAnT), respectively, and having a $\dot{V}O_{2max}$ greater than 55 $mL \cdot kg^{-1} \cdot min^{-1}$.

c. A respiratory gas-sampling rate set at 20 s intervals.

d. The measurement of performance on the sprint task as a measure of anaerobic capacity.

e. The methodology used to determine IVT, IAT, and $\dot{V}O_{2max}$.

1.8 Limitations

This study will be limited by:

a. The data collection capabilities of the SensorMedics Vmax 29 series metabolic cart and the interlaced Data Acquisition System.
b. The individual's metabolic responses to the testing protocols.
c. The individual's effort during testing procedures (e.g. the individual's ability to perform maximally during the exercise tasks).
d. The ability to determine IAT and IVT from the data collected.

CHAPTER II: LITERATURE REVIEW

2.1 Introduction

In performance athletics the ability to maintain power output and ward off fatigue is essential to success. In activities that are intermittent in nature or between competitions that are shortly spaced apart (e.g. tournament setting) the capacity for an athlete to recover quickly is a determining factor in their performance. While the mechanism(s) of fatigue to date remain undetermined, the historical view has identified the accumulation of La^- as the causative factor (Hill & Kupalov, 1929). Over time this paradigm has shifted to La^- being an agent in the development of fatigue through its influence as an anion on the acid-base status of the muscle and blood (Lindinger et al., 1995; Stewart, 1981). The knowledge that the use of AR results in an increased clearance rate of blood La^- and the anecdotal reports of the advantageous use of AR in athletic training regimens has led to a belief that AR is beneficial to repeated performance (Gisolfi et al., 1966). Despite the common belief that AR is beneficial to subsequent performance, the research evidence is inconclusive with some research showing a benefit (Ahmaidi et al., 1996; Bogdanis et al., 1996b; Connolly et al., 2003; Corder et al., 2000; Signorile et al., 1993; Spierer et al., 2004; Thiriet et al., 1993) and some showing no benefit (Bond, Adams, Tearney, Gresham, & Ruff, 1991; Franchini et al., 2003; McAinch et al., 2004; Siebers & McMurray, 1981; Watson & Hanley, 1986; Weltman et al., 1979; Weltman & Regan, 1983). Furthermore, within the research that supports the use of AR to improve performance, the relationship of La^- clearance to performance is inconsistent (Bogdanis et al., 1996b; Connolly, Brennan, & Lauzon, 2003; Spierer et al., 2004; Thiriet et al., 1993). Interestingly, this lack of correlation between La^- clearance and improved performance may be explained by more recent evidence that is shifting the cause of fatigue away from La^- accumulation and the associated increase in acidity to an accumulation of inorganic phosphate (Westerblad, Allen, & Lannergren, 2002). Nonetheless, the mechanism(s) for the development of fatigue still remain unresolved and are most likely a result of a complex interaction of events related to the availability and accumulation of metabolites. Therefore the role of La^- as an agent in the development of muscular fatigue remains valid as the onset of fatigue coincides with its accumulation. From a practical perspective, regardless of the role of La^- in fatigue, the use of AR as a means to prevent decrements in performance in successive exercise remains a compelling research area particularly since AR may be beneficial for certain types of physical activity.

2.2 The New View on Lactate

Historically, the accumulation of La^- in the blood associated with high-intensity exercise has been viewed negatively and La^- has been labeled as a metabolic waste product that resulted from glycolysis in hypoxic conditions (Gladden, 2004). The initial concept of a relationship between La^- accumulation and hypoxia stemmed from research in the early part of the last century (Fletcher & Hopkins, 1907; Hill, Long, & Lupton, 1924). Gladden (2004) has identified the time frame from the 1930s to the 1970s as the dead-end waste product era of La^-. During this time La^- was believed to be a dead-end metabolite of glycolysis in hypoxic conditions (Wasserman, 1984). This paradigm has shifted greatly over the past few decades, as it has been repeatedly shown that the production of La^- is not necessarily a result of O_2 lack (e.g. Richardson, Noyszewski, Leigh, & Wagner, 1998). Currently, the perspective on La^- metabolism is very different in light of the advent of the La^- shuttle hypothesis (Brooks, 1985) and the widely held acceptance of the extracellular La^- shuttle[b]. Lactate is now recognized as a metabolic intermediate rather than an end, and a movable source of energy substrate that can be passed about the body for metabolism.

2.3 Lactate and its Relation to Fatigue During High-Intensity Exercise

During high-intensity exercise, anaerobic metabolic processes are heavily utilized to meet the energy demand. As the glycolytic production of ATP increases, the mitochondria's ability to aerobically oxidize pyruvate is exceeded (Spriet, Howlett, & Heigenhauser, 2000). This leads to an increased concentration of pyruvate and NADH, which are then converted to La^- and NAD by the near-equilibrium enzymatic reaction of La^- dehydrogenase (LDH, ref. Spriet et al., 2000). It is the processes that govern the production of pyruvate and NADH that predominantly control the production of La^- (Spriet et al., 2000). Blood La^- concentration is ultimately the result of the balance between production and clearance processes. With sufficiently high-intensities of exercise the balance between the production and clearance of La^- is shifted to disequilibrium and La^- begins to accumulate.

The knowledge that La^- accumulates in exercised muscle has a long history dating back to the work of Berzelius in 1807, who noted that hunted stags had elevated acid concentrations in their muscles (as cited in Needham, 1971). More recently other researchers noted the

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[b] Brooks has also argued for an intracellular La^- shuttle (Brooks, 1998) in addition to the original cell-to-cell (or extracellular) La^- shuttle, and has provided support for its existence (Brooks, Dubouchaud, Brown, Sicurello, & Butz, 1999). However, other researchers have failed to find supporting evidence to some of the central tenets of the hypothesis (Rasmussen, van Hall, & Rasmussen, 2002; Sahlin, Fernstrom, Svensson, & Tonkonogi, 2002; Yoshida et al., 2007). Consequently, the cell-to-cell La^- shuttle hypothesis is more or less unanimously accepted while the state of the intracellular La^- shuttle remains to be determined with future research

accumulation of La^- in working muscles and noted the effects of AR on its clearance (Jervell, 1928; Newman et al., 1937). In the early twentieth century, Hill and Kupalov (1929) proposed that lactic acid accumulation was the cause of muscular fatigue. This formed the initial framework for the conception that La^- accumulation was the cause of fatigue. This conception was maintained for years to come (Karlsson & Saltin, 1970), and is still very prevalent amongst most laypersons.

Currently, more researchers attribute the associated development of fatigue that arises with La^- accumulation to the associated decrease in pH (i.e. increase hydrogen ion concentration ($[H^+]$)) rather than to the increase in La^- anion itself (Fitts, 2003). Remember that the formation of lactic acid at a physiological pH results in its immediate dissociation into the La^- anion and a proton. Some researchers have misinterpreted the direct donation of this proton from lactic acid as the cause of the decrease in pH. This may be a result of attempting to simplify the explanation. However, the relationship of body fluid acid-base status is more complex than this. The re-introduction of the concepts of earlier researchers such as Henderson and van Slyke by Peter Stewart (Stewart, 1981) has helped to clarify this understanding (Lindinger, 2003). In this view acid-base status is determined by the independent effects of carbon dioxide (P_{CO2}), the concentration of weak acid buffers ($[A_{tot}]$, in plasma mainly the amino acids in plasma proteins), and the strong ion difference ([SID], i.e. the sum of the strong cations minus the sum of the strong anions, ref. Kowalchuk, Heigenhauser, Lindinger, Sutton, & Jones, 1988; Stewart, 1981). Lactate being a strong anion decreases pH by causing a decrease in the [SID]. The accumulation of La^- in the blood that occurs with high-intensity exercise has been shown to increase the plasma $[H^+]$, P_{CO2}, and osmolarity (Kowalchuk et al., 1988; Lindinger et al., 1995). The increased $[H^+]$ is believed to cause fatigue by: (1) inhibiting the glycolytic enzyme phosphofructokinase, (2) reduction of myosin crossbridge activation via competitive inhibition of Ca^{2+} binding to troponin C, and (3) inhibiting sarcoplasmic reticulum (SR) ATPase reducing Ca^{2+} re-uptake and subsequently Ca^{2+} release (Fitts, 2003).

2.3.1 Inorganic Phosphate as a Cause of Fatigue

Recently the role of increased $[H^+]$ in the development of fatigue has come into question as the initial studies that attributed H^+ accumulation to fatigue were not performed at physiological temperatures, and recent investigations at physiological temperatures do not support the role of H^+ in fatigue development (Gladden, 2004; Westerblad et al., 2002). Two landmark studies that have been integral in this shift are that of Bangsbo et al. (1996) and Nielsen et al. (2001). Bangsbo et al. (1996) showed that muscle acidity in humans during intense

exercise did not reduce glycogenolysis/glycolysis. While Nielsen et al. (2001) demonstrated that the reduced muscular force that normally developed with increased extracellular potassium concentration ($[K^+]_e$) could be reduced with induced acidosis and was accompanied by the regeneration of action potential development. In place, the cause of fatigue is attributed to the accumulation of inorganic phosphate (P_i) within the muscle. It is hypothesized that P_i causes muscular fatigue by: (1) reducing maximum crossbridge force, or (2) altering SR Ca^{2+} handling via direct action on SR Ca^{2+} release channels, inhibition of Ca^{2+} uptake, or formation of Ca^{2+}-P_i precipitate (for review see Westerblad et al., 2002). However, Gladden (2004) notes that the time course of fatigue development and the accumulation of P_i within the muscle do not coincide, since the majority of PCr is broken down in the initial seconds of high-intensity exercise. Similarly, Fitts (2003) states that it may be premature to dismiss the role of H^+ accumulation in fatigue development since studies have not completely elucidated the effects of a combined low pH, elevated P_i and reduced Ca^{2+} release. Recent research by Fitts and colleagues has provided support for the latter statement since they found that a combined reduction in myoplasmic Ca^{2+} and increased concentration of P_i act synergistically to reduce muscular force (Debold, Romatowski, & Fitts, 2005). It yet remains to determine the role of changes in acidity.

2.3.2 Other Mechanisms of Fatigue

It should be noted that there are previous reports that have linked the La^- anion itself to fatigue. In 1995, Hogan et al., using dog preparations showed that La^- infusion at a constant arterial pH (7.40) reduced muscle tension development by 15%. However, more recently the effects of the La^- anion on muscle contractility have been shown to be minimal (Posterino, Dutka, & Lamb, 2001). Still, other researchers subscribe to the theory of muscular fatigue induced via the accumulation of extracellular K^+ (Lindinger et al., 1995; Renaud, 2002), where La^- may have an indirect effect on fatigue development. Lindinger (1995) states that in order for proper muscle function to continue La^- must be removed, and intracellular K^+ concentration ($[K^+]_i$) must be maintained, as La^- clearance is essential for the recovery of $[H^+]$ and restoration of $[K^+]_i$ is necessary for both the recovery of $[H^+]$ and sarcolemmal and transverse tubule membrane potentials. In this model La^- influences acid-base status, which in turn regulates the membrane excitability. Similarly, Renaud (2002) proposes a model of fatigue in which $[K^+]_e$ is increased by the activation of ATP-sensitive K^+ channels (K_{ATP} channel) to prevent a decrease in cellular ATP levels or prevent accumulation of metabolic end-products. Lactate, as well as ADP and H^+ have been identified as potential modulators of the K_{ATP} channel and thus modulators of fatigue in this model (Renaud, 2002).

To date, the exact mechanisms of muscular fatigue during high-intensity exercise remain undetermined and are most likely the result of a multitude of interacting factors. Fitts (2003) states that fatigue recovery from high-intensity exercise has both a rapid and slow component likely caused by separated mechanisms. The rapid component being reversible is related to P_i and changes in the excitation-contraction coupling and Ca^{2+} regulation, while the slower component involves several sites and steps in the contraction process that are at least partially mediated by H^+ and P_i (Fitts, 2003). While the role of La^- in the development of fatigue has shifted from causative factor to a potential mediator, the fact that fatigued muscles display increased La^- concentrations attests to the fact that La^- accumulation has some role in the development of fatigue.

2.4 Active Recovery and Lactate Clearance

Numerous studies have provided unequivocal evidence that AR expedites blood La^- clearance compared to passive recovery (Belcastro & Bonen, 1975; Davies et al., 1970; Hermansen & Stensvold, 1972; Jervell, 1928; Newman et al., 1937; Rämmal & Ström, 1949; Siebers & McMurray, 1981; Stamford et al., 1981; Weltman et al., 1979). Beginning with Jervell (1928) it was noted that blood La^- concentration declined more rapidly during exercising recovery. This was the first scientific paper on what would later become known as AR. Later, Margaria et al. (1933) discovered that La^- clearance rate is proportional to its concentration. Newman et al. (1937) concluded that La^- clearance rate increases in approximate proportion to metabolic rate up to a critical intensity, which varies among individuals and is higher in those that are trained. Subsequent studies confirmed these initial findings (Belcastro & Bonen, 1975; Davies et al., 1970; Hermansen & Stensvold, 1972; Rämmal & Ström, 1949; Siebers & McMurray, 1981; Stamford et al., 1981; Weltman et al., 1979), and the literature is conclusive that AR increases the rate of La^- clearance from the blood. At the same time AR has been shown to have the reverse effect on muscle La^- concentration in the exercised muscle groups (McAinch et al., 2004; Peters-Futre, Noakes, Raine, & Terblanche, 1987). These researchers found that AR increased muscle La^- concentration, and attributed the increase to increased local metabolic activity that resulted in La^- production. However, these findings are not unanimous as other investigators have found AR to decrease muscle La^- concentration (Bangsbo, Graham, Johansen, & Saltin, 1994; Spencer, Bishop, Dawson, Goodman, & Duffield, 2006).

While it is agreed that AR clears blood La^-, the fate of the cleared La^- has been less obvious. There are two main biochemical processes that have been identified as the route for La^- elimination: (1) gluconeogenesis and glyconeogenesis, and (2) oxidation in the tricarboxylic

cycle to CO_2 and H_2O with energy production (Rontoyannis, 1988). Gladden (2003) takes the division further and differentiates between glyconeogenesis in muscle and uptake by the liver and/or kidneys with subsequent formation of glucose and/or liver glycogen. Previously it was believed that La^- was predominantly removed via glyconeogenesis in the liver (Rowell et al., 1966). However, evidence shows that post-exercise the majority of La^- (55-75%, ref. Brooks & Gaesser, 1980) is not resynthesized to glycogen, but rather is oxidized in the muscles (Bangsbo et al., 1994; Hermansen & Stensvold, 1972; Peters-Futre et al., 1987; Rontoyannis, 1988). Human studies estimating the post-exercise conversion of La^- to glycogen have shown varied results: approximately 70% (Hermansen & Vaage, 1977), approximately 50% (Astrand, Hultman, Juhlin-Dannfelt, & Reynolds, 1986), and between 13-27% (Bangsbo, Gollnick, Graham, & Saltin, 1991). More recently it has been shown that in humans post-exercise La^- makes minor contributions to glycogen synthesis (Bangsbo, Madsen, Kiens, & Richter, 1997). Furthermore, it has been shown that La^- is specifically oxidized in the oxidative muscle fibres (type I), whereas it is predominantly produced in the glycolytic fibres (i.e. type II, ref. Donovan & Pagliassotti, 2000; Gladden, 2000). Taken in consideration with the 'lactate shuttle' hypothesis introduced in 1984 by Brooks which states: "the shuttling of lactate through the interstitium and vasculature provides a significant carbon source for oxidation and gluconeogenesis during rest and exercise" (Brooks, 1985) it is apparent that the La^- produced by glycolytic muscle fibres is subsequently oxidized by oxidative fibres. When AR is performed after high-intensity exercise the accumulated La^- is cleared via oxidation in type I fibres that are active during low-intensity exercise. Therefore, the major clearance pathway of La^- post-exercise, especially with AR, appears to be oxidation (Gladden, 2003). It is now known that La^- traverses the plasma membrane via stereo-specific, pH-dependant transmembrane proteins called monocarboxylate transporters (MCTs, ref. Bonen, 2001). In human skeletal muscle the transporter is present in two isoforms (MCT1 and MCT4, ref. Bonen, 2001), with MCT1 being important to La^- clearance (Thomas et al., 2005).

It is proposed that AR maintains blood flow post-exercise thereby allowing the transport and circulation of La^- to sites where it can subsequently be oxidized, primarily by skeletal muscle and additionally in smaller quantities by other tissues (i.e. heart, liver, kidneys). In addition to blood flow and membrane transport, La^- release is dependent on exercise intensity and duration, training status, and age (Graham, Sinclair, & Chapler, 1976; Juel, 1997).

2.4.1 Exercise Intensity, Mode, and Duration

In order for AR to successfully clear La^-, the intensity must be such that the production of La^- does not exceed its clearance rate. If La^- production surpasses La^- clearance (i.e. utilization) then it will accumulate in the muscle and blood. The general agreement regarding the optimal intensity with which to perform AR is that the intensity should be moderate (approx. 30-45% $\dot{V}O_{2max}$, ref. Boileau, Misner, Dykstra, & Spitzer, 1983; Davies et al., 1970). Nevertheless intensities ranging from approximately 16-70% of $\dot{V}O_{2max}$ have been reported in the literature (Corder, Potteiger, Nau, Figoni, & Hershberger, 2000; Hermansen & Stensvold, 1972).

Using a bicycle ergometer Davies et al. (1970) investigated the effects of four different recovery intensities on blood La^- clearance in a group of four subjects. Following 6 min of exercise at 80% $\dot{V}O_{2max}$ subjects performed 40 min of recovery exercise at approximately 15, 30, 45 or 60% $\dot{V}O_{2max}$. The results showed that the optimal La^- clearance rate occurred between 30-45% $\dot{V}O_{2max}$. In contrast, using treadmill exercise consisting of three 60 s maximal efforts separated by approximately 4 min of rest with the final work bout being followed by 30 min of AR at one of four intensities (approx. 30, 60, 70 or 80% $\dot{V}O_{2max}$) Hermansen and Stensvold (1972) found that La^- cleared fastest at approximately 63% (range 55-70%) $\dot{V}O_{2max}$. In accordance with Davies et al. (1970), Belcastro and Bonen (1975) reported that optimal exercise intensity for La^- clearance on a bicycle ergometer was predicted at 32% $\dot{V}O_{2max}$ and additionally those subjects were able to self-select adequate intensities to clear La^-. Later, using treadmill exercise Bonen and Belcastro (1976) reported that self-selected running intensity, corresponding to approximately 61.4% (range 45.2-70.6%) $\dot{V}O_{2max}$, resulted in statistically significantly faster La^- clearance rates compared to self-selected intermittent exercise and resting recovery. They noted that though these findings are similar to that of Hermansen and Stensvold (1972), their study did not allow the assertion of whether this intensity was optimal for La^- clearance, since the two AR were self-selected (Bonen & Belcastro, 1976). In an attempt to differentiate between recoveries below and above AT, Stamford et al. (1981) demonstrated that the apparent rate of clearance could be manipulated by selecting different baseline asymptotes. Graphing semilogarithmic plots of La^- disappearance using a resting baseline La^- value of 0.9 $mmol \cdot L^{-1}$ showed statistically significantly faster clearance with AR at 40% $\dot{V}O_{2max}$ compared to AR at 70% $\dot{V}O_{2max}$ and PR. Conversely, using the same data with experimentally determined baseline values of La^- (1.3 $mmol \cdot L^{-1}$ and 3.5 $mmol \cdot L^{-1}$, respectively) yielded no differences in disappearance between active recoveries. Therefore, both AR intensities were able to return La^-

levels to their respective baselines faster than PR, but AR at 40% cleared La^- faster overall. It may be that the 70% recovery may not have been above threshold as evidenced by the low baseline La^- value of 3.5 $mmol \cdot L^{-1}$. This may explain the lack of difference between recovery intensities, as La^- would not have been accumulating in significant quantity if the workload was in fact below AT.

In order to investigate whether the inconsistencies surrounding clearance rate and recovery intensity found above were related to exercise mode (i.e. bicycle vs. treadmill) Boileau et al. (1983) compared bicycle and treadmill recovery exercise at various intensities. They found no statistically significant differences between optimal La^- clearance rate intensities across modalities (35.9% and 32.5% $\dot{V}O_{2max}$ for cycling and running, respectively). It should be noted that this experiment was comprised of a small sample of three females. In addition as suggested from another study with a larger sample size of seven males blood La^- cleared fastest at moderate intensities (i.e. 28.2-43.1% $\dot{V}O_{2max}$, ref. Boileau et al., 1983). This study was conducted using only bicycle ergometry.

It appears that the optimal recovery intensity to remove blood La^- is moderate. This is more definite for bicycle ergometer work, but it may be the case that the optimal intensity is higher for treadmill recovery. It has been shown that for a given O_2 consumption there is a greater rate of lactate production for bicycle exercise compared to running on a treadmill (Peters-Futre et al., 1987). From a theoretical perspective this may be a consequence of the greater amount of musculature involved in running versus cycling, which may provide more potential for the oxidation of La^-. However, the difference in recovery modes within individuals has not been extensively investigated, as the one study that has addressed this issue used a small sample (Boileau et al., 1983).

The inconsistencies of the aforementioned studies (Belcastro & Bonen, 1975; Bonen & Belcastro, 1976; Davies et al., 1970; Hermansen & Stensvold, 1972) with respect to the optimal intensity for clearance may also be a result of the quantification of exercise intensity. It is known that two subjects with similar maximal aerobic power (MAP) may display different thresholds for the accumulation of La^- (Dodd et al., 1984). Since previous research has typically quantified recovery loads as a percentage of $\dot{V}O_{2max}$ it follows that within and between studies subjects exercise capacities relative to La^- thresholds may have varied. Logically this can be avoided by basing recovery intensities on La^- thresholds. McLellan and Skinner (1982) investigated the intersubject variability of La^- clearance rates when expressed relative to $\dot{V}O_{2max}$ or aerobic threshold (AerT). Aerobic threshold was defined by the authors as the first "break" in the plot of

$\dot{V}_E$ versus $\dot{V}O_2$ and the initial continuous rise in La^-. They found that in 15 males ($\dot{V}O_{2max}$ of 51.5 $mL \cdot kg^{-1} \cdot min^{-1}$) AerT values varied between 45-62% ($\bar{X}$ = 52.9%) $\dot{V}O_{2max}$ and that recovery intensity expressed relative to AerT explained 13% more variance than $\dot{V}O_{2max}$ (77% and 64%, respectively). Therefore it is slightly more advantageous to quantify recovery intensity by AerT than solely by $\dot{V}O_{2max}$. Moreover, peak La^- clearance rate was predicted to be 10% below AerT (i.e. 43% $\dot{V}O_{2max}$), which is in agreement with values previously reported (Davies et al., 1970).

Subsequent research typically has selected AR intensities from the aforementioned studies (e.g. Thiriet et al., 1993). As a result, the current belief is that AR at intensities between 30-45% $\dot{V}O_{2max}$ is optimal for La^- clearance. This perception has been reinforced by research that has shown that La^- clears fastest at moderate workloads (Dodd et al., 1984; Gmada et al., 2005; McAinch et al., 2004; Spierer et al., 2004). However, there are still investigations that have used higher intensity recoveries for both cycling (50% $\dot{V}O_{2max}$, ref. Monedero & Donne, 2000) and running (approx. 60% $\dot{V}O_{2max}$, ref. Bonen & Belcastro, 1976) and reported substantial La^- clearance.

More recently Baldari et al. (2004; 2005) have quantified recovery intensity in relation to both the IVT and IAT. They compared the effects of four 30 min recovery intensities on blood La^- clearance after 6 min of treadmill running at 75% of the difference between IAT and $\dot{V}O_{2max}$ (approx. 90% $\dot{V}O_{2max}$) in both soccer players and triathletes (Baldari et al., 2004; Baldari et al., 2005, respectively). The recovery intensities were: IVT, $IVT_{+50\%\Delta T}$, $IVT_{-50\%\Delta T}$ and PR, where ΔT is the difference between IAT and IVT. In soccer players it was found that the two lowest intensity recoveries ($IVT_{-50\%\Delta T}$ and IVT) were the most efficient for La^- clearance and statistically faster than $IVT_{+50\%\Delta T}$ and PR (Baldari et al., 2004). In the triathletes $IVT_{-50\%\Delta T}$ removed La^- statistically faster than the other three intensities (Baldari et al., 2005). The authors note that all recovery intensities used were within the range (30-70% $\dot{V}O_{2max}$) previously reported for optimal La^- clearance.

Other investigations have used combined recoveries (CR, i.e. more than one recovery method within one recovery period). Taoutaou et al. (1996) compared PR (20 min seated rest on bicycle ergometer followed by 40 min seated rest) to partially AR (20 min bicycling at 40% $\dot{V}O_{2max}$ followed by 40 min seated rest) and found that the partially AR cleared La^- 1.5- and 3-fold faster in untrained and trained individuals, respectively. Donne and Monedero (2000) investigated blood La^- clearance and subsequent exercise performance after four different 15 min recovery interventions. The recovery interventions were: PR, AR at 50% $\dot{V}O_{2max}$, massage

recovery, and CR consisting of AR at 50% $\dot{V}O_{2max}$ for 3.75 min then massage for 7.5 min followed by AR for the remainder. Their performance indicator was the difference in completion time for a 5 km bicycle time trial simulation performed before the recovery intervention and after on each of the testing days. Their results showed that AR and CR were superior to PR recovery with respect to La^- clearance, that the fastest clearance rate occurred with AR, and that the clearance rate was fastest during the AR exercise periods of the CR. The increase in 5 km completion time was significantly less in the CR intervention compared to all other recoveries. The authors speculate that the greater performance maintenance in the CR trial was due to the combination of greater La^- clearance in the active periods and increased intramuscular glycogen restoration during the passive massage periods.

Post-exercise blood La^- levels peak after a small lag in time of approximately 1-7 min (Bret et al., 2003; Dodd et al., 1984; Gmada et al., 2005; Taoutaou et al., 1996; Thiriet et al., 1993), since La^- must be transported out of the cell via MCTs to the circulatory system. The time to reach peak blood La^- appears to be dependent on metabolic rate, since it peaks faster with higher intensity recovery intensities, and sooner with AR versus PR (Gmada et al., 2005; Stamford et al., 1981; Taoutaou et al., 1996). Afterwards blood La^- levels will begin to decrease provided that the energy demand is below the threshold at which blood La^- accumulates. Depending on the peak blood La^- levels and recovery intensity, values may remain elevated for up to 1.5 hours before reaching resting levels (Bret et al., 2003; Choi, Cole, Goodpaster, Fink, & Costill, 1994; Taoutaou et al., 1996).

Studies have utilized a wide variety of recovery durations ranging from 4-90 min to investigate La^- clearance (Corder et al., 2000; Taoutaou et al., 1996). When comparing clearance rates between AR and PR statistically significant differences are usually not evident until approximately 10 min (Gmada et al., 2005; Monedero & Donne, 2000). Interestingly, the two investigation previously discussed by Baldari et al. (2004; 2005) both demonstrated that all active recoveries examined did not show further decreases in blood La^- after the twentieth minute of recovery. The investigations examined two different subject pools: soccer players and triathletes ($\dot{V}O_{2max}$ of 62.3 and 69.7 $mL \cdot kg^{-1} \cdot min^{-1}$, ref. Baldari et al., 2004; Baldari et al., 2005, respectively). The recovery intensities were: IVT, $IVT_{+50\%\Delta T}$, $IVT_{-50\%\Delta T}$ and PR, and ranged between the previously described intensities of 30-70% $\dot{V}O_{2max}$ that have been suggested to be optimal for La^- clearance (range 39-60% $\dot{V}O_{2max}$).

2.4.2 Combined Active Recovery and Lactate

Dodd et al. (1984) and more recently Gmada et al. (2005) have investigated the effects of a two stage CAR on La^- clearance. The precedence for their research came from the work of Stamford et al. (1981) who compared the effects of three different 40 min recoveries on La^- clearance: PR, and AR at 40% and 70% $\dot{V}O_{2max}$ (AR40% and AR70%, respectively). They found that La^- clearance was greater in the AR40% from 30-40 min compared to PR and AR70%. Additionally it was shown that La^- levels peaked faster with AR70%. Since La^- uptake is proportional to its concentration (Jorfeldt, 1970; Margaria et al., 1933) it was theorized that as La^- concentration decreases, uptake decreases in proportion, resulting in a decrease in clearance rate since La^- is still being produced during active recovery, albeit at lower levels. Thus it was hypothesized that optimal La^- clearance may occur with an exercise intensity that matches its concentration (Stamford et al., 1981). That is, as La^- concentration progressively decreases the exercise intensity should decrease to limit La^- production and therefore enhance clearance.

The first group to test the latter hypothesis was Dodd et al. (1984). They combined moderate- to high-intensity and moderate- to low-intensity work within one recovery period in an attempt to optimize La^- clearance. A sample of seven trained males ($\dot{V}O_{2max}$ of 48.7 $mL \cdot kg^{-1} \cdot min^{-1}$) performed 50 s of supramaximal bicycle work at 150% $\dot{V}O_{2max}$ followed immediately by one of four 40 min recoveries: PR, AR at 35% $\dot{V}O_{2max}$ (AR35%), AR at 65% $\dot{V}O_{2max}$ (AR65%), or 7 min at 65% followed by 33 min at 35% $\dot{V}O_{2max}$ (CAR). Differences in clearance rate at the 6^{th} min and from min 20 to 40 of recovery were examined, and results demonstrated that from minute 20 to 40 La^- clearance was statistically significantly faster in the AR35% and the CAR trial. No statistically significant differences were observed between AR35% and CAR during this time frame and the authors therefore concluded "that these data do not support the hypothesis that following maximal work, a combination of submaximal exercise intensities is more beneficial in lowering blood La^- concentrations than a single intensity" (Dodd et al., 1984). Conversely, in retrospect their results could be interpreted such that CAR was able to clear La^- to the same extent as AR35% since there were no statistical differences between the two conditions. The question can then be proposed that if the two recovery strategies differ in intensity but still clear La^- to the same extent, do they differ in any other respects. For example, do they result in differences in performance independent of their respective effects on La^-.

Gmada et al. (2005) re-investigated this hypothesis with several modifications to the work of Dodd and colleagues. A larger sample of fourteen subjects (7 trained and 7 untrained, $\dot{V}O_{2max}$ of 56.5 and 42.0 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively) performed three supramaximal intermittent

exercise bouts at 120% MAP for 60% of the time to exhaustion (TTE) separated by 5 min intervals with the third bout being followed by 20 min of recovery. The recovery bouts were as follows: PR, AR at 20% less than the first ventilatory threshold (VT1), AR at 20% less than the second ventilatory threshold (VT2), and CAR consisting of 7 min at VT2 followed by 13 min at VT1. Their findings demonstrated that peak blood La^- occurred faster in CAR and VT2 conditions for both trained and untrained subjects (4^{th} and 7^{th} min, respectively). This was in accordance with previous work (Dodd et al., 1984; Stamford et al., 1981) and confirmed that blood La^- peaks sooner post-exercise with higher-intensity recovery. In contrast to Dodd et al. (1984) it was determined that La^- disappeared statistically significantly faster in both groups with CAR, the effect being more pronounced in the trained group (Gmada et al., 2005).

The discrepancy between results with respect to the efficacy of CAR of the two studies could be due the differences in subject's fitness level or the protocols used. Dodd et al. (1984) used only one group of seven trained subjects with an average $\dot{V}O_{2max}$ of 48.7 $mL \cdot kg^{-1} \cdot min^{-1}$. Whereas Gmada et al. (2005) used two groups of seven subjects, one trained and one untrained with $\dot{V}O_{2max}$ of 56.5 and 42.0 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively. Furthermore, Gmada et al. (2005) quantified the recovery intensities specific to ventilatory thresholds, while Dodd et al. (1984) only used $\dot{V}O_{2max}$. Since two subjects with a similar $\dot{V}O_{2max}$ can exhibit different anaerobic thresholds, the possibility exist that the recoveries chosen in the study by Gmada et al. may have been less likely to surpass an individual's anaerobic threshold and therefore result in less La^- production during the recovery period (2005). Furthermore, Gmada et al. used a repetitive exercise task to induce increased blood La^-, which resulted in higher blood La^- concentrations. Since the rate of La^- uptake is proportion to its concentration (Jorfeldt, 1970; Margaria et al., 1933) it may be that the higher blood La^- levels had some effect on clearance rate. However, with respect to the actually blood La^- concentrations this does not appear to be the case, although it may be that the effect was not evident in blood La^- concentrations. Additionally, the 5 min recoveries between the repetitive exercise bouts in the study by Gmada et al. (2005) matched the longer duration recovery period, with respect to recovery intensity, and may have influenced the results.

In summary it has been shown that the use of AR facilitates La^- clearance compared to PR. It may be that the optimal AR intensity to clear blood La^- is dependent on recovery mode or the active muscle mass. Moderate intensity AR is recommended to clear blood La^- fastest when performing bicycle exercise. However, the optimal intensity with which to clear La^- remains

undetermined and may be dependent on individual thresholds or occur with the use of more than one recovery intensity.

2.5 Lactate and Performance

As shown above AR is an appropriate means to remove La^- following high-intensity exercise in which there is an accumulation of La^-. However, the real interest surrounding AR for coaches, athletic trainers, strength and conditioning coaches, and athletes is whether or not AR improves performance. The evidence for the use of AR as a means of improving performance following previous exercise is less obvious.

Lactate is the one of the most researched metabolites. From an early time it was believed to be the direct cause of fatigue (Hill & Kupalov, 1929). The concept that AR from exercise bouts may be beneficial to subsequent performances like many training and competition techniques in the sport sciences has some of its origin in anecdotal observations. Gisolfi, Robinson and Turrell highlight this in their 1966 paper in which they conclude that their work provides a physiological basis for the practice of AR post-competition; which they note athletes had already learned from experience. The authors formulate this conclusion after finding that moderate aerobic exercise (approx. 38-53% $\dot{V}O_{2max}$) for 30-35 minutes following exhaustive treadmill running reduced the "oxygen debt" and cleared La^- faster than a resting recovery in the four subjects they examined (Gisolfi et al., 1966). No attempt was made to examine performance in this investigation and their conclusion was likely established based on current beliefs of that time pertaining to La^- and fatigue.

Later, more empirical evidence would imply that high "lactic acid" concentration may be the reason for exhaustion in high-intensity exercise (Karlsson & Saltin, 1970). To test the hypothesis that high blood La^- concentration may limit maximal exercise performance Klausen et al. (1972) had subjects perform maximal bicycle work that was either preceded by rest or high-intensity arm ergometry. Therefore, high blood La^- concentration was induced and its effect on a previously non-exercised muscle group was examined. While their results were not statistically significant they did observe an average 10% reduction in TTE in the condition in which leg work was preceded by arm work. The authors concluded that increased La^- concentration in the working muscles inhibited further La^- production, but that the hypothesis that La^- is a limiting factor in exercise was not confirmed since only a trend to reduced endurance time was observed. In a similar experiment in which arm exercise preceded leg exercise (series A-L) and vice versa (series L-A, i.e. leg exercise preceding arm exercise) on a separated occasion, it was observed that TTE occurred earlier in both conditions with respect to

TTE without preceding exercise (Karlsson et al., 1975). The authors concluded that because La^- was elevated prior to the second exercise bout in both conditions and therefore reached peak values sooner, these peak La^- values (20-30 $mmol \cdot kg^{-1}$ wet muscle) or related factors serve as limiting factors to muscular performance.

However, the latter two studies (Karlsson et al., 1975; Klausen et al., 1972) had small sample sizes (n = 4 and 3, respectively) limiting the conclusiveness of their results. It is also worthwhile to note that the two investigations did not use SE bouts. Nonetheless, the investigations are foundational to the belief that increased blood La^- concentration can limit performance, or perhaps more appropriately stated, is related to performance decrements. Furthermore, it is this foundational belief that allows the conclusion that AR may be beneficial to performance since AR can clear blood La^- faster than a resting recovery.

2.6 Active Recovery and Performance

The first empirical evidence that AR was beneficial to subsequent high-intensity performance came from Weltman et al. (1977). After performing an initial all-out SE task (bicycling for 1 min at 5.5 kg resistance) subjects underwent one of eight recovery interventions followed by the same supramaximal criterion exercise task. Recovery interventions consisted of AR at 1 kg resistance or PR for either 10 or 20 min breathing either room air or 100% O_2. All combinations of the aforementioned variables were examined. Their findings showed that La^- clearance and subsequent performance were statistically significantly improved with active and 20 min recovery compared to the other interventions. However, they also concluded that other factors beside La^- clearance are critical to subsequent performance since these variables were not correlated.

In the time to come research findings would be divided on the effects of AR on subsequent performance. While all studies showed that AR could decrease La^- concentration better than PR, many showed no additional benefits to subsequent exercise performance (Bond, Adams, Tearney, Gresham, & Ruff, 1991; Franchini et al., 2003; McAinch et al., 2004; Siebers & McMurray, 1981; Watson & Hanley, 1986; Weltman et al., 1979; Weltman & Regan, 1983), while others would show benefits (Ahmaidi et al., 1996; Bogdanis et al., 1996b; Connolly et al., 2003; Corder et al., 2000; Signorile et al., 1993; Spierer et al., 2004; Thiriet et al., 1993).

Typically studies aimed at investigating subsequent performance have used recovery durations of 10-20 min. This is likely due to the fact that this time frame coincides with requirements and restraints put on sporting events. Previously it has been suggested that 20-40 min of AR should be used to prevent a decrease in power output (Ainsworth et al., 1993).

Interestingly, with respect to performance, recent evidence suggests that shorter durations of AR may be beneficial to performance (i.e. approx. 6 min) rather than longer durations (i.e. 15 min or longer, ref. Ahmaidi et al., 1996; Bogdanis et al., 1996b; Spierer et al., 2004).

2.6.1 No Performance Benefit with Active Recovery

A substantial body of research exists that shows that AR does not significantly improve performance. Weltman et al. (1979) studied the effects of four recoveries (PR, AR<AT, AR>AT, and AR>AT + 100% O_2) on La^- clearance and subsequent performance for an endurance task consisting of 5 min of cycling in nine males. Subjects cycled for 5 min at MAP, recovered for 20 min at one of the respective recovery intensities, and then performed a second 5 min cycle at MAP. They reported that while AR>AT + 100% O_2 cleared La^- significantly faster than PR, and AR>AT there were no significant differences in performance among recovery conditions. Performance was determined by work done over the 5 min and assessed by pedal revolutions completed. Siebers and McMurray (1981) investigated blood La^- clearance and subsequent performance of a 200 yd swim 15 min after a 2 min swim at 90% $\dot{V}O_{2max}$ on a swimming ergometer in six females. The 15 min recoveries consisted of either walking at a moderate pace (2.5-3 mph) for 10 min followed by 5 min of PR or swimming continuous front crawl lengths at a moderate pace for 10 min followed by 5 min of PR. Despite reporting that swimming recovery cleared 22% more La^- than walking, there were no statistically significant differences for 200 yd swim time. Subjects took slightly longer than 2 min to complete the 200 yd swims for the swim and walk recoveries (125.9 ± 5.9 s and 127.2 ± 5.8 s, respectively).

In an attempt to amalgamate the literature, Weltman and Regan (1983) investigated the effects of 20 min of active and passive recovery on subsequent maximal constant load (i.e. no work drop-off allowed) exercise performance. At this point in time research supporting the contention that elevated blood La^- concentration has a detrimental effect on subsequent performance had used a constant load protocol (Karlsson et al., 1975; Klausen et al., 1972) and research not in support had used maximal effort work (i.e. allowing a work drop-off, ref. Weltman et al., 1979). In contrast to the previous work utilizing constant load tasks, they found no statistical differences in work output between the recovery conditions.

An applied investigation had eight hockey players perform two simulated hockey tasks separated by one of three 15 min recovery modes: skating, bench-stepping, or PR (Watson & Hanley, 1986). The hockey task was comprised of six 45 s sprints each separated by 90 s PR, with the distance skated determining each player's performance. Results demonstrated that only bench-stepping reduced La^- values with statistical significance compared to rest, but that neither

of the AR altered performance with statistical significance. However, the practical nature of this investigation resulted in difficulty standardizing recovery duration and intensity, since the AR condition had substantial passive periods to take measurements and the PR condition had substantial active periods to prepare for the subsequent exercise task. The authors note that the skating recovery may have resulted in slowed La^- clearance because of the ability of the players to glide, limiting the amount of muscular work they performed. The effect of this on performance cannot be determined.

Bond et al. (1991) investigated the effects active and passive recovery on subsequent isokinetic muscle function. They used a 60 s bicycle ergometer task at 150% $\dot{V}O_{2max}$ to elevate blood La^- levels followed by either 20 min of AR at 30% $\dot{V}O_{2max}$ or PR. Subjects then performed 60 repeated isokinetic knee extensions at an angular velocity of $150^{\circ}\cdot s^{-1}$. Results again confirmed the superiority of AR to PR with respect to La^- clearance but no differences in peak torque, total work or fatigue index were noted between recovery conditions or control values. Another applied investigation used two simulated hockey tasks consisting of seven 40 s 'shifts' separated by 90 s rest with 15 min of AR or PR between skating tasks (Lau, Berg, Latin, & Noble, 2001). The AR, which consisted of self-selected resistance "low-intensity" cycling, did not clear La^- faster than the PR or effect subsequent performance in a beneficial way with respect to statistical significance. However, the authors did note a trend towards a greater distance skated in the second bout of simulated hockey shifts with AR. Additionally, the authors note that their findings may have been limited by the fact that the recovery intensity was self-selected and that the skating bouts only induced moderate La^- concentrations. Furthermore, similar to Watson & Hanley's (1986) investigation the recoveries were not strictly passive or active due to constraints of the experimental design.

Recently two investigations have examined the effects of active versus passive recovery on La^- clearance and subsequent anaerobic and aerobic performance, respectively (Franchini et al., 2003; McAinch et al., 2004). In one of the investigations a group of seventeen subjects first participated in a 5 min judo combat followed by 15 min of AR (running at 70% of the anaerobic threshold velocity) or PR (Franchini et al., 2003). They then completed an intermittent anaerobic task comprised of four upper body WAnT each separated by 3 min PR. Performance on the WAnT was not altered by recovery mode. The authors note that this finding is in accordance their observations that AR seems to be beneficial to performance when the recovery period is 6 min or less, and that with recoveries of 15 min or longer AR does not appear to be beneficial. This is further supported by the work of McAinch et al. (2004). They investigated the effects of

AR (40% $\dot{V}O_{2max}$) and PR on muscle biopsies and plasma La^- clearance, as well as performance of intense aerobic exercise. Seven male subjects performed two 20 min bicycle trials separated by 15 min of recovery. No differences in work performed or muscle glycogen and La^- concentrations were observed, but plasma La^- concentration was significantly lower in the AR protocol.

2.6.2 Performance Benefit with Active Recovery

Despite the wealth of literature that has shown that AR does not appear to be beneficial to performance there is a similar amount of literature to the contrary. In addition to the initial research that exhibited that AR may be advantageous to recovery and/or maintenance of performance (Karlsson et al., 1975; Klausen et al., 1972; Weltman et al., 1977) others have produced supporting literature. Pendergast et al. (1983) confirmed the earlier results of Karlsson et al. (1975) that preceding high-intensity exercise considerably reduces the potential for further supramaximal performances. They found that endurance for both aerobic and anaerobic work was reduced in the presence of high blood La^-. Other investigators have documented a similar relationship of increased blood La^- concentration and reduced muscular endurance (Yates et al., 1983). These investigators looked at the muscle contractile properties (maximum voluntary contraction (MVC), peak rate of tension development, peak rate of relaxation, one-half contraction time, and one-half relaxation time) of the elbow flexors 6 min after 1 min of intense cycling at a fixed load of 5 kg versus a control (i.e. no prior cycling). They found no statistically significant differences in muscle contractile properties after the cycle ergometer bout, but endurance time at 40% MVC was reduced by 25% with prior exercise. After the endurance task there was a statistically significant reduction in MVC, peak rate of tension development and peak rate of relaxation. It was concluded that the elevation of blood La^- by intense exercise of one muscle group reduced the endurance of a second non-exercised muscle group. However, the aforementioned investigations (Pendergast et al., 1983; Yates et al., 1983) did not actually investigate the effects of AR, but rather showed that increased blood La^- and associated changes result in decrements of performance. The investigators both suggest that the reduction of La^- concentration should therefore be beneficial to performance, as increases in La^- are detrimental to performance. This suggestion is more of an anecdotal assertion than scientific fact, but nonetheless provided a conceptual framework for the conviction that AR and hence blood La^- clearance is beneficial to performance.

Thiriet et al. (1993) investigated the effects of AR and PR on repeated SE. They had 16 male subjects perform four cycling bouts to exhaustion at 130% MAP. Each bout was separated

by a 20 min recovery period of either leg or arm ergometric exercise at 30% MAP or rest. Active recovery cleared La^- faster and maintained work performance more than PR. However, they also noted a non-significant correlation between power output and La^- levels, which has been observed by others (Siebers & McMurray, 1981; Weltman et al., 1977; Weltman et al., 1979; Weltman & Regan, 1983). Therefore, they concluded that the relationship between power output and La^- is not one of cause and effect. Other researchers have corroborated the finding that power output may be maintained to a greater extent with AR compared to PR (Ahmaidi et al., 1996; Bogdanis et al., 1996b; Connolly et al., 2003; Signorile et al., 1993; Spierer et al., 2004). Signorile et al. (1993) examined the effect of AR versus PR on power output during eight 6 s supramaximal bicycle sprints separated by 30 s. Active recovery consisted of pedaling against 1 kg of resistance at 60 rpm while PR consisted of sitting on the bicycle motionless. Mean PP and mean TW performed were statistically significantly greater in the AR protocol. This investigation used a fixed load for the recovery intensity based on previous work (Weltman et al., 1977), which limits the control of interindividual differences in fitness and work capacity. Biochemical variables were not measured and therefore the interpretation of the data is limited solely to performance parameters. Bogdanis et al. (1996b) compared the effects of recovery type on performance of two maximal 30 s bicycle sprints separated by 4 min. Active recovery resulted in statistically significantly higher MP output compared to PR. The difference in power output could be attributed to the differences observed in the initial 10 s of the sprint. The authors suggest that the increased blood flow during AR may have increased resynthesis of PCr or allowed an initially faster glycolytic rate as an explanation for the performance improvement based on results of their previous work (Bogdanis, Nevill, Lakomy, & Boobis, 1994b). Blood La^- concentration did not differ significantly between recovery conditions (Bogdanis et al., 1996b). Another investigation examined the effects of recovery type on repetitive 6 s bicycle sprints using incremental resistive forces separated by 5 min of active (32% MAP) or passive recovery in ten male subjects (Ahmaidi et al., 1996). The results showed that at the higher resistive forces the AR protocol enabled greater maintenance of power and also cleared La^- faster than the PR protocol.

Dorado et al. (2004) examined the effects of recovery mode on aerobic and anaerobic energy yield as well as performance during high-intensity intermittent exercise. Ten trained subjects ($\dot{V}O_{2max}$ of 58 $mL \cdot kg^{-1} \cdot min^{-1}$) performed four supramaximal constant intensity cycling bouts to exhaustion at 110% maximal power output each separated by one of three 5 min recoveries. The recoveries were: AR at 20% $\dot{V}O_{2max}$ (HITA), stretching recovery of the lower

limbs (HITS), or PR (HITP). Performance was 3-4% better and aerobic energy yield was 6-8% greater in the HITA condition. The greater aerobic yield was due to faster $\dot{V}O_2$ kinetics and the authors concluded that this was the source of improved performance in the AR trial. It was proposed that the faster $\dot{V}O_2$ kinetics were a result of either increased blood flow or maintenance of aerobic regulatory enzyme activation (Bangsbo et al., 1994). It has previously been shown that aerobic metabolism makes a significant contribution to metabolism during high-intensity exercise (Bogdanis, Nevill, Boobis, & Lakomy, 1996a) and the authors argued that this was the case in their investigation (Dorado et al. 2004).

More recently, a study investigated the use of a short 3 min recovery period on La^- clearance and power output (Connolly et al., 2003). In congruence with previous work utilizing similar work-to-rest intervals (Bogdanis et al., 1996b), it was found that power output on six 15 s bicycle ergometer sprints was greater with AR compared to PR, but La^- values did not differ with respect to the recovery used (Connolly et al., 2003). Spierer et al. (2004) examined both moderately trained ice hockey players ($\dot{V}O_{2max}$ of 45.6 $mL \cdot kg^{-1} \cdot min^{-1}$) and sedentary ($\dot{V}O_{2max}$ of 36.9 $mL \cdot kg^{-1} \cdot min^{-1}$) subjects on their ability to perform serial WAnT interspersed with either 4 min AR at 28% $\dot{V}O_{2max}$ or PR. Their results showed that PP output did not differ significantly between recovery types in both groups. However, sedentary subjects displayed statistically significantly greater MP output and TW values with AR, whereas only TW performed was significantly improved in moderately trained individuals with AR. Capillary blood La^- differed with statistical significance in the moderately trained group only when AR was employed. It should be noted that there were statistical differences between groups with respect to age, gender, height, and mass in addition to $\dot{V}O_{2max}$, which complicates the inter-group comparison since the group differences outside of fitness level are a source of uncontrolled variability.

Ainsworth et al. (1993) examined the effect of AR duration on blood La^- and power in 16 male competitive cyclists ($\dot{V}O_{2max}$ of 67.6 $mL \cdot kg^{-1} \cdot min^{-1}$). Following a 45 s bicycle bout subjects performed 6, 9, or 12 min of AR at a fixed resistance of 5.5 kg, after which they immediately performed another 45 s bicycle bout. Results showed that power output was decreased with statistical significance between bouts during the 6 min recovery, but was maintained in the 9 and 12 min recoveries. However, no statistically significant differences were observed in the ability to produce PP in all of the recovery durations. Recovery blood La^- was only statistically decreased in the 12 min recovery protocol. It was therefore concluded that in this population 9 min of AR at approximately 30% $\dot{V}O_{2max}$ was sufficient to restore power output to resting levels

following 45 s of supramaximal cycling. In support of this finding, another investigation in which 8 males performed two 30 s bouts of bicycle ergometer work separated by 6 min of PR, PP and MP output were only 92% and 85% of initial control values (Bogdanis, Nevill, & Lakomy, 1994a). However, this investigation used passive recovery and therefore a direct comparison of results is contentious. Nonetheless, it appears that following maximal work durations of 30-45 s greater than 6 min of recovery is necessary for power recuperation.

2.6.3 Performance and Active Recovery Duration

One commonality of the aforementioned research on AR and performance that has not found an improvement in subsequent performance is that the recovery durations were all 15 minutes or longer. Therefore, it may be that recoveries of this duration mask any additional benefits of an AR that may only be evident in the initial part of the recovery period. This contention is supported by the fact that much of the literature that has shown improved performance with AR has used recovery durations of 6 min or less (Ahmaidi et al., 1996; Bogdanis et al., 1996b; Signorile et al., 1993; Spierer et al., 2004). This trend has been previously noted (Franchini et al., 2003). Of the studies reporting improved performance with AR in this literature review only one used a long recovery duration (i.e. 20 min, ref. Thiriet et al., 1993). Interestingly, a study that investigated performance on a resistance training (i.e. parallel squat) task in which exercise sets were separated by 4 min of cycling or PR it was shown that AR cleared La^- faster and resulted in better performance (Corder et al., 2000). This is in agreement with the aforementioned trend. Also of interest, is the fact that several of the studies reporting improved performance with AR did not correlated or associated this improvement with La^- clearance (Bogdanis et al., 1996b; Connolly et al., 2003; Signorile et al., 1993; Spierer et al., 2004; Thiriet et al., 1993). Therefore, while it appears that a short duration AR may be beneficial to performance it may not be related to the clearance of La^- and could be a result of increased blood flow and its effects on subsequent metabolism (Bogdanis et al., 1994b; Bogdanis et al., 1996b).

2.7 Summary

It has been unequivocally shown that the use of AR can remove La^- at a faster rate than PR. It is proposed that the accelerated clearance rate with AR is a result of heightened blood flow that serves to circulate La^- and provide a fuel source for the working muscles while also being oxidized by resting muscles and other tissues to a lesser extent. While it has been postulated that increased La^- clearance should be beneficial to subsequent exercise performance the literature is indeterminate. Active recovery may be only beneficial for short duration

recovery because it can maintain blood flow increasing the aerobic energy contribution to exercise, whereas with longer durations the return to homeostasis may be more similar between recovery types and result in similar performance.

CHAPTER III: METHODS

3.1 Subjects

The subjects were comprised of a sample of 12 volunteer, trained, male, university-aged (18-35 years) cyclists. An a priori power calculation performed using the G*Power software package (G*Power Version 2.0, Germany) was used to determine sample size (Faul & Erdfelder, 1992). The analysis was performed with the intent of detecting a 10% decrement in MP output assuming an average final power output of 800 W with a standard deviation of 50 W. A 10% power decrement was selected because a decrease of this magnitude or less in muscle performance may be sufficient to limit whole body exercise performance in a competitive setting (Sprague & Mann, 1983). The power output stated above is based on previous research examining the performance of competitive road cyclist on a single WAnT (Tanaka, Bassett, Swensen, & Sampedro, 1993) and preliminary pilot data from this laboratory.

Subjects were recruited from the University of British Columbia campus and Vancouver area. All subjects were non-smokers and not under any pharmacological or special dietary treatment during the investigation. Subjects were defined as trained for this investigation if their training included both a heavy aerobic and anaerobic component. Additionally, subjects were required to meet at least three of the following four criteria: currently participating in regular competitive level cycling, have PP and MP outputs on the WAnT greater than or equal to 11 and 9 $W \cdot kg^{-1}$, respectively, and have a $\dot{V}O_{2max}$ greater than 55 $mL \cdot kg^{-1} \cdot min^{-1}$.

Prior to participation all subjects were informed of potential risks and benefits associated with participation and completed a written Informed Consent, approved by the UBC Clinical Research Ethics Board (see Appendix X), and a Physical Activity Readiness Questionnaire (PAR-Q). Subjects were required to weigh less than 95 kg, as the resistance load on the Monark (Ergomedic 874E, Monark Exercise AB, Sweden) ergometer becomes less accurate with athletes over this weight (Inbar, Bar-Or, & Skinner, 1996). However, no subjects outside of this requirement attempted to participate in the study.

3.2 Experimental Design

A randomized counterbalanced within-subjects design was used to evaluate the treatment effects. Subjects were randomly assigned to one of three recovery modes (AR, PR, and CAR) and the order in which they performed each trial was counterbalanced in order to control for treatment order effects.

3.3 Facilities and Instrumentation

All testing was completed at the John M. Buchanan Exercise Science Laboratory within the University of British Columbia Aquatic Centre. Equipment was calibrated, as per manufacturers' instructions, prior to testing. Anthropometric measurements of height and weight were taken for each subject on the first testing day prior to exercise. Additionally, the sum of five skinfolds (biceps, triceps, subscapular, iliac crest, and medial calf) were taken as a measurement of body composition according to the procedures set out in The Canadian Physical Activity, Fitness & Lifestyle Appraisal Manual (CSEP, 1998), using Standard Harpenden calipers (Baty International, UK).

3.3.1 Wingate and Sprint Tests

Performance was measured using a repetitive sprint task comprised of five 10 s maximal sprints separated by 30 s of PR. Both the sprints and WAnT were performed on a pan load Monark Ergomedic 874E (Monark Exercise AB, Sweden) bicycle ergometer. Subjects cycled against a set load of 0.09 kg·(kg body mass)$^{-1}$, for the respective test durations, as recommended for athletes on the WAnT (Inbar et al., 1996). In order to calculate power outputs the velocity of the bicycle flywheel is determined by way of an optical sensor (SMI OptoSensor, USA) that records pulses from reflective markers fitted to the flywheel. The sensor was interfaced with a PC equipped with SMI POWER Version 5.2.8 software (SMI, USA). The software then calculates power parameters based on the measured flywheel velocity and belt friction (i.e. applied resistive force). Peak Power and MP were calculated for the WAnT for use as subject inclusion criteria. Peak Power, MP, and FI were calculated for each of the five sprints, and TW was calculated for the entire sprint trial using Microsoft Excel Version 5.1.2600 (Microsoft Corporation, USA). The SMI POWER (SMI, USA) standard power outputs were used rather than the corrected power outputs. The variables calculated were defined as:

- Peak power – the highest mechanical power output over 1 s achieved during the sprint.
- Mean power – the average power output maintained over the entire 10 s sprint.
- Fatigue index – the difference between PP and the lowest 1 s power output divided by PP and expressed as a percentage.
- Total Work – the sum of the product of mean power and time across all five sprints.

The ability to objectively quantify the capacity for intense activity is one of the most difficult components of measuring athletic performance (Inbar et al., 1996). Since anaerobic ATP production is an intracellular process there are no precise methods to quantify the energy release, and therefore no direct "gold standard" method of validation (Gastin, 2001; Inbar et al.,

1996). Therefore like other measures of anaerobic capacity the validity of this type of sprint task is contestable. However, it has been stated that "the choice of an anaerobic test depends on the aims and subjects of a study and its practicability" (Vandewalle, Peres, & Monod, 1987). Therefore, due to practicability and the desire to examine repeat high-intensity exercise performance, the model of five 10 s sprints was chosen as the performance indicator for this investigation.

3.3.2 Ventilatory and Gas Exchange Variables

Maximum oxygen consumption was evaluated by an incremental stage bicycle ergometer test, performed on an electronically braked SensorMedics Egrometrics 800 bicycle (SensorMedics, USA) utilizing the SensorMedics Vmax 29 series metabolic measurement cart (SensorMedics, USA). Breath-by-breath values of $\dot{V}O_2$, $\dot{V}CO_2$ (volume of CO_2 produced), $\dot{V}_E$ (minute ventilation) were averaged over 20 s intervals and recorded for analysis. The exact protocol used was adapted from previous incremental bicycle and treadmill tests (Baldari & Guidetti, 2000; Stegmann et al., 1981). Subjects started at an initial workload of 120 W and performed 3 min stages with step increments of 30 W until the attainment of their IAT. After which, the stage durations were decreased to 1 min and the step increments were maintained at 30 W until exhaustion. The criteria used to determine the attainment of $\dot{V}O_{2max}$ was operationally defined as the achievement of any two of the following: volitional fatigue, plateau in $\dot{V}O_2$ (i.e. change < 2.1 $mL \cdot kg^{-1} \cdot min^{-1}$), 90% age predicted maximum HR, respiratory exchange ratio ($\dot{V}CO_2/\dot{V}O_2$) exceeding 1.15, or blood La^- concentration greater than 8 $mmol \cdot L^{-1}$ (Duncan, Howley, & Johnson, 1997). Subjects were asked to maintain a pedal cadence above 80 rpm when possible to facilitate the ease of cycling on the mechanically braked bike, but were allowed to pedal at any cadence that was above 60 rpm.

The same apparatus was used on subsequent testing days to record ventilatory data during the exercise and recovery bouts. Data was again sampled breath-by-breath and averaged over 20 s intervals for analysis.

3.3.3 Blood Lactate

Lactate measurements were made using a portable La^- analyser (ARKRAY Inc., Japan). Briefly, the finger to be lanced was disinfected with an alcohol pad before a fingertip puncture was made. The first drop of blood was cleared away (along with any non-evaporated alcohol) with sterile gauze and then a second drop was pressured out to fill a reagent strip via capillary action with approximately 5 μL of blood. Lactate in the sample reacts with potassium ferricyanide and La^- oxidase to form potassium ferrocyanide and pyruvate. A given voltage is

then applied oxidizing ferrocyanide, releasing electrons and creating a current. The current is measure amperometrically and is directly proportional to the blood La^- concentration of the sample (Pyne, Boston, Martin, & Logan, 2000). Capillary blood La^- values have been shown to accurately reflect arterial blood La^- values (Williams, Armstrong, & Kirby, 1992). The Lactate Pro has been shown to be accurate, reliable and demonstrate a high degree of agreement with other La^- analysers (Pyne et al., 2000).

3.3.4 Heart Rate

Heart rate was measured and recorded using a Polar s120 HR Monitor and uploaded to a PC using Polar Infrared Connection (Polar Electro, Finland). Heart rate data was provided every 5 s.

3.4 Testing Procedures

Subjects attended the lab on six days separated by at least 48 hours. One exception to this guideline was made for Subject 3 who completed testing days 2 and 3 within 28 hours due to a conflict of schedule. On all testing days subjects were asked to report to the laboratory two hours after a suggested snack of a whole wheat bagel and banana. In addition, to control for the effects of nutritional and exercise status on performance subjects were asked to follow similar dietary and exercise habits throughout the experiment on both testing and off-days. Furthermore, in the 24 hours preceding a testing day subjects were asked to avoid intense physical activity, alcohol intake, and refrain from caffeine intake in the 3 hours preceding a testing session. Prior to all testing days, subjects were asked to report whether or not they complied with the aforementioned guidelines.

3.4.1 Day 1: Anthropometric Measures, $\dot{V}O_{2max}$, and WAnT

On subjects' first testing day anthropometric measurements and skinfolds were taken before performing a $\dot{V}O_{2max}$ test (as described above), followed by the WAnT. Bicycle seat and handle bar height were adjusted to the individuals comfort and recorded for subsequent use throughout the experiment. Blood La^- measures were taken during the $\dot{V}O_{2max}$ test at the end of each 3 min stage, until the IAT was determined. A final blood La^- measure was taken 3 to 4 minutes after the incremental test. During this time subjects cycled at 40 W in order to alleviate any discomfort associated with the cessation of intense physical activity. The IAT was operationally defined as the workload corresponding to the second La^- increase of at least 0.5 $mmol \cdot L^{-1}$ from the previous value (Baldari & Guidetti, 2000). That is, the IAT is the workload corresponding to the stage that elicits a second La^- increase greater than or equal to 0.5 $mmol \cdot L^{-1}$. However, in the aforementioned investigation the investigators demonstrated that the

maximal La^- steady state (MLSS) was more accurately predicted when the workload from the antecedent test stage was attributed to the blood La^- value, rather than the workload of the same stage. Therefore in this investigation the workload used to quantify the IAT was the workload corresponding to the stage antecedent to that in which the IAT blood La^- value was observed.

Additionally the respiratory data collected during the final minute of each test stage was averaged and used to calculate the IVT. Specifically, $\dot{V}_E/\dot{V}O_2$ was plotted as a function of $\dot{V}O_2$ and the level of $\dot{V}O_2$ at which $\dot{V}_E/\dot{V}O_2$ was the lowest corresponds to the IVT (Baldari & Guidetti, 2001, see Figure 2 for visual representation). The workload corresponding to the stage in which the IVT was found was used to quantify the recovery wattage.

Twenty minutes post-$\dot{V}O_{2max}$ subjects performed a WAnT. During the 20 min recovery period subjects cycled at 40 W and were permitted to consume fluids at will. In the last 5 min of the recovery period subjects were allowed to dismount the bicycle ergometer and stretch. Subjects then transferred to the Monark (Ergomedic 874E, Monark Exercise AB, Sweden) bicycle and the seat and handle bar height were adjusted and recorded for subsequent use. Subjects were instructed to start from a "rolling" start, cycling at a light cadence (approximately 60-80rpm) against no resistance. They were then given a 10 s and 5 s warning for the commencement of the test. The SMI POWER software (SMI, USA) was set to have a 3 s count-down which was audibly counted for subjects. Subjects were instructed to achieve maximum pedal cadence by the end of the 3 s count and at this time the pan loaded resistance (0.09 kg·(kg body mass)$^{-1}$) was applied. Subjects were instructed to remain seated and to attempt to maintain maximum pedal cadence throughout the entire 30 s period. At the completion of the WAnT subjects were allowed to cycle at a self selected resistance on either bicycle ergometer to allow for venous blood return and prevent blood pooling as well as alleviate any discomfort associated with the cessation of high-intensity exercise.

3.4.2 Day 2: Time to Exhaustion Test

On Day 2, after a warm-up (10 min at a workload corresponding to 50% of $\dot{V}O_{2max}$), subjects performed a TTE ride at an intensity corresponding to 120% MAP. The same bike used for the $\dot{V}O_{2max}$ test was used for this ride and later exercise trials (Egrometrics 800, SensorMedics, USA). Subjects were given verbal encouragement throughout the task and were instructed to maintain a cadence over 60 rpm. A verbal warning was given when their cadence fell below 60 rpm and they were allowed a few seconds to bring their cadence back up. Exhaustion was operationally defined as an inability to maintain a cadence above 60 rpm, and subjects were told they could receive up to three warnings before the test would be terminated.

However, in practice all but one subject were unable to bring their cadence back above 60 rpm after the initial decline and warning. Sixty percent of the TTE was then used for the duration of the subjects' work intervals on the following familiarization and trial days.

3.4.3 Day 3: Familiarization Tasks

For Day 3 subjects performed familiarizations of the sprint and work tasks in order to ensure that they could complete the required intensities and to improve their ability to reproduce the test protocol (Le Panse et al., 2005). After an equivalent warm-up to Day 2, followed by a brief transition to the Monark (Ergomedic 874E, Monark Exercise AB, Sweden) bicycle, subjects performed the sprint familiarization. Identical instructions to the WAnT were given with respect to the start and countdown. Subjects performed five 10 s sprints against a resistance of 0.09 kg·(kg body mass)$^{-1}$, with 30 s of recovery between sprints. During the 30 s recoveries subjects were instructed not to pedal for the initial 20 s, but were allowed to pedal for the final 10 s of recovery leading into the ensuing sprint. Post-sprint task subjects performed 20 min of light cycling at a self-selected resistance, between 40-100 W, before performing a familiarization trial of the exercise task. The familiarization trial consisted of three square wave bouts at 120% MAP for 60% of the TTE, at that same workload, with 5 min of cycling at 50 W between bouts at any cadence, provided it was greater than 60 rpm.

3.4.4 Days 4 to 6: Testing

Testing days (Day 4, 5, and 6) were comprised of a similar protocol to the familiarization trial, the only difference being the randomized recovery intensity (i.e. AR, PR, or CAR) over the three days. Subjects reported to the laboratory rested and hydrated, two- to three- hours post-partum. After an identical warm-up to the previous data collection days, subjects were given a short period of time (3-5 min) to allow them to recuperate, mentally prepare, as well as fit the mouth piece for respiratory data collection, before commencing the exercise trial. The exercise trial was similar to the familiarization trial with respect to duration and work intensity, with the recovery intensity varying in addition to the addition of a third recovery period of 14 min that separated the exercise trial from the sprint task (discussed below). The 5 min recoveries were as follows on the respective test days:

- PR – subjects remained seated and stationary on the bicycle ergometer, but were permitted to cycle against negligible resistance for five to ten revolutions to prevent blood pooling and discomfort every minute.
- AR – subjects cycled at an intensity equivalent to the 50% of the difference between the IAT and the IVT (Δ_T) below the IVT ($IVT_{-50\%\Delta_T}$). That is, the corresponding workloads

for the IAT and IVT (W_{IAT} and W_{IVT}, respectively) were used to calculate the workload at $IVT_{-50\%\Delta T}$ ($W_{IVT-50\%\Delta T}$) from the following equation:

$$W_{IVT-50\%\Delta T} = W_{IVT} - \frac{1}{2}(W_{IAT} - W_{IVT})$$

The use of $IVT_{-50\%\Delta T}$ to determine recovery intensity has been previously shown to be the optimal intensity for La^- clearance in soccer players and triathletes during treadmill running exercise when compared against three other single intensity recoveries (Baldari et al., 2004; Baldari et al., 2005). Thus, it was selected as the intensity quantification for this investigation. Evidence exists that recovery intensities for La^- clearance between cycling and running exercise may be different (Belcastro & Bonen, 1975; Bonen & Belcastro, 1976; Davies et al., 1970; Hermansen & Stensvold, 1972). However, the $IVT_{-50\%\Delta T}$ intensity relative to $\dot{V}O_{2max}$ was 39.3 ± 6.8% and 51.1 ± 4.9% for the soccer players and triathletes, respectively, which fall within the range of 30-70% $\dot{V}O_{2max}$ previously reported to be optimal for La^- clearance. Additionally, an investigation by Boileau et al. (1983), showed that recovery intensity for La^- clearance did not significantly differ between cycling and running exercise, and therefore the $IVT_{-50\%\Delta T}$ intensity was deemed appropriate for this investigation.

- CAR – subjects cycled at an intensity equivalent to the IAT for 2 min and then the $IVT_{-50\%\Delta T}$ for the remaining 3 min.

Blood La^- measures were taken immediately after the first two SE bouts and just before the second and third exercise bouts (see Figure 3).

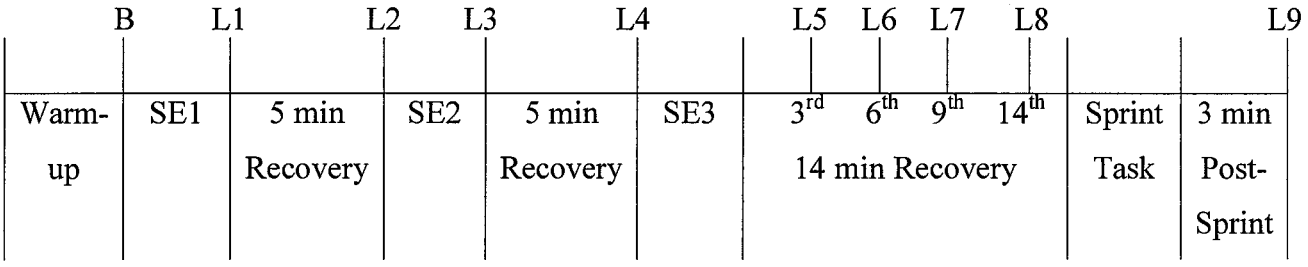
Following the third exercise bout the subjects recovered for 14 min before performing the sprint task. This recovery was performed at the same intensity(s) as that performed between the SE bouts. During the PR trial subjects were allowed to dismount the bicycle, but were required to remain seated for the initial 8 min of the recovery period. During the eighth minute subjects remounted the bicycle so that for the final 5 min of recovery they could ride at their $IVT_{-50\%\Delta T}$. This was done so that subjects did not begin the sprints cold. Additionally this effectively started subjects with similar cardiac outputs for all recoveries as the ended as the same exercise intensity. For the AR trial subjects cycled for the full recovery duration at their $IVT_{-50\%\Delta T}$. In the case of the CAR trial the subjects cycled at their IAT intensity for 5 min and at their $IVT_{-50\%\Delta T}$ for the remaining 9 min. In all trials post-recovery 2 min were allotted to allow subjects to transition to the Monark (Ergomedic 874E, Monark Exercise AB, Sweden) bicycle. Therefore, in actuality, subjects underwent 14 min of the respect AR intensity, with an additional 2 min of

recovery to allow the bike transition. Approximately fifteen minutes of recovery was selected because it is the typical intermission period for most intermittent sports (e.g. hockey, basketball, soccer). During the recovery after the third exercise bout blood La^- measures were made at the 3^{rd}, 6^{th}, 9^{th}, and 14^{th} min.

The final procedure on the testing days was the sprint task. Subjects performed five 10 s sprints against a resistance of 0.09 kg·(kg body mass)$^{-1}$, with an identical protocol and instructions to the familiarization sprint task. Three minutes post-sprint task a final blood La^- measure was taken. After the sprints subjects were recommended to perform and additional recovery to help return the body to homeostasis and relieve any discomfort associated with the intense sprint tasks. However, this was not mandatory.

Heart rate was recorded throughout the testing procedure, during both the exercise bouts and recovery intervals, as well as during the sprint task. Samples were taken every 5 s during exercise, and sample points that temporally corresponded to the closest La^- sample time were used for analysis (i.e. HR1-9). In addition, a HR measure was made at the start of first SE bout and used as a baseline value (HRB). During the sprint task the maximum HR achieved after each individual sprint was selected for analysis. Ventilatory parameters were recorded throughout the exercise bout and recovery periods, but not for the sprint task. These measurements were sampled breath-by-breath and averaged over 20 s. Again, the 20 s average that corresponded temporally closest to the La^- sample time was selected for analysis (i.e. $\dot{V}O_2$1-8, since ventilatory parameters were not measured after the 14 min recovery period). Similar to the HR measures an additional baseline measure was taken ($\dot{V}O_2$B). Figure 3 visual depicts the temporal layout of the HR and $\dot{V}O_2$ sample times.

Figure 3. Schematic Representation of the Testing Day Protocol



B, baseline HR and $\dot{V}O_2$ sample time; L1-9, La^- (HR and $\dot{V}O_2$) sample times from post-SE1 to 3 min post-sprint task (no $\dot{V}O_2$ measure was made for the L9 time point)

3.5 Data Analysis

The dependent variables that were measured in this investigation were: Blood La^- Concentration ($mmol \cdot L^{-1}$), PP Output (W), MP Output (W), FI (%), and TW (J). Additionally HR (bpm) and $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) were recorded. Blood La^- comparisons were made using a group (3) by time (9) repeated-measures analysis of variance (ANOVA). Similarly, a group (3) by time (5) repeated-measures ANOVA was used to compare parameters from the sprint tests (i.e. PP and MP outputs and FI). A one-way repeated-measures ANOVA was used to compare TW in the three treatment groups. Heart rate and $\dot{V}O_2$ during the exercise and recovery bouts were assessed using group (3) by time (10 and 9, respectively) ANOVA's. While HR during the sprint task was assessed using a group (3) by time (5) repeated-measures ANOVA. Statistical significance was set *a priori* at a level of $p \leq 0.05$. When the omnibus F-test showed a significant interaction effect dependent T-tests were used *post hoc* to determine where differences were, since the repeated-measures within-subjects design for ANOVA in SPSS version 15.0 (SPSS Inc., USA) does not produce *post hoc* tests. Significant omnibus F-tests for the main effects were followed up with pairwise comparisons to determine where differences occurred. The Bonferroni adjustment was made to account for multiple comparisons.

All values are reported as means ± standard deviations ($\bar{X} \pm SD$). Statistical analyses of ANOVA were performed using SPSS version 15.0 for Windows (SPSS Inc., USA) and T-tests were performed with Microsoft Excel Version 5.1.2600 (Microsoft Corporation, USA).

CHAPTER IV: RESULTS

4.1 Subject Characteristics

Twelve trained male cyclists volunteered to participate in this investigation. All subjects completed all of the testing protocols. However, four La^- values were not obtained due to complications measuring the samples in two subjects. In Subject 2 the PR L3, AR L1, and CAR L2 values are missing. In Subject 11 the PR L4 value is missing. Therefore, in the initial analysis these subjects were excluded from the blood La^- ANOVA, since the default setting for SPSS (SPSS Inc., USA) is to do a listwise deletion for missing data. Since statistical significance was achieved, despite the smaller sample size, no measures were taken to replace the missing data. Additionally, HR data from Subject 10 on the AR day and Subject 12 on all days was not able to be uploaded due to technological difficulties. Furthermore, HR could not be electronically monitored in Subject 11 due to a morphological anonmaly. Therefore, subjects 10-12 were excluded from the HR analysis. Subject's characteristics and workload and recovery characteristics are presented in Table 1 and 2, respectively.

4.2 Blood Lactate

On each of the Testing Days (i.e. Day 4, 5 and 6) nine blood La^- samples were taken. Sample times were: post-exercise bout 1 (L1), pre-exercise bout 2 (L2), post-exercise bout 2 (L3), pre-exercise bout 3 (L4), 3 min post-exercise bout 3 (L5), 6 min post-exercise bout 3 (L6), 9 min post-exercise bout 3 (L7), 14 min post-exercise bout 3 (L8), and 3 min post-sprint (L9, see Figure 3 for visual description).

Subject 2 and 11 both had missing blood La^- values and are therefore excluded from this analysis. The ANOVA revealed a significant interaction (group × time) effect for the blood La^- data ($p \leq 0.001$). Thus, the effects of the recovery group on blood La^- differed depending on the sampling time. Larger decreases in blood La^- were observed in the AR and CAR protocols compared to the PR protocol. This effect was more evident in the recovery portion of the protocol (i.e. L5-8, see Figure 4). Mean differences from L5 to L8 were 4.2, 6.7, and 6.5 $mmol \cdot L^{-1}$ for PR, AR and CAR, respectively. Targeted dependent T-tests were performed for the recovery and post-sprint blood La^- values (i.e. L5-9). T-test showed that during PR blood La^- values were significantly greater from the L6-8 sample times when compared to AR ($p \leq 0.05$). Furthermore, PR blood La^- values were significantly greater than CAR values for all recovery samples (i.e. L5-8, $p \leq 0.05$). However, the final blood La^- values post-sprint (L9) in both AR and CAR were not statistically different from PR ($p > 0.05$, see Table 3). Differences between the two active recoveries were not significantly different, despite the lower blood La^- values throughout

the CAR from L5-8 ($p>0.05$). Post-sprint (L9) blood La^- was highest in the PR, followed by AR and then CAR (see Table 3). However, none of these differences were statistically significant ($p>0.05$).

Table 1. Subject Characteristics

Subject	Age (yrs)	Height (cm)	Weight (kg)	So5S (mm)	$\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	PP ($W \cdot kg^{-1)}$	MP ($W \cdot kg^{-1}$)
1	29.5	176.0	72.7	29.5	71.2	13.7	8.5
2	21.8	181.5	68.1	31.5	76.5	15.5	11.2
3	28.8	185.6	77.9	54.4	58.9	13.6	10.2
4	29.6	182.2	69.8	25.0	75.2	15.2	9.1
5	27.0	172.2	75.1	41.2	58.1	14.5	9.6
6	27.1	188.0	87.9	40.1	57.2	16.5	10.6
7	34.0	181.0	65.3	34.1	65.4	16.8	9.7
8	30.1	169.6	68.5	45.8	59.5	14.3	9.9
9	23.8	186.4	72.0	28.5	61.1	13.7	8.5
10	21.2	181.0	71.6	39.9	61.5	14.1	9.3
11	25.0	180.3	69.8	43.5	65.7	15.1	9.4
12	30.9	193.5	89.4	34.3	63.8	15.6	9.0
$\bar{X}$	27.4	181.4	74.0	37.3	64.5	14.9	9.6
SD	3.9	6.7	7.6	8.4	6.6	1.1	0.8

So5S, sum of five skinfolds; PP, peak power (on WAnT, Wingate Anaerobic Test); MP, mean power (on WAnT); $\bar{X}$, mean value; SD, standard deviation

The main effect for (treatment) group was also significant ($p \leq 0.001$). Pairwise comparisons demonstrated that both AR and CAR groups resulted in significantly lower blood La^- values compared to the PR group ($p \leq 0.05$ and $p \leq 0.001$, respectively for the former and latter).

Table 2. Subject Workload and Recovery Characteristics

Subject	120% MAP (W)	60% TTE (s)	IAT (W)	$\dot{V}O_{2IAT}$ (% max)	$IVT_{-50\%\Delta T}$ (W)	$\dot{V}O_{2IVT-50\%\Delta T}$ (% max)
1	540	76.5	270	69.6	180	47.7
2	576	72.4	330	82.3	240	61.7
3	396	111.2	270	76.3	180	54.4
4	504	52.4	240	73.9	195	52.0
5	468	91.4	210	65.1	120	44.7
6	540	89.4	270	66.1	180	55.5
7	432	76.8	240	75.9	105	43.1
8	396	75.4	240	75.5	150	51.3
9	468	81.9	210	66.7	120	46.9
10	432	74.5	240	67.6	150	51.3
11	504	72.9	270	75.9	135	45.0
12	612	127.6	390	83.1	165	49.0
$\bar{X}$	489.0	83.5	265.0	73.2	160.0	50.2
SD	69.4	19.7	50.9	6.1	38.0	5.3

MAP, maximum aerobic power; TTE, time to exhaustion; IAT, individual anaerobic threshold; $IVT_{-50\%\Delta T}$, 50% of the difference between the IAT and IVT (individual ventilatory threshold) below IVT

The sphericity assumption was violated for the main effect of (sample) time and therefore the Greenhouse-Geisser correction was applied. The main effect of time on blood La^- values was significant ($p \leq 0.001$). This demonstrated that the work and recovery protocol intensities were sufficient to induce statistically significant increases in blood La^- as well as being appropriate for clearance. Pairwise comparisons revealed where the difference existed between sample times and Table 4 presents the differences. Blood La^- levels increased throughout the exercise protocol and peaked at L5 before decreasing during the recovery portion of the exercise protocol. Lactate levels where again elevated post-sprint task (L9) to an overall maximum across all nine sample times.

Table 3. Mean Blood Lactate Values

Blood Lactate Sample ($mmol \cdot L^{-1}$)	PR	AR	CAR
L1	5.6 ± 1.6	5.6 ± 1.8	5.4 ± 2.2
L2	8.9 ± 2.0	7.8 ± 1.3	8.2 ± 1.3
L3	12.1 ± 2.1	9.2 ± 2.7	10.6 ± 2.4
L4	12.0 ± 3.0	9.9 ± 2.2	10.0 ± 2.1
L5	14.4 ± 1.8	13.6 ± 2.6	12.4 ± 2.2*
L6	13.8 ± 1.8	10.7 ± 2.7*	10.3 ± 3.0*
L7	13.4 ± 3.6	9.6 ± 3.2*	9.2 ± 3.1*
L8	10.3 ± 3.1	6.9 ± 2.5*	5.9 ± 2.6*
L9	14.6 ± 1.6	13.8 ± 2.3	13.6 ± 2.4
$\bar{X}$	11.7 ± 3.6	9.7 ± 3.5†	9.5 ± 3.5‡

n = 10, Subjects 2 and 11 are excluded; T-tests were only performed for recovery La^- (i.e. L5-9); L1-9, blood La^- sample times (see Figure 3)

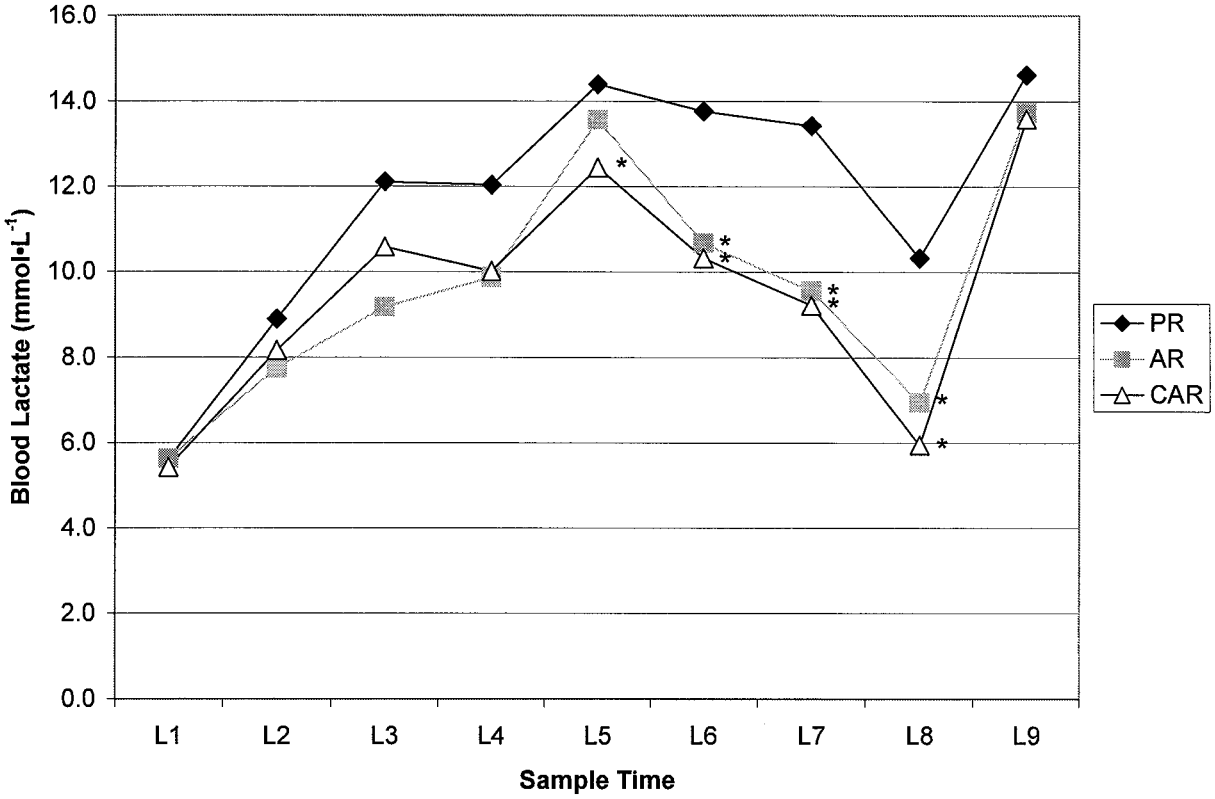
* T-tests significantly different from PR value ($p \leq 0.05$); † Pairwise comparison significantly different from PR ($p \leq 0.05$); ‡ Pairwise comparison significantly different from PR ($p \leq 0.001$)

Table 4. Mean Blood Lactate Differences for Sample Time

Sample Time	Mean Blood Lactate ($mmol \cdot L^{-1}$)	Significantly Different From ($p \leq 0.05$)
L1	5.6 ± 1.8	L3-7, L9
L2	8.3 ± 1.6	L3-6, L9
L3	10.6 ± 2.6	L1-2
L4	10.6 ± 2.6	L1-2, L5, L8-9
L5	13.5 ± 2.3	L1-2, L4-8
L6	11.6 ± 2.9	L1-2, L5, L8-9
L7	10.7 ± 3.7	L1, L5, L8-9
L8	7.7 ± 3.3	L4-9
L9	14.0 ± 2.1	L1-2, L4, L6-8

n = 10, Subjects 2 and 11 are excluded

Figure 4. Mean Blood Lactate Values



n = 10, Subjects 2 and 11 are excluded

* Significantly different from PR value ($p \leq 0.05$)

Comparing the ten subjects included in the blood La^- statistical analysis, eight reached their overall (i.e. among all three testing days) maximum blood La^- value on the PR day. The other three overall maximum values occurred on the AR day, as one subject shared the same maximum value on both the AR and PR days. Of the two subjects excluded from the analysis, one was the only subject to have their overall maximum blood La^- on the CAR day. The other excluded subject followed the group trend, having his highest overall value on the PR day. Daily maximum blood La^- values typically occurred at the third minute of recovery (L5), with 5, 8, and 6 of the subjects having their daily maximum La^- at L5 for PR, AR, and CAR days respectively. In general, the overall highest post-sprint blood La^- (L9) predominantly occurred on the PR test day, with 7 of 10 subjects peaking on this day. One subject peaked post-sprint on the AR day and three peaked on the CAR day (with one subject having the same L9 La^- value on both the PR and CAR days). Of the excluded subjects, Subject 2 had the same maximum L9 value on the PR and CAR day, while Subject 11 had their overall L9 maximum on the PR day.

The L1-L4 scores were excluded from the determination of minimum La^- values as these samples were taken during the fatiguing exercise bout and the interest for lowest La^- values was within the 14 min recovery period. The majority of subjects had their lowest overall La^- values on the CAR day (7 of 10). The other three subjects had their lows on the AR day. Subject 2 and 11 had their overall minimum La^- values on the PR and CAR day, respectively. Overall minimum La^- values were observed at L8 in all subjects (n = 10). Subject 2 (excluded) was the only participant to have an overall minimum La^- value at a sample time other than L8 (i.e. L7). On the CAR day all subjects (n = 10) had their daily minimum La^- value at the L8 sample. Nine subjects had their daily low at L8 and one at L7 on the PR day. Eight subject's daily lows occurred at L8 and two at L7 on the AR day. Subject 11 had his daily La^- minimum at L8 on all days, while Subject 2 had his daily lows at L7 on the PR and AR days and L8 for the CAR day.

Of the subjects included in the La^- ANOVA, none experienced their overall La^- minimum on the PR day, while the majority of maxima (i.e. 8) were achieved on this day. However, Subject 2 (excluded from ANOVA) was an exception to this, as he experienced his overall La^- minimum of the PR day. Conversely, no subjects experienced an overall La^- maximum on the CAR day, while the majority of La^- minima occurred on this day. Again, Subject 2 was the exception to this, as he was the sole participant to achieve his overall La^- maximum on the CAR day. Subject 9 was the only subject to experience their overall maximum and minimum La^- values on the same day. This occurred on the AR day. Of the eight subjects that achieved their overall maximum La^- value on the PR day, six also had their overall final (i.e. 3 min post-sprint) maximum La^- value too. Subject 7 was the only participant to achieve his overall final maximum La^- on the AR day, and interestingly, also had his overall minimum on this day just prior to the sprint task. On the CAR day, three subjects achieved their overall final maximum La^- value after previously having their overall minimum La^- value at L8. Additionally, Subject 2 achieved his overall final maximum La^- score on the CAR day (which was the same on the AR day), but did not experience an overall minimum at L8. Table 5 summarizes the daily and overall maximum and minimum La^- values for all subjects.

4.3 Sprint Task Performance

Subjects performed five 10 s sprints, each separated by 30 s of recovery. Each individual sprint was assessed for PP, MP and FI. In addition, the performance across the sprint task was assessed by analyzing TW across all five sprints. All subjects were able to complete all five sprints on each test day. Group means for all sprint task performance variables are presented in Table 6.

Table 5. Group Means for Peak Power, Mean Power, Fatigue Index and Total Work

	PP (W)	MP (W)	FI (%)	TW (J)
PR	1017.7 ± 134.4	782.2 ± 111.7	37.0 ± 9.2	39108.3 ± 4852.9
AR	1013.6 ± 145.6	800.1 ± 114.5*	34.2 ± 11.3	40003.3 ± 5110.2*
CAR	1021.8 ± 134.4	786.7 ± 118.0	37.4 ± 10.6	39335.8 ± 5022.6

* Significantly different from PR value ($p \leq 0.05$)

4.3.1 Peak Power

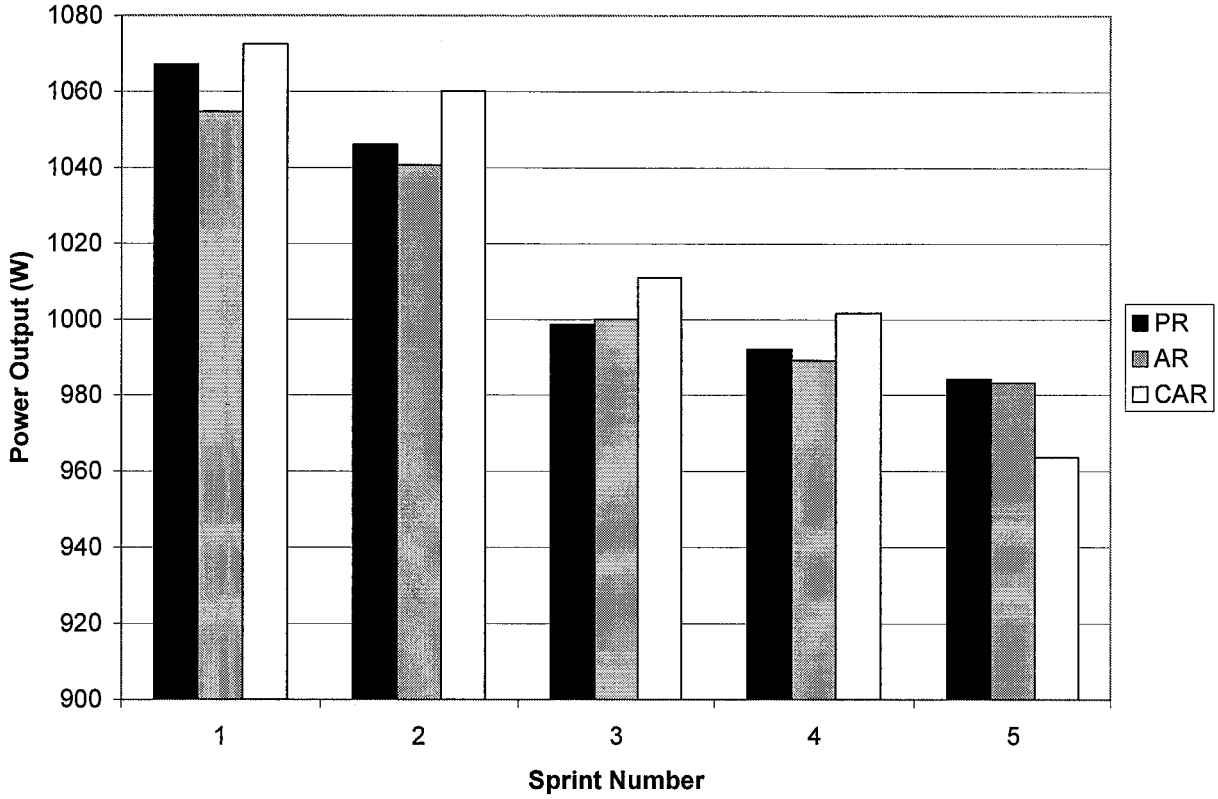
The interaction (group × time) effect for PP was not found to be significant ($p > 0.05$). Therefore, the effect of time on significantly decreasing PP did not change as a result of the group level (i.e. recovery mode). Peak power decreased in all three groups as the number of sprints performed increased. Table 6 shows the mean PP outputs and Figure 5 graphically depicts them.

Table 6. Mean Peak Power Outputs

Peak Power (W)	PR	AR	CAR
1	1067.2 ± 137.1	1054.8 ± 160.1	1072.5 ± 158.1
2	1046.1 ± 137.6	1040.7 ± 130.0	1060.2 ± 141.5
3	998.8 ± 121.1	1000.1 ± 152.1	1011.0 ± 126.2
4	992.1 ± 145.2	989.2 ± 151.1	1001.7 ± 143.9
5	984.2 ± 132.4	983.3 ± 143.9	963.6 ± 113.5

The group main effect for PP was not significant ($p > 0.05$). Subjects achieved similar PP outputs regardless of the recovery intensity. Nonetheless, the group mean PP output was greatest in the CAR group, followed by PR, then AR (see Table 5). The PP output was 4.1 W greater than the next closest group mean PP output for each group difference.

Figure 5. Mean Peak Power Outputs



A significant main effect of (sprint number) time on PP output was found ($p \leq 0.001$). Therefore, subjects were unable to maintain PP across the sprint task. Pairwise comparisons showed where the differences were (see Table 7). Peak power output was significantly greater on the first two sprints compared to the last three ($p \leq 0.05$). No difference in PP was observed between Sprint 1 and 2 or between the last three sprints ($p > 0.05$).

Table 7. Sprint Number Means for Peak Power, Mean Power, and Fatigue Index

Sprint Number	PP (W)	MP (W)	FI (%)
1	$1064.8 \pm 147.9^{*3-5}$	$864.5 \pm 108.7^{*2-5}$	$29.8 \pm 11.1^{*4-5}$
2	$1049.0 \pm 132.7^{*3-5}$	$825.7 \pm 99.7^{*1, 3-5}$	$34.8 \pm 10.2^{*4-5}$
3	$1003.3 \pm 130.1^{*1-2}$	$778.6 \pm 102.1^{*1-2, 4-5}$	$36.4 \pm 9.3^{*4-5}$
4	$994.3 \pm 135.4^{*1-2}$	$748.4 \pm 99.5^{*1-3}$	$39.6 \pm 9.4^{*1-3}$
5	$977.0 \pm 127.1^{*1-2}$	$730.9 \pm 101.8^{*1-3}$	$40.5 \pm 8.8^{*1-3}$

* Significantly different from listed sprints ($p \leq 0.05$)

In general the overall maximum PP outputs were achieved in either the first or second sprint. The exception was Subject 4, who shared the same overall maximum score for both his second and third sprint. The majority of overall maximum PP outputs were attained on the CAR day (8 of 12). Additionally, three overall maxima were attained on the AR day and one on the PR day.

As a general rule, most subjects achieved their highest daily PP output in either the first or second sprint. Three subjects achieved their daily maximum PP in the third sprint with one on the AR day and two on the CAR day (one subject shared the same score in the second and third sprint on the CAR day) and one in the fifth sprint on the PR day. The other daily maximum values were distributed between the first and second sprint (23 and 10, respectively). Seven daily maxima occurred in the first sprint on the PR and CAR days respectively, and nine in the first sprint on the AR day. Four daily maxima occurred in the second sprint on the PR and CAR days respectively, and two happened in the second sprint on the AR day.

Overall minimum PP outputs tended to occur on the PR and CAR day, with five on each respectively. The other two occurred on the AR day. In which sprint the overall minima occurred was more varied than the maxima and displayed no distinct trend. Seven arose in the fifth sprint, with one of these being shared with the second sprint score in Subject 3. Of the seven in the fifth sprint, three occurred on each of the PR and CAR day, respectively, and one on the AR day. The rest of the overall minimum PP outputs were spread over all the other sprints, with one in the first sprint, two in the second (one being a shared score), two in the third, and one in the fourth.

Daily minimum PP outputs tended to take place in the later sprints, but were evident throughout the sprint task. On the PR day six daily minima (two were shared) occurred in the fifth sprint, four in the fourth (one shared), three in the third, and one (shared) in the second sprint. Five occurred in the fifth sprint on the AR day, five in the fourth, and one in both the first and third sprints. On the CAR day, seven occurred in the fifth, one in the first, second and fourth sprint, and two in the third. Table 17 summarizes the daily and overall maximum and minimum PP outputs.

4.3.2 Mean Power

The interaction (group × time) effect for MP was not statistically significant ($p>0.05$). Thus, similar to the effect of time on PP, MP decreased in all three groups as the number of sprints performed increased. Table 8 shows the mean MP outputs, which are also illustrated in Figure 6.

Table 8. Mean Mean Power Outputs

Mean Power (W)	PR	AR	CAR
1	861.3 ± 102.9	872.3 ± 114.0	859.7 ± 118.0
2	820.3 ± 97.2	834.3 ± 106.1	822.7 ± 103.9
3	767.9 ± 100.6	791.1 ± 109.5	776.9 ± 103.8
4	738.3 ± 106.7	757.5 ± 99.2	749.5 ± 100.2
5	723.1 ± 104.1	744.9 ± 109.1	724.8 ± 99.6

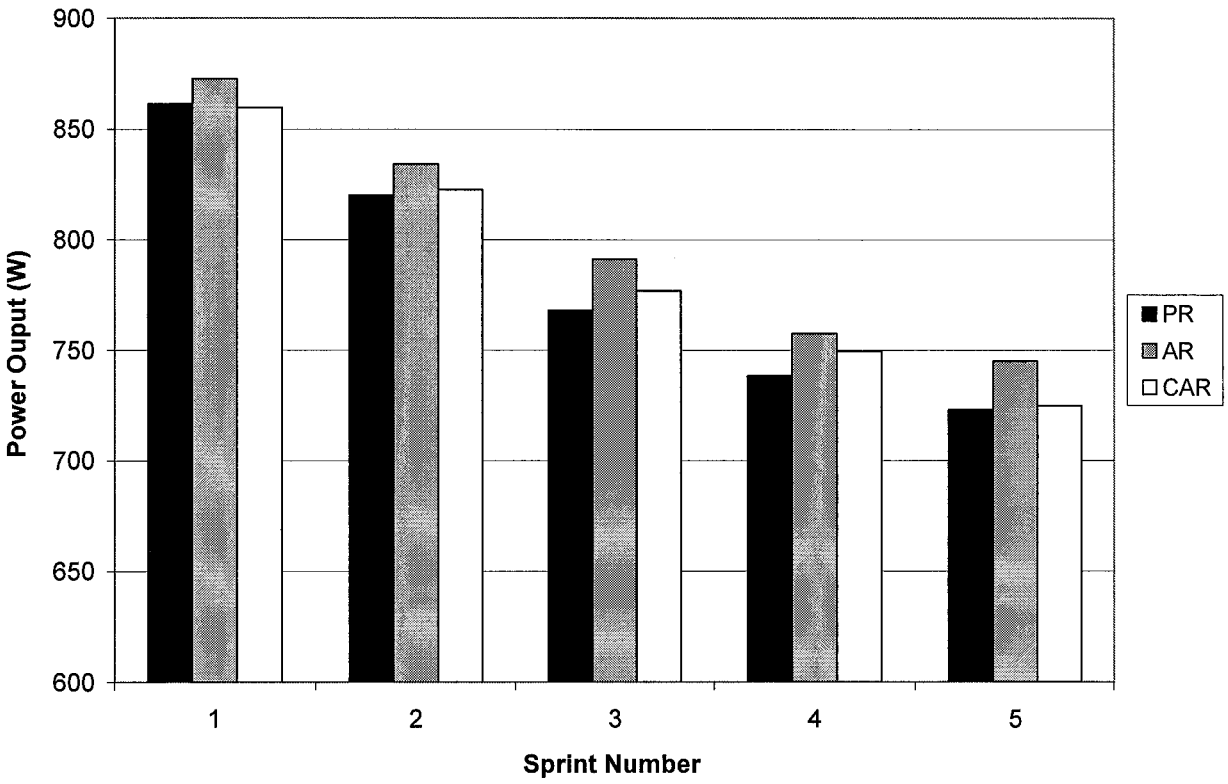
The main effect for group MP was statistically significant ($p \leq 0.05$). The group mean MP was highest on the AR day, followed by CAR, then PR (see Table 5). Pairwise comparisons showed that the significant differences were between the PR and AR MP outputs ($p \leq 0.05$). Neither of the other two pairwise comparisons (AR vs. CAR and PR vs. CAR) were significant ($p > 0.05$).

The sphericity assumption for the main effect of (sprint number) time was violated and therefore the Greenhouse-Geisser correction was applied. The main effect of time on MP outputs was significant ($p \leq 0.001$). Therefore, in addition to subjects not being able to maintain PP across the sprint task, they were also unable to maintain MP. Pairwise comparisons showed where the differences were (see Table 7). All MP outputs, except on the fourth and fifth sprint, were significantly different from all other sprints. The fourth and fifth sprint did not differ significantly from each other ($p > 0.05$), but were significantly less than the initial three sprints ($p \leq 0.05$). Mean power outputs decreased as the number of sprints completed increased. Each subsequent sprint was significantly less than the previous sprint ($p \leq 0.05$), except for the final two sprints.

The majority of overall maximum MP outputs were accomplished on the AR day. Seven overall maximum MP outputs were achieved on the AR day, three on the PR day, and two on the CAR day. All but one of these occurred in the first sprint, with the exception occurring in the second sprint. All daily maximum MP outputs, except for one, occurred in either the first or second sprint, with the vast majority (32 of 35) occurring in the first sprint. In the one exception the maximum transpired in the third sprint. On the PR day twelve of the daily maxima happened in the first sprint and one occurred in the second sprint. Subject 11 reproduced the same daily maximum value for the first and second sprint on the PR day. Ten daily maxima were achieved

in the first sprint on the AR day, one in the second, and one in the third. On the CAR day ten daily maxima were achieved in the first sprint and two in the second.

Figure 6. Mean Mean Power Outputs



On the PR day five of the overall minimum MP outputs occurred. Of these, two occurred in the fourth sprint and the other three in the fifth sprint. Only one overall minimum occurred on the AR day. It was obtained in the fourth sprint. Six overall minimum MP outputs occurred on the CAR day and they were all in the fifth sprint. On the PR day, three daily minimum MP outputs occurred in the fourth, and nine in the fifth sprint. Subject 1 shared the same daily minimum MP score on the first and fifth sprint for the AR day. Three subjects had their daily minimum in the fourth sprint and nine had it in the fifth. All but one subject had their daily minimum in the fifth sprint on the CAR day. The one anomaly experienced his daily minimum in the fourth sprint.

Subject 1 and 4 were the only participants to experience both their overall maximum and minimum MP output on the same day (i.e. on the CAR and PR days respectively). On the AR day there were five subjects that experienced their overall maximum MP in addition to also

achieving their overall final (i.e. fifth sprint) maximum MP. In total there were eight subjects that achieved their overall final maximum MP output on the AR day. Table 18 summarizes the daily and overall maximum and minimum MP outputs.

4.3.3 Fatigue Index

The interaction (group × time) effect for FI was not statistically significant ($p>0.05$). Fatigue index increased in all three groups as the number of sprints increased. The FI for all three treatment groups across the sprint times are shown in Table 9 and graphically depicted in Figure 7.

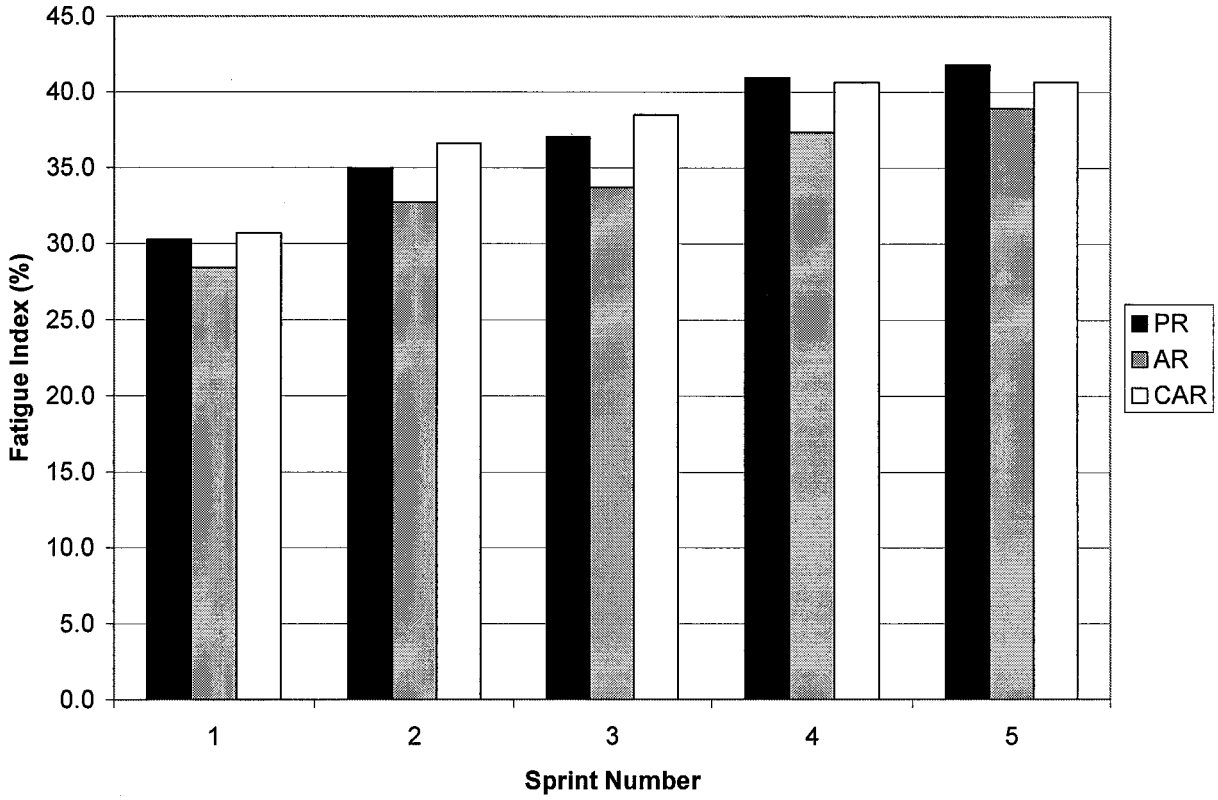
Table 9. Mean Fatigue Indexes

Fatigue Index (%)	PR	AR	CAR
1	30.3 ± 9.6	28.4 ± 12.5	30.7 ± 12.0
2	35.0 ± 10.4	32.7 ± 11.0	36.6 ± 12.0
3	37.0 ± 7.5	33.7 ± 10.3	38.5 ± 10.0
4	40.9 ± 7.4	37.3 ± 11.1	40.7 ± 9.7
5	41.8 ± 7.1	38.9 ± 9.9	40.7 ± 9.6

The main effect for group for FI was not found to be significant ($p>0.05$). The group mean for FI was greatest in the CAR protocol, followed by the PR then AR protocols. However, the differences were very minute (see Table 5).

The sphericity assumption for the main effect for (sprint number) time was violated and consequently the Greenhouse-Geisser correction was applied. The main effect for time was found to be significant ($p\leq0.001$). Fatigue index increased across the sprint task. Pairwise comparisons demonstrated where the differences occurred (see Table 7). The FI for the first three sprints were significantly less than the last two sprints ($p\leq0.05$). Conversely, there were no significant differences amongst the first three sprints or amongst the final two sprints. Thus, over the course of the sprint task, subjects developed larger differences between their maximum and minimum power outputs within a sprint.

Figure 7. Mean Fatigue Indexes



The trends for maximum and minimum data values for FI were less discernible than the other performance variables. The overall maximum FI were relatively evenly distributed among the three protocols. Four maxima occurred on the PR day, three on the AR day, and five on the CAR day. Of the four subjects who experienced an overall maximum FI on the PR day one was in the fourth sprint and the other three were in the fifth sprint. On the AR day all three occurred in the fifth sprint. Lastly, on the CAR day one occurred in the second sprint, three in the fourth, and one in the fifth. Daily maximum FI tended to occur in the later sprints, but several subjects experienced their maxima early on in the sprint task. During the PR trial, two subjects experienced their daily maximum FI in the second sprint, six in the fourth, and four in the fifth. On the AR day, two experienced their maxima in the first sprint, three in the fourth, and seven in the fifth. During the CAR protocol, one subject had their daily maximum in the first sprint, two in the second, one in the third, and four in each of the fourth and fifth sprint.

Four subjects experienced overall minimum FI on the PR day. Five subjects had their overall minimum FI on the AR day and four had theirs on the CAR day. Subject 3 had the same overall minimum FI on both the AR and CAR days. On the PR day, two of the overall minima

that occurred were in the first sprint and one occurred in each of the second and third sprints respectively. Three overall minimum FI occurred in the first sprint on the AR day, and one occurred in each of the second and third sprints respectively. On the CAR day the overall minima were divided between the first and second sprint, with two occurring in each.

The majority of daily minimum FI occurred in the first sprint, followed next by the second sprint. On the PR day, seven daily minima happened in the first sprint. In the second and third sprint there were three daily minima that occurred in each (Subject 2 had the same FI in both the second and third sprint). During the AR protocol, eight subjects had their daily minimum FI score in the first sprint. There were two subjects who had their daily minimum FI in the second sprint, one in the third, and one in the fifth. On the CAR day, nine subjects had their daily minimum FI in the first sprint and three had it in the second sprint.

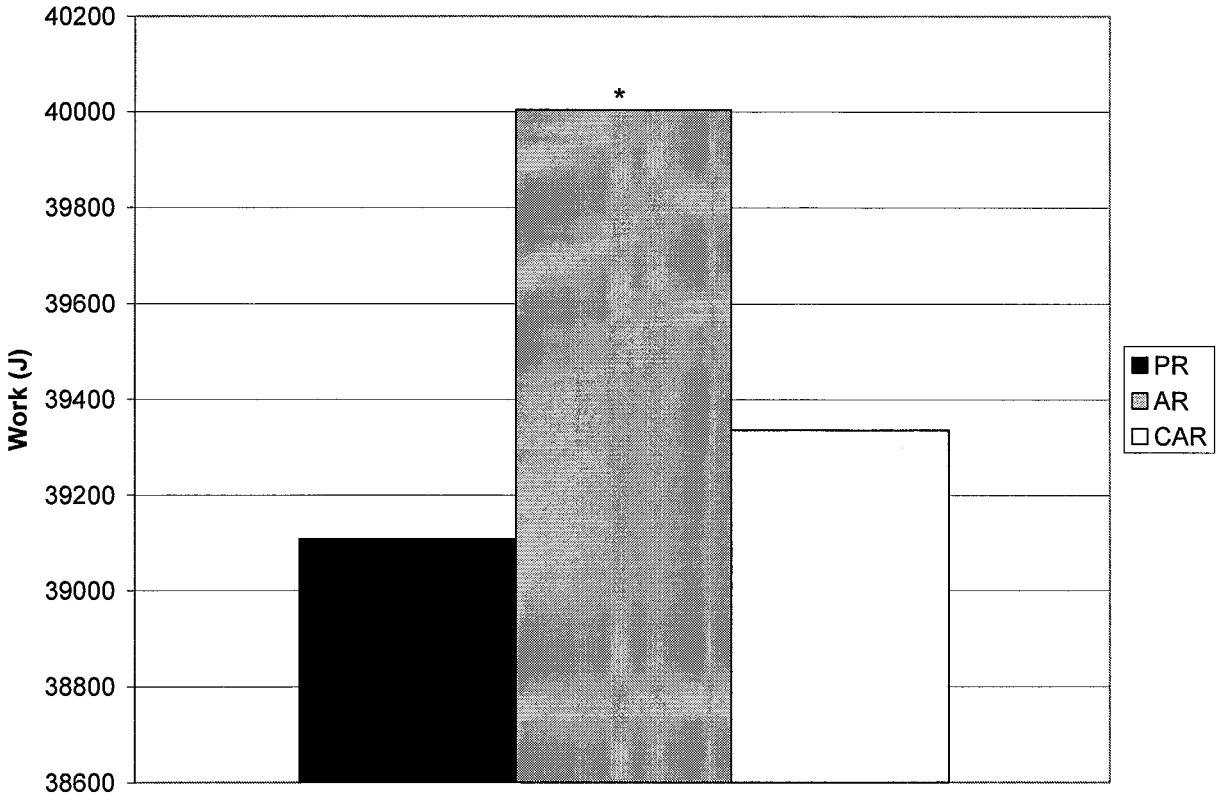
Overall initial and final maximum FI values were relatively evenly dispersed among the three testing days. Table 19 summarizes the daily and overall FI scores and in which sprint they occurred.

4.3.4 Total Work

The one-way repeated-measures ANOVA performed to compare the treatment effects on TW revealed the same results as the ANOVA MP group effect. This is because the TW score is simply an aggregate score of the MP across the sprint trial. Therefore, there were significant differences between the treatments with respect to TW ($p \leq 0.05$). Again, the pairwise comparison between AR and PR was found to be significant ($p \leq 0.05$). The mean values for TW for each group are shown in Table 5. Figure 8 graphically presents the data. The AR group mean TW was greatest, followed by the CAR, then the PR. No significant differences existed among AR and CAR, and PR and CAR pairwise comparisons ($p > 0.05$).

On an individual basis, seven subjects had their highest TW output on the AR day. Four subjects had their overall maximum TW output on the CAR day, and only one subject had their highest TW output on the PR day. Conversely, six subjects had their overall minimum TW output on the PR day, five did on the CAR day, and only one did on the AR day. Table 14 summarizes the maximum and minimum TW outputs for each subject.

Figure 8. Total Work



* Significantly different from PR value ($p \leq 0.05$)

4.4 Heart Rate

Heart rate was recorded during both the exercise and recovery bouts, as well as during the sprint task. During the exercise and recovery bouts HR measures were matched to the corresponding La^- sample points for analysis, with the addition of a baseline measure (HRB) prior to SE1. However, due to technological difficulties several HR files were not able to be uploaded for analysis, while in another subject a HR reading was not obtainable due to the subject's (Subject 11) chest morphology. Therefore the exercise and recovery HR ANOVA was comprised of nine subjects.

The maximum HR achieved after each individual sprint was selected for analysis of HR during the sprint task. In addition to the aforementioned technical difficulties in uploading HR data, two other HR data sets were excluded from analysis due to erratic values during the sprint task. Thus the sprint task HR (SHR) data set was comprised of seven subjects.

4.4.1 Exercise and Recovery Heart Rate

There was a significant interaction (group × time) effect for the HR results ($p \leq 0.001$). Heart rate responses varied differently over the three recovery intensities for the different sample times. Dependent T-tests revealed that HR was greater in both the AR and CAR compared to the PR from the HR2 sample time to HR7 ($p \leq 0.05$). Heart rate was greater in the AR trial compared to the CAR trial at HR4, but HR was then greater at HR5-6 in the CAR trial compared to the AR trial ($p \leq 0.05$). Mean HR values are shown in Table 10.

Table 10. Mean Exercise and Recovery Heart Rate Values

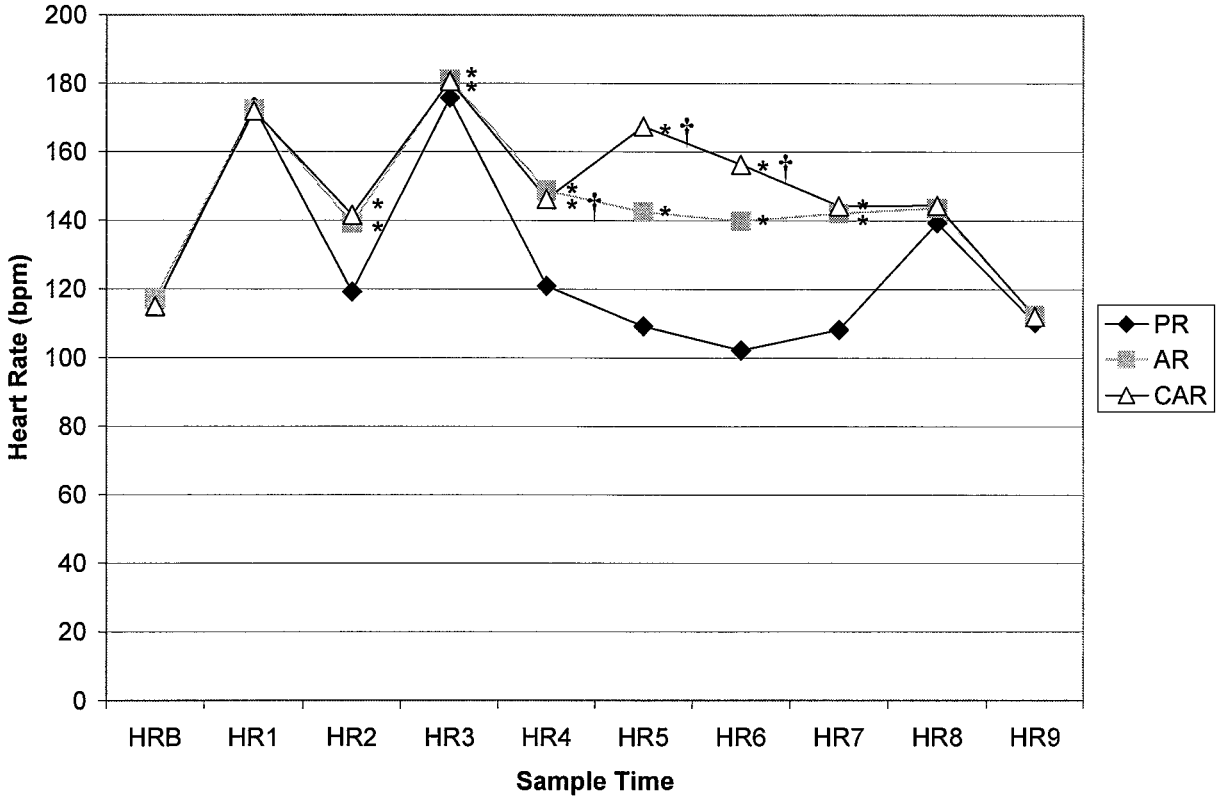
Heart Rate (bpm)	PR	AR	CAR
HRB	115.0 ± 11.0	117.2 ± 12.9	115.0 ± 8.5
HR1	173.0 ± 8.8	172.2 ± 10.0	171.9 ± 9.9
HR2	119.2 ± 10.1	139.1 ± 10.5*	141.6 ± 8.3*
HR3	175.7 ± 9.8	181.1 ± 10.1*	180.6 ± 11.4*
HR4	120.9 ± 10.1	148.7 ± 8.4*	146.2 ± 9.1*†
HR5	109.2 ± 9.7	142.4 ± 9.3*	167.3 ± 12.5*†
HR6	102.2 ± 8.1	139.4 ± 8.4*	156.3 ± 12.1*†
HR7	108.2 ± 8.9	142.1 ± 9.4*	144.2 ± 11.1*
HR8	139.2 ± 8.9	143.7 ± 11.3	144.4 ± 12.9
HR9	110.3 ± 9.0	112.3 ± 11.4	112.1 ± 6.3
$\bar{X}$	127.3 ± 27.0	143.9 ± 22.3	148.0 ± 23.5

n = 9, Subjects 10-12 were excluded; T-test were performed for all HR (i.e. HRB-9); HRB, HR-Baseline

* Significantly different from PR value ($p \leq 0.05$); † significantly different from AR value ($p \leq 0.05$)

The sphericity assumption for the group main effect was violated and therefore the Greenhouse-Geisser correction was applied. The main effect for group was significant ($p \leq 0.001$). Pairwise comparisons showed that group mean HR was significantly greater in the AR and CAR versus PR ($p \leq 0.001$), and in CAR versus AR ($p \leq 0.05$). Figure 9 graphically presents the mean HR values.

Figure 9. Mean Exercise and Recovery Heart Rate Values



n = 9, Subjects 10-12 are excluded

* Significantly different from PR value ($p \leq 0.05$); † significantly different from AR value ($p \leq 0.05$)

The main effect for time was found to be significant ($p \leq 0.001$). Pairwise comparisons revealed where the differences between sample times existed. Table 11 shows the sample time means and describes the differences and Table 21 presents individual daily and overall maximum and minimum HR values for the nine subjects evaluated.

Table 11. Mean Exercise and Recovery Heart Rate Differences for Sample Time

Sample Time	Heart Rate (bpm)	Significantly Different From ($p \leq 0.05$)
HRB	116.0 ± 10.6	HR1-8
HR1	172.4 ± 9.2	HRB, HR2-9
HR2	133.1 ± 13.8	HRB, HR1, HR3, HR9
HR3	179.1 ± 10.3	HRB-2, HR4-9
HR4	138.6 ± 15.6	HRB-1, HR3, HR7, HR9
HR5	139.9 ± 26.4	HRB-1, HR3, HR6-7, HR9
HR6	132.8 ± 24.9	HRB-1, HR3, HR5, HR8-9
HR7	131.6 ± 19.3	HRB-1, HR3-5, HR8-9
HR8	142.6 ± 10.8	HRB-1, HR3, HR6-7, HR9
HR9	112.0 ± 8.8	HR1-8

n = 9, Subjects 10-12 were excluded

4.4.2 Sprint Task Heart Rate

The interaction (group × time) effect for SHR reached statistical significance ($p \leq 0.05$). The increase in SHR across the sprint task differed depending on the treatment group. Dependent T-tests revealed that SHR was greater in both the CAR compared to the PR after the third and fourth sprint ($p \leq 0.05$). Mean SHR values are shown in Table 12.

Table 12. Mean Maximum Sprint Task Heart Rate

Heart Rate (bpm)	PR	AR	CAR
SHR1	167.3 ± 6.6	168.7 ± 5.8	168.0 ± 6.8
SHR2	173.1 ± 6.5	175.9 ± 6.5	176.4 ± 6.8
SHR3	175.0 ± 6.4	177.3 ± 7.7	178.1 ± 5.9*
SHR4	175.6 ± 5.1	178.6 ± 7.4	180.1 ± 7.4*
SHR5	176.1 ± 5.2	179.3 ± 7.3	179.4 ± 7.1
$\bar{X}$	173.4 ± 6.5	175.9 ± 7.6	176.4 ± 7.8

n = 7, Subjects 7 and 9-12 are excluded

* Significantly different from PR value ($p \leq 0.05$)

The group main effect for SHR was not significant ($p>0.05$). The group mean for SHR was greatest in the CAR protocol, followed by the AR then PR protocols. However, the differences were very small.

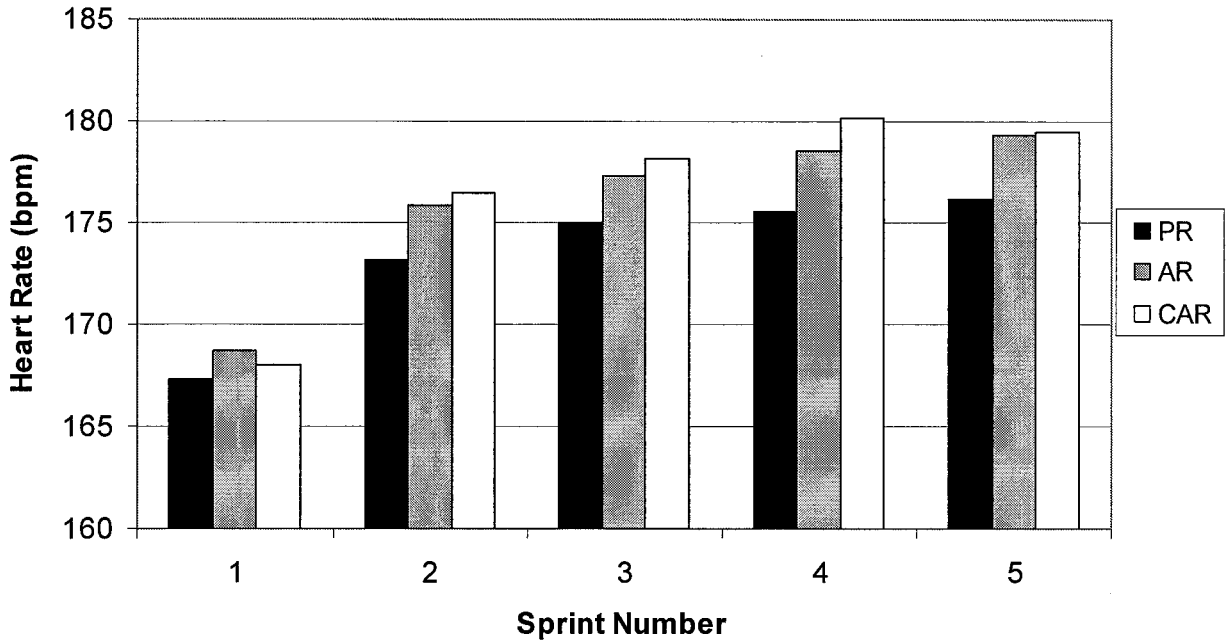
The main effect for time was found to be significant ($p\leq0.001$). Maximum HR values post-sprint increased across the sprint task. Pairwise comparisons demonstrated where the differences occurred (see Table 13). Mean SHR steadily climbed over the course of the sprint task. The SHR for the first sprint was significantly less than all other values ($p\leq0.05$). After the second sprint maximum HR was significantly greater than the first sprint, but less than the last two sprints ($p\leq0.05$). Heart rate after the third sprint was only significantly different form the SHR1 value ($p\leq0.05$). Heart rate after the last two sprints was significantly greater than after the first three sprints ($p\leq0.05$). The SHR for all three groups across the sprint times are graphically depicted in Figure 10. Table 22 presents individual daily and overall maximum and minimum SHR values for the seven subjects evaluated

Table 13. Mean Sprint Task Heart Rate Differences for Sample Time

Sprint Number	Heart Rate (bpm)	Significantly Different From ($p\leq0.05$)
SHR1	168.0 ± 6.1	SHR2-5
SHR2	175.1 ± 6.4	SHR1, SHR4-5
SHR3	176.8 ± 6.5	SHR1
SHR4	178.1 ± 6.7	SHR1-2
SHR5	178.3 ± 6.4	SHR1-2

n = 7, Subjects 7 and 9-12 are excluded

Figure 10. Mean Sprint Task Heart Rate



n = 7, Subjects 7 and 9-12 are excluded

4.5 Volume of Oxygen Consumed

The interaction (group × time) effect for the $\dot{V}O_2$ scores was significant ($p \leq 0.001$). Responses of $\dot{V}O_2$ to the exercise and recovery protocol varied differently over the three treatments across the sample times. Dependent T-tests revealed that $\dot{V}O_2$ in both the AR and CAR protocols was significantly greater than the PR protocol at the $\dot{V}O_2$2 sample, and from $\dot{V}O_2$4-7 ($p \leq 0.001$). Volume of O_2 consumed during CAR was greater than AR at the $\dot{V}O_2$5 and $\dot{V}O_2$6 sample times ($p \leq 0.001$). Mean $\dot{V}O_2$ was lower during the PR protocol from $\dot{V}O_2$B to $\dot{V}O_2$7, except at the $\dot{V}O_2$1 and $\dot{V}O_2$3 samples. At the $\dot{V}O_2$8 sample the mean $\dot{V}O_2$ during PR was actually significantly higher than during AR ($p \leq 0.05$), and greater than during the CAR ($p > 0.05$). Mean $\dot{V}O_2$ was higher in the CAR protocol than in the AR protocol at all measures except at baseline ($\dot{V}O_2$B) and pre-exercise bout 3 ($\dot{V}O_2$4). Mean $\dot{V}O_2$ values are shown in Table 14.

Table 14. Mean Volume of Oxygen Consumed Values

Volume of Oxygen Consumed ($mL \cdot kg^{-1} \cdot min^{-1}$)	PR	AR	CAR
$\dot{V}O_2B$	13.6 ± 3.0	15.1 ± 4.1	14.8 ± 3.9
$\dot{V}O_21$	56.5 ± 6.8	57.0 ± 7.1	57.8 ± 6.8
$\dot{V}O_22$	14.3 ± 4.9	36.5 ± 6.1*	37.7 ± 8.0*
$\dot{V}O_23$	58.9 ± 6.5	60.5 ± 7.4	61.6 ± 8.8
$\dot{V}O_24$	13.6 ± 4.0	38.6 ± 6.1*	38.2 ± 6.7*
$\dot{V}O_25$	10.6 ± 1.7	36.5 ± 6.6*	51.4 ± 9.1*†
$\dot{V}O_26$	8.0 ± 1.6	34.3 ± 6.4*	40.0 ± 8.2*†
$\dot{V}O_27$	11.1 ± 3.2	32.9 ± 6.6*	34.6 ± 6.8*
$\dot{V}O_28$	33.0 ± 6.4	31.7 ± 6.0*	31.9 ± 7.3
$\bar{X}$	24.4 ± 19.7	38.1 ± 14.2	40.9 ± 15.4

T-test were performed for all $\dot{V}O_2$ (i.e. $\dot{V}O_2B$-8)

* Significantly different from PR value ($p \leq 0.001$); † significantly different from AR value ($p \leq 0.05$)

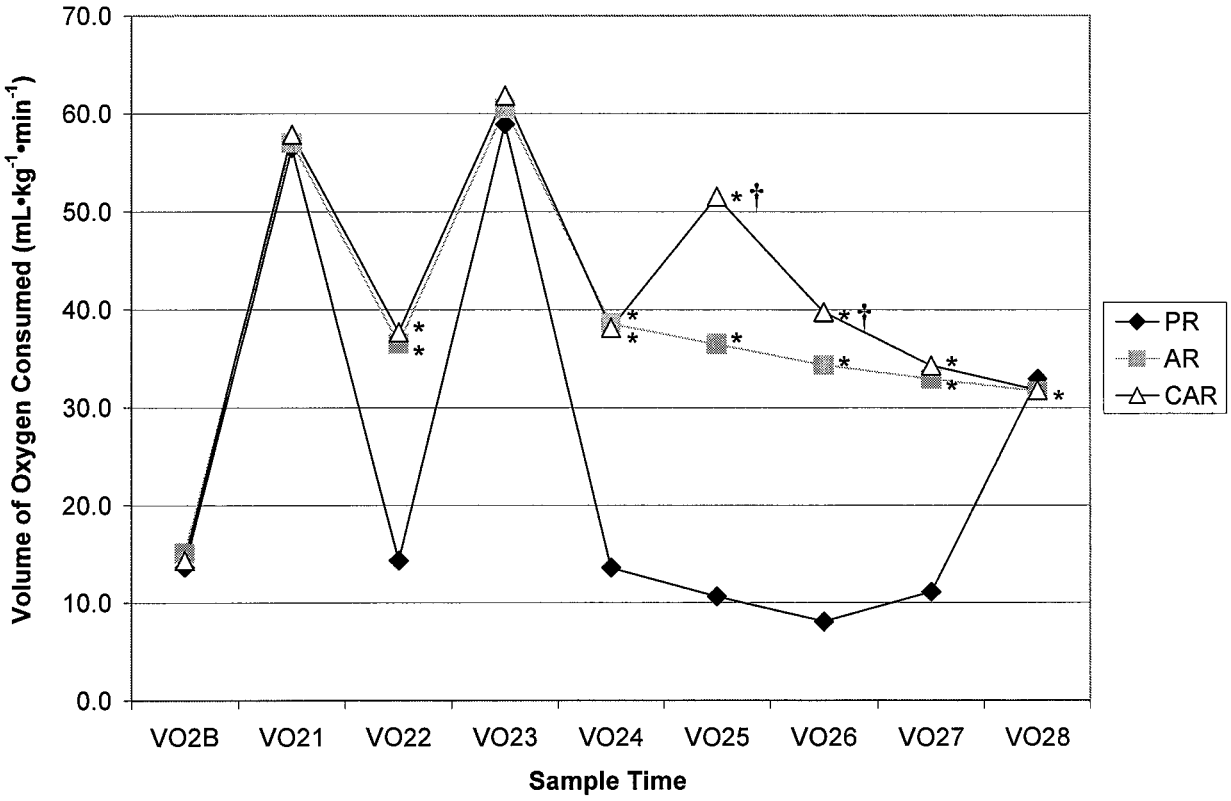
The sphericity assumption for the main effect of group was violated and therefore the Greenhouse-Geisser correction was applied. The effect was then found to be significant ($p \leq 0.001$). Pairwise comparisons showed that all three groups were significantly different with respect to group mean $\dot{V}O_2$ scores ($p \leq 0.001$). Volume of O_2 consumed was greatest in the CAR protocol, followed by AR then PR.

The sphericity assumption for the main effect for time was violated and consequently the Greenhouse-Geisser correction was applied. The $\dot{V}O_2$ main effect for time was found to be significant ($p \leq 0.001$). Pairwise comparisons revealed where the differences between sample times existed. Table 15 shows the sample time means and describes the differences. Figure 11 graphically presents the mean $\dot{V}O_2$ values. Table 23 presents individual daily and overall maximum and minimum $\dot{V}O_2$ values for all subjects.

Table 15. Mean Volume of Oxygen Consumed Differences for Sample Time

Sample Time	Mean Volume of Oxygen Consumed ($mL \cdot kg^{-1} \cdot min^{-1}$)	Significantly Different From ($p \leq 0.05$)
$\dot{V}O_2B$	14.5 ± 3.6	$\dot{V}O_2$1-8
$\dot{V}O_2$1	57.1 ± 6.7	$\dot{V}O_2B$, $\dot{V}O_2$2, $\dot{V}O_2$4-8
$\dot{V}O_2$2	29.5 ± 12.6	$\dot{V}O_2B$-1, $\dot{V}O_2$3, $\dot{V}O_2$5
$\dot{V}O_2$3	60.4 ± 7.5	$\dot{V}O_2B$-1, $\dot{V}O_2$4-8
$\dot{V}O_2$4	30.1 ± 13.1	$\dot{V}O_2B$-1, $\dot{V}O_2$3, $\dot{V}O_2$6-7
$\dot{V}O_2$5	32.8 ± 18.2	$\dot{V}O_2B$-4, $\dot{V}O_2$6-7
$\dot{V}O_2$6	27.5 ± 15.3	$\dot{V}O_2B$-1, $\dot{V}O_2$3-5, $\dot{V}O_2$8
$\dot{V}O_2$7	26.2 ± 12.2	$\dot{V}O_2B$-1, $\dot{V}O_2$3-5, $\dot{V}O_2$8
$\dot{V}O_2$8	32.2 ± 6.4	$\dot{V}O_2B$-1, $\dot{V}O_2$3, $\dot{V}O_2$6-7

Figure 11. Mean Volume of Oxygen Consumed Values



* Significantly different from PR value ($p \leq 0.05$); † significantly different from AR value ($p \leq 0.05$)

CHAPTER V: DISCUSSION

5.1 Subject Characteristics

The mean age (27.4 yrs) of the subjects tested in this investigation was similar to that of Stamford et al. (1981) and Dodd et al. (1984), but older than that of more recent investigations (Baldari et al., 2004; Baldari et al., 2005; Gmada et al., 2005). Furthermore, the subjects investigated displayed greater $\dot{V}O_{2max}$ scores than those reported by Stamford et al. (1981), Dodd et al. (1984), and Gmada et al. (2005), but slightly lower than the populations investigated by Baldari et al. (2004; 2005). This was expected as the two early investigations did not use trained populations. Interestingly, the subjects had $\dot{V}O_{2max}$ scores that were reasonably higher than the trained group examined by Gmada et al. (2005). This is attributed to the criteria utilized to determine training status in the respective investigations. However, the group did exhibit similar $\dot{V}O_{2max}$, threshold and MAP data to those reported in other groups of trained cyclists (Faria, Parker, & Faria, 2005; Tanaka et al., 1993).

Compared to Gmada et al. (2005) workloads at 120% MAP were substantially higher (489.0 ± 69.4 vs. 310.0 ± 14.0 W (trained) and 280.0 ± 15.0 W (untrained) respectively). The 60% TTE duration was shorter than the trained group (83.5 ± 19.7 vs. 102.0 ± 20.0 s), but longer than the untrained group (73.0 ± 21.0 s). These differences may be explained by differences in the parameters used to define training status, as well as differences in the protocol used to achieve $\dot{V}O_{2max}$. During the $\dot{V}O_{2max}$ protocol in the study by Gmada et al. (2005) subjects were required to pedal at a set cadence (60 rpm) which is suggested to be more economical, but lower than preferred cadences of trained cyclists (Marsh, Martin, & Foley, 2000). The only cadence restriction in the current investigation was that subjects could not drop below 60 rpm. This may have had some effect on the $\dot{V}O_{2max}$ and, more particularly, MAP scores. Though the major contributor to the differences is more likely directly related to fitness levels.

5.2 Blood Lactate

The changes observed in mean blood La^- during the recovery portion of the exercise bout were as anticipated. That is, mean blood La^- was lowest in the CAR trial at the end of the recovery period, albeit only slightly less than the AR trial. Mean blood La^- was always higher in the PR protocol. However, the differences observed between clearance rates in the AR and CAR protocols were non-significant, and thus it cannot be said with any certainty that chance alone could not account for the differences. Nonetheless, blood La^- concentration was lower at all sample points during the 14 min recovery period for CAR. Furthermore, the fact that blood La^- was lowest in the CAR trial at the L5 sample shows that La^- likely peaked fastest during CAR.

This is in agreement with the suggestion put forth by Stamford et al. (1981) and also agrees with the findings of Gmada et al. (2005).

Blood La^- almost always reached a minimum at the L8 sample time, with the exception of three cases in the ten subjects analyzed over all the testing days. This suggests that La^- values were still decreasing at the 14^{th} min of recovery. Analysis of the final La^- scores at the end of the recovery period renders this more plausible, as the values are still sufficiently above resting levels. However, due to the experimental protocol design, this was not to be investigated. Recently, Baldari et al. (2004; 2005) reported that during a 30 min recovery period, significant decreases in La^- did not occur after the 20^{th} min of recovery. However, decreases in La^- in their investigation were expressed as percentages, whereas the current investigation used absolute values. When expressed as percentages of maximum mean values, La^- values at the end of recovery are substantially higher (72, 51, and 48% for PR, AR and CAR respectively) in this study than those reported by Baldari et al. (2004; 2005) at 15 min of similar intensity recovery (approx. 20%, (2004); and 12-25% (2005)). It therefore follows that blood La^- was likely still significantly decreasing after 14 min of recovery and would require a longer recovery time to reach baseline levels after similar intensity work. Blood La^- values did however decrease to similar absolute values as those reported by Gmada et al. (2005). This difference is probably due to the type of exercise performed, as Gmada et al. (2005) used an intermittent exercise protocol which resulted in higher overall La^- values, similar to this investigation, compared to the single high-intensity bout employed by Baldari et al. (2004; 2005).

As such, the finding that blood La^- clearance was greater with an AR protocol is in agreement with numerous other investigations (Baldari et al. 2005, Dodd et al., 1984; Gmada et al., 2005; McAinch et al., 2004; Spierer et al., 2004; Siebers & McMurray, 1981; Stamford et al., 1981). Among all the subjects analyzed, blood La^- was always lowest across the three testing days in either the AR or CAR trial. Therefore, the findings are in complete agreement with the vast majority of findings that show blood La^- clearing faster with AR. Investigations that do not show improved La^- clearance with AR have used shorter recovery periods, much less than that of the present study (Dorado et al., 2004; Spencer et al., 2006). Such short durations appear to not allow enough time for La^- levels to decrease substantially.

It is now currently believed that the major fate of La^- is oxidation (Gladden, 2003). This is especially true during exercise, when as much as 75% of La^- is oxidized, with the remainder being disposed through gluconeogenesis (Brooks, 2007). During high-intensity exercise La^- is predominantly produced in the glycolytic muscle fibres and then is shuttled to adjacent and

remote oxidative muscle fibres, as well as other tissue sites, such as the heart and liver. During AR the maintenance of a lower metabolic power output, compared to the previously higher power output during exercise, provides an energy demand that is met by a large contribution of La^- metabolism (i.e. oxidation, Bangsbo et al., 1994; Rontoyannis, 1988). Additionally, the metabolic power demand results in higher cardiac output and hence, higher blood flow, which is believed to contribute to largescale La^- shuttling to more remote bodily locations. The collective outcome is a greater clearance of blood La^-.

5.2.1 Combined Active Recovery and Lactate Clearance

Stamford et al. (1981) initially suggested that a recovery intensity that decreased in relation to a decreasing blood La^- concentration may clear blood La^- faster than a single intensity recovery. Dodd et al. (1984) and Gmada et al. (2005) have previously examined this, with conflicting results. The current study is in agreement with the latter two, in that CAR was able to clear La^- at a rate at least equal to AR performed at a single intensity. However, results did not confirm Gmada et al.'s (2005) finding that CAR was able to clear La^- faster than AR in a trained population and to a lesser extent in an untrained population. Differences in findings between the current investigation and those of Gmada et al. (2005) could be related to subject fitness level and/or recovery intensity. The trained group in the study by Gmada et al. (2005) had a mean $\dot{V}O_{2max}$ of 56.5 ± 3.5 $mL \cdot kg^{-1} \cdot min^{-1}$, compared to 64.5 ± 6.6 $mL \cdot kg^{-1} \cdot min^{-1}$ in this study. Additionally, while recovery intensities in Gmada et al.'s (2005) work were based on individual thresholds, the workloads were determined relative to ventilatory thresholds minus 20% $\dot{V}O_{2max}$ (specifically VT1 and VT2). The authors selected these workloads because they would be roughly equivalent to 35% and 65% of $\dot{V}O_{2max}$; both which had been previously used for La^- clearance analysis in other investigation (Dodd et al., 1984; Stamford et al., 1981). Recovery intensities in this investigation were quantified relative to IAT and IVT, determined via blood La^- analysis and ventilatory data (respectively), and recently examined in a similar population for their effectiveness for La^- clearance (Baldari et al., 2005). Furthermore, we felt that recovery intensities based on the AT determined with blood La^- would be more accurate than solely from ventilatory data since the measure is more direct. It is important to have a recovery intensity that does not surpass the AT and result in a significant increase in La^- production in order to facilitate La^- clearance with AR (Dodd et al., 1984).

Relative to $\dot{V}O_{2max}$, recovery intensity in this study was higher than that of Gmada et al. (2005). Volume of O_2 consumed at VT2 was 61.0 ± 4.5 and 54.5 ± 6.0% $\dot{V}O_{2max}$, and at VT1 was 37.5 ± 5.0 and 33.5 ± 4.5% $\dot{V}O_{2max}$ for trained and untrained subjects, respectively.

Conversely, $\dot{V}O_2$ at IAT and $IVT_{-50\%\Delta T}$ in the present investigation were 73.2 ± 6.1 and 50.2 ± 5.3% $\dot{V}O_{2max}$, respectively. Accordingly, it may be that CAR did not result in significantly greater La^- clearance than AR because the IAT workload may have resulted in substantial production of La^- within the muscle as the workload was higher than Gmada et al. (2005), which may have limited clearance from the blood. Interestingly however, it was observed that La^- was lowest at the L5 sample point in CAR, so it seems that the IAT workload was at a sufficient intensity to clear La^- quickly enough to have an earlier peak. Despite the early peak, it seems that CAR did not incur a significant advantage to overall La^- clearance, above that obtain with AR.

It appears that the protocol used to determine the IAT in this investigation was adequate, as there was no evidence of an increase in blood La^- in the CAR trial. However, it may be that the workload was below the AT. Nonetheless, the workload could not have been much lower than the AT since subjects $\dot{V}O_2$ at IAT was sufficiently high. Therefore the treadmill protocol adopted from Baldari et al. (2000) and modified for use on a bicycle ergometer seems to be valid for determination of the IAT.

5.3 Sprint Task Performance

Performance results on the sprint task followed the hypothesized trends with respect to PP, but did not fulfill the other predictions. Across the three testing days, no significant differences were observed in PP output or FI. Mean power outputs was significantly greater in the AR group compared to the PR group. Additionally, TW was significantly greater during AR compared to PR.

5.3.1 Peak Power

Peak Power did not significantly differ amongst the recovery interventions. However, over the course of the five sprints, subjects fatigued and PP significantly dropped. As a trend among individuals, subjects tended to achieve their highest PP on the CAR day. These occurred in either the first or second sprint. Conversely, the highest PP in the final sprint tended to occur on the AR and PR days.

The finding that PP was not significantly affected by recovery in this investigation is in agreement with several other studies (Ainsworth et al., 1993; Spierer et al., 2004; Weltman, Stamford, Moffatt, & Katch, 1977). Ainsworth et al. (1993) demonstrated that as little as 6 min of AR at 80 W was sufficient to restore 5 s PP output on a 45 s bicycle sprint task. While Spierer et al. (2004) showed that PP on repeat 30 s WAnT in both trained and untrained subjects was not significantly different when interspersed with 4 min of either PR or AR at 28% $\dot{V}O_{2max}$. Based on these findings, it was hypothesized that 14 min of recovery (with an additional 2 min

transition period, totaling 16 min) would be more than adequate to allow complete restoration of PP output in this study. Then from this point the sprint task would be identical, regardless of the recovery trial, and thus no differences in PP were expected on the latter sprints.

Recent investigations have shown that AR can actually be detrimental to performance of sprint tasks when compared to PR (Dupont et al., 2004; Dupont, Moalla, Matran, & Berthoin, 2007; Spencer et al., 2006). However, these investigations used very short recovery durations (15 s and 21 s, respectively), and therefore cannot be directly compared to the present study. There are likely different mechanisms responsible for changes in performance over such short durations compared to longer durations. It was suggested that AR impaired performance in the aforementioned investigations by limiting PCr resynthesis via competition for limited O_2 supplies (Spencer et al., 2006). However, any potential limitations to PCr resythesis in this investigation did not cause any observable detrimental effect to PP performance in either of the active recoveries.

Peak power output, when assessed over a short duration, is primarily controlled by energy release from free ATP and the cycling of the PCr system. Indeed, PCr resynthesis has been strongly correlated with subsequent sprint performance (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995). Both free ATP and PCr energy sources are quickly depleted during high-intensity exercise, yet the half-time for PCr resynthesis is short, approximately 21-60 s (Harris et al., 1976; Yoshida & Watari, 1993; Bogdanis et al., 1995). Accordingly, maintenance of PP output requires adequate replenishment of PCr. As maintenance of PP output was observed in the current study, it follows that PCr levels were adequately replenished in all three recoveries. Thus, during 14 min recovery, there seems to have been ample time to replenish PCr levels, despite a significant O_2 demand to the working muscles in both the AR and CAR. Previously, it has been reported that AR can result in decreased oxyhemoglobin/oxymyoglobin recovery as well as prolong PCr resynthesis when a short (15 s to 2 min) recovery period is used (Dupont et al., 2007; Spencer et al., 2006; Yoshida, Watari, & Tagawa, 1996). Oxygen competition between working muscle and the aerobic process of PCr repletion did not appear to be a factor over the longer recovery period in this study. It may be that O_2 competition effects were dispersed over the extended recovery period and were thus nullified. That is to say, despite recovery intensities ranging from the IAT (73.2 ± 6.1% $\dot{V}O_{2max}$) to the $IVT_{-50\%\Delta T}$ (50.2 ± 5.3% $\dot{V}O_{2max}$), which imposed substantial $\dot{V}O_2$ demands, it seems the overall recovery duration was sufficient to allow potentially limited PCr repletion over a long enough time period to result in adequate replenishment. However, as PCr levels were not actually measured in the current

investigation, their successful repletion can only be inferred from subject's ability to achieve PP outputs equivalent to their baseline values. During the 30 s recovery periods between the five sprints PCr levels likely depleted, and this is evident in the significant main effect for time. Inter-sprint recoveries were equivalent across testing days and therefore this effect was similar on all days and is assumed to have had no discernable effect on the treatment groups.

Peak power was operationally defined as the highest 1 s mechanical power output during each individual sprint. Therefore, it may be that due to the operational definition of PP the sprint task was not sensitive enough to find statistical differences in the assessment of fatigue. It is possible that any potential effects of energy metabolism on PP output were masked by the criteria used to determine PP. That is, subjects may have been able to reproduce PP for 1 s despite physiological changes that would not allow them to maintain those power outputs over a longer duration. Though it is worth mentioning that the cyclist tested were significantly trained, as well as motivated, and thus it is more likely that they would have been able to reproduce their performances with greater reliability.

A 1 s duration was selected for the determination of PP as the proportion of the total sprint (10%) is most similar to the proportion of typical time, 5 s (16.7%), used to determine PP in a standard 30 s WAnT. Though, it should be noted that the measurement precision is lower (approx. ± 1.7%) with a shorter sample time for PP (SMI POWER, 2000). Furthermore, flywheel inertia was not considered in the calculations of power output. Lakomy (1986) showed that during a WAnT maximal power output was achieved before peak velocity and was 30-40% greater than power output at peak velocity. Therefore, it may be that power outputs reported in this study are lower than the power outputs actually achieved. However, as subjects were allowed to start sprints from a rolling start, the effects of not considering flywheel inertia in power calculations were reduced. Additionally, as all sprints were started in a similar matter, any error incurred became systematic and would not have compromised results with random error.

5.3.2 Mean Power

Mean MP output during AR was significantly greater than during PR. This appeared to be a result of a greater ability to maintain power output in the latter sprints in the AR trial compared to the PR trial. As the difference between MP scores became more prominent in the last three sprints. Additionally, MP decreased significantly over time within a given sprint task. At the individual level, maximum MP over the three days tended to happen in the AR trial. Furthermore, the majority of the initial and final maximum MP outputs occurred during the AR trial. Concurrent with these trends, subjects tended to experience their minimum MP outputs on

either the CAR or PR days. Thus, in this investigation AR showed a strong tendency toward maintenance of power output during the sprint task, compared to the other two recovery modes.

Several other researchers have noted a greater ability to maintain performance with the use of an AR (Ahmaidi et al., 1996; Bogdanis et al., 1996b; Connolly et al., 2003; Corder et al., 2000; Signorile et al., 1993; Spierer et al., 2004; Thiriet et al., 1993). Of the aforementioned investigations, only several have noted benefits to MP specifically with an AR (Ahmaidi et al., 1996; Bogdanis et al., 1996b; Spierer et al., 2004). Ahmaidi et al. (1996) found that AR resulted in higher MP outputs at high braking forces compared to PR during a repeat force-velocity test with increasing loads. The observed increased power performance with AR was associated with a decreased plasma La^- concentration. The authors suggest that the greater La^- clearance may have resulted in improved power outputs by reducing the amount of H^+ accumulation, which has been previously shown to inhibit glycolysis. While the finding that AR recovery resulted in improved MP output is in agreement with the current investigation, the interpretation of why is not. In this study AR and CAR both resulted in enhanced La^- clearance compared to PR. However, despite a La^- clearance rate that was equivalent to AR with CAR, no performance benefit was observed in the CAR trial. Therefore, the performance benefit observed in the AR trial cannot be solely attributed to enhanced La^- clearance.

Bogdanis et al. (1996b) noted greater MP output on a repeat 30 s sprint task separated by 4 min, when AR was used compared to PR. The improvement was not associated with a lower blood La^- concentration or higher blood pH. These findings are very similar to this investigation, with respect to the improved performance being dissociated from La^- clearance. The authors suggest four possible mechanisms for the improved performance with AR: (1) greater PCr resynthesis, (2) lower muscle La^- and $[H^+]$, (3) increased aerobic contribution to energy supply, and (4) changes in mechanical efficiency from increased muscle water content; all of which have the potential to be affected by blood flow (Bogdanis et al., 1996b). Previously it has been shown that during the initial 10 s of a 30 s sprint (approx. 45% of the TW of the sprint) a large portion of the ATP demand is supplied through anaerobic metabolism (Bogdanis et al., 1996a). Specifically, in the initial 10 s PCr accounted for 34% and glycolysis 42% of the energy supply. Since the noted performance benefits in Bogdanis et al.'s (1996a) investigation were attributed to a greater power output in the initial 10 s of the sprint, it was hypothesized that AR may have either enhanced PCr resynthesis, or increased the initial glycolytic contribution to the energy supply via H^+ removal. Nevertheless, despite this supposition, more recent works, with recovery durations varying from 15 s to 15 min, have shown a decreased muscle oxygenation (Dupont et

al., 2004; Dupont et al., 2007) and PCr resynthesis (McAinch et al., 2004; Spencer et al., 2006) with AR compared to PR. These findings imply that PCr resynthesis is impaired with AR, rather than enhanced. Thus it may be more plausible to infer that the improvement in performance noted by Bogdanis et al. (1996a) was likely due to an increased initial glycolytic contribution to the energy demand. It is possible that a similar mechanism would be responsible for the improved performance noted in this investigation.

Muscle La^- concentration was not determined in this investigation, but it has been previously shown that changes in plasma La^- and muscle La^- are independent of one another (McAinch et al., 2004). Thus, the finding that the two AR intensities in this investigation resulted in lower blood La^- concentrations has no direct bearing on the muscle La^- concentrations. Furthermore, investigations have reported both higher (McAinch et al., 2004; Peters-Futre et al., 1987) and lower (Bangsbo et al., 1994; Spencer et al., 2006) muscle La^- concentrations with AR. Therefore, a postulation as to what the effects of the recovery intensities used in this study were on muscle La^- cannot be made. However, it can be hypothesized that muscle pH would have been higher in the active recoveries as AR has been shown to increase muscle pH (Sairyo, Ikata, Takai, & Iwanaga, 1993; Sairyo et al., 2003) as well as blood pH (Siegler, Bell-Wilson, Mermier, Faria, & Robergs, 2006). A potentially lower $[H^+]$ in the muscle may have contributed to improved performance via a reduced inhibition of glycolytic enzymes and consequently greater glycolysis in the AR trial (Ahmaidi et al., 1996; Karlsson, Hulten & Sjodin, 1974). Slightly contrary to this statement is the fact that Siegler et al. (2006) did not find any performance benefits with AR despite an increased blood pH. However, blood pH is not necessarily indicative of muscle pH, and it may be that a decreased muscle pH results in improved performance.

Numerous investigations have shown that with repeat sprint tasks the aerobic contribution to energy supply increases with the number of sprints performed (Bogdanis et al., 1996a; Gaitanos, Williams, Boobis, & Brooks, 1993; Dorado et al., 2004; Trump, Heigenhauser, Putman, & Spriet, 1996). When AR is performed between the sprints the subsequent aerobic contribution is greater than with PR (Dorado et al., 2004). The increase in aerobic contribution has been attributed to faster $\dot{V}O_2$ kinetics (Dorado et al., 2004; Spriet, Lindinger, McKelvie, Heigenhauser, & Jones, 1989), similar to those observed when a warm-up is performed prior to high-intensity exercise (Bangsbo et al., 1994). Dorado et al. (2004) concluded that AR enhanced work capacity during high-intensity intermittent exercise by increasing the aerobic energy yield via faster $\dot{V}O_2$ kinetics and a longer working time. Though the precise mechanism by which AR

enhances work capacity was unclear. It may be that the improved performance in the AR trial in this investigation was a result of expedited $\dot{V}O_2$ kinetics too. However, ventilatory data was not measured during the sprint task, and therefore it cannot be determined for certain if this was in fact the case. At the $\dot{V}O_28$ sample (i.e. 14th min recovery) the $\dot{V}O_2$ was highest in the PR protocol, followed by the CAR then AR protocols. This seems contrary to expected outcome since $\dot{V}O_2$ should be lower during PR as the metabolic demand is minimal. However, the exercise and recovery protocol design had subjects complete the last 5 min of the 14 min recovery at the $IVT_{-50\%\Delta T}$, in order to give them a 'warm-up' prior to the sprint task, as well as to minimize the differences in pre-sprint starting conditions with respect to HR, blood flow, and $\dot{V}O_2$, by having all three recoveries finish at the same workload. In actuality, the 'warm-up' in the PR trial resulted in a significantly greater $\dot{V}O_2$ (33.0 ± 6.4 mL·kg^{-1}·min^{-1}) at the end of recovery ($\dot{V}O_28$) compared to AR (31.7 ± 6.0 mL·kg^{-1}·min^{-1}). It may be that the addition of exercise and increased $\dot{V}O_2$ may have resulted in O_2 competition between energy supply for exercise and the restorative processes of recovery. This contention is further support by the differences in the HR7 and HR8 values for PR and AR (108.2 ± 8.9 vs. 142.1 ± 9.4 bpm, $p \leq 0.05$; and 139.2 ± 8.9 vs. 143.7 ± 11.3 bpm, $p > 0.05$, respectively). From the 9th to 14th min of recovery during PR HR and $\dot{V}O_2$ increased drastically (11.1 ± 3.2 to 33.0 ± 6.4 mL·kg^{-1}·min^{-1}) whereas in AR HR was relatively steady while $\dot{V}O_2$ was gradually decreasing (32.9 ± 6.6 to 31.7 ± 6.0 mL·kg^{-1}·min^{-1}). Thus it would seem O_2 extraction would be greater in the PR trial at this time since overall $\dot{V}O_2$ was on the rise but close to the values from the AR and CAR trials, while HR was lower. The further uptake would be a result of the added exercise stimulus since any restorative processes would have already been in operation prior to the additional workload. Thus, it would appear that the $\dot{V}O_2$ kinetics may very well have been slower in the PR trial, since it has been previously shown that $\dot{V}O_2$ kinetics are faster when the O_2 content of arterial blood is elevated (Balsom, Ekblom, & Sjodin, 1994).

Two possible mechanisms suggested for faster $\dot{V}O_2$ kinetics are: increased blood flow to exercised muscle group and/or greater maintenance of aerobic regulatory enzyme activation levels (Bangsbo et al., 1994; Dorado et al., 2004). As to why only AR recovery resulted in improved MP output in this investigation remains unclear. To our knowledge this is the only investigation to have examined the effects of a two-tiered intensity recovery on subsequent performance. It seems plausible that both the CAR and AR would both maintain leg blood flow. Furthermore, leg blood flow during the PR recovery would have been close to the two active

recoveries at the start of the sprint task since all subjects finished at least the last 5 min of the last recovery period cycling at their respective $IVT_{-50\%\Delta T}$ workloads. Though, throughout the greater portion of the last recovery (i.e. 9 min of the 14 min) leg blood flow would have been substantially lower in the PR trial compared to the two AR trials. Thus, while reduced leg blood flow could account for the absence of a performance benefit in the PR trial, it does not provide an explanation for the lack of a benefit in the CAR trial. The higher intensity portions of the CAR (at IAT) may have been too intense to allow sufficient recuperation, despite a greater blood flow. This would have then had to have had a carry-over effect into the following recovery period, as the remainder of the recovery period was performed at the same workload as the AR trial. It is unlikely that any carry over effects would have had to do with PCr levels, as the duration at the $IVT_{-50\%\Delta T}$ recovery intensity would have allowed sufficient PCr resynthesis. The elevated intensity may have had an effect on blood or muscle pH levels, or the hydroelectric balance of the muscle by changing Na^+-K^+ pump activity (Bangsbo et al., 1992). Similarly there may have been some form of carry-over effect in the CAR trial with respect to oxidative enzyme activation levels. Again, the higher intensity recovery may have caused physiological changes, such as a decreased pH, that would have potentially limited oxidative enzyme activation. However, the aforementioned arguments are speculative and further research is needed to substantiate the claims.

Lastly, it has been suggested that water shifts from the blood to the muscle, as seen during intense sprint exercise, may increase intramuscular pressure and alter mechanical efficiency (Bogdanis et al., 1996b). Large increases in total muscle water from intense exercise have previously been reported (Sjogaard & Saltin, 1982). The shifts in water are likely driven by changes in osmolarity due to the production of metabolites and increases in blood pressure (Bogdanis et al., 1996b). The elevated intramuscular pressure over that of blood pressure may offset vasodilation and restrict local blood flow to certain areas of the muscle (Bogdanis et al., 1996b). A faster removal of such metabolites (e.g. La^-) may help to reduce the osmolarity imbalance and restore homeostatic conditions more rapidly and reduce muscular inefficiency.

Notwithstanding to these findings, there is some evidence to the contrary. In a very applied study Franchini et al. (2003) used a similar duration recovery (15 min) to examine the effects of a prior judo combat match on repeat upper body WAnT performance. No differences in performance were observed in this investigation between the different recoveries. Franchini et al. (2003) note that previous investigations finding performance benefits with AR versus PR, in general, used a shorter recovery duration (i.e. 6 min or less). Interestingly, this study is then one

of few investigations finding improved performance with an AR recovery over a duration greater than 6 min. One major difference between this investigation and that by Franchini et al. (2003) is that the current investigation exercise modalities were similar between the fatiguing work bouts, recovery, and performance task (i.e. all performed on bicycle ergometers). In the investigation by Franchini et al. (2003), subjects were fatigued during a simulated judo combat, recovered running, and had their performance assessed by repeat upper body WAnT. Thus, direct comparisons between the studies are limited due to changes in the exercised muscle groups as well as the intensities used. There is similar trend throughout the literature that limits between study comparisons since there are a wide variety of experimental designs that have been used to assess the effects of recovery intensity on performance (e.g. exercise intensity/duration/type, recovery intensity/duration/type, etc.).

5.3.3 Fatigue Index

There were no significant differences in FI amongst the recovery groups, contrary to the prediction that differences in FI would be inverse to MP. As expected FI increased over the number of sprints performed, demonstrating a larger difference between the PP and minimum power outputs. Interestingly, the mean FI was lower for all sprints in the AR trial, indicating a trend towards greater maintenance of power output. Despite being evident as a significantly greater MP output in the AR trial, the trend of a lower FI in the AR trial was not statistical significant.

Individual scores for FI showed less distinct trends than the other performance task variables. The amount of subjects who had their daily and overall maximum FI scores was relatively evenly distributed amongst the three testing days. This was also the case for the minimum FI scores as well as the initial and final maximum scores. Two possible explanations can account for this amount of variance among the individual scores. Firstly, it has been shown that performance decrement and fatigue indices scores inherently have large variations, even in trained populations, and thus should be interpreted cautiously (Glaister et al., 2007; McGawley & Bishop, 2006). Secondly, it is possible that the FI data did not reach significance for similar reasons described for PP. That is, the operational definitions for PP and minimum power used 1 s averages, which may not have been sensitive enough to detect statistical differences. Again, the 1 s duration for PP, and also minimum power, was selected to minimize the proportion of the sprint that each variable comprised. However, the shorter time comes at a cost of reduced precision for the measurement (SMI POWER, 2000). Decrement scores have been suggested to be more reliable for repeat-sprint exercise compared to FI scores (Glaister, Stone, Stewart,

Hughes, & Moir, 2004). As such, it is worth noting that assessment of fatigue across the sprint task (TaF) was analyzed using the formula described by Fitzsimons et al. (1993) and recommended by Glaister et al. (2004) and did not show statistical significance. This formula uses MP power to assess the amount of decrement from an ideal power output to an actual power output and therefore avoids the problem of reduced precision that the 1 s average may have introduced to the FI score. The main limitation to this formula is the assumption that maximum power output occurs on the first sprint (Glaister et al., 2004). As this criterion was not met, FI were presented in the results. Interestingly, despite the use of MP (i.e. a 10 s versus a 1 s average) the TaF score was still not significantly different between groups. This lends more support to the first interpretation, that a lack of statistical significance was due to the inherent variability in measures of fatigue, rather than the second interpretation. The TaF formula and scores (see Table 24) are presented in Appendix IX.

Many studies examining performance after active or PR have not included a measure of fatigue. This is partly due to the fact that several investigations have used TTE tests as their performance criterion (Dorado et al., 2004; McAinch et al., 2004; Siegler et al., 2006). Other investigations have simply not reported a measure of fatigue (Gaitanos et al., 1993) or used a design that did not allow assessment of fatigue by way of FI or decrement scores (e.g. Ahmaidi et al., 1996). Bogdanis et al. (1996b), Signorile et al. (1993), and Spencer et al. (2006) all reported fatigue scores, and did not find any significant changes to fatigue after AR. Bogdanis and colleagues (1996b) used FI as their measure of fatigue, whereas Signorile et al. (1993) used fatigue rate (i.e. power decrement over time, $W \cdot s^{-1}$), and Spencer et al. (2006) used a work decrement score (Fitzsimons et al., 1993). Furthermore, more similar to this study, Bogdanis et al. (1996b) and Signorile et al. (1993) did not find statistical differences in their measures of fatigue despite improved power outputs with AR. Conversely, Spencer et al., (2006) did not find statistical differences in work decrement, but found a reduced power output with AR. The similar lack of change to the fatigue measures is likely due to the greater variability associated with the measure of fatigue. In contrast, Spierer et al. (2004) found that AR resulted in a reduced fatigue index per bout (i.e. FI/# of bouts) during AR compared to PR in sedentary subjects but not in moderately trained hockey players. Congruent with this, sedentary subjects achieved higher MP outputs with AR whereas there was no significant difference in MP output between recoveries in the hockey players. Differences in the findings from Spierer et al. (2004) compared to the other investigations are likely due to methodological differences. Specifically, the other investigations performed a fixed number of sprints, whereas subjects in the latter investigation

performed serial WAnT until exhaustion (i.e. inability to complete the test) or until power output was reduced to less than or equal to 70% of the first sprint.

5.3.4 Total Work

Total work demonstrated the same results as MP, which is expected, as TW is the aggregate of MP scores. That is, contrary to the hypothesized results, AR in fact resulted in the greatest TW followed by CAR then PR. Differences were only significant between AR and PR TW scores. However, CAR may result in greater TW than PR, but these findings cannot say with certainty whether the improvement was a result of chance alone.

Among individual subjects the majority (seven) had their overall maximum TW output on the AR day. That was followed by the CAR day (four) then the PR day (one). Consistent with this trend is the fact that most (six) of subjects had their overall minimum TW output on the PR day, followed by the CAR day (five), then the AR day (one). Interestingly, Subjects 3 and 4, the exceptions to the trend, experienced their overall minimum and maximum on the AR and PR days respectively, and had the other extreme score default to the CAR day. The effect of the CAR on TW appears to be quite varied as an almost equal number of subjects experience TW maxima and minima on this day (four and five, respectively).

The exact mechanism by which AR resulted in improved TW remains unclear, although it is obviously due to the same factors proposed for the improved MP output. Thus, it seems that there is an interaction between maintenance of blood flow to the working muscle and an optimal intensity with which to allow restorative processes to take place. An alternative interpretation would be that there may be an optimal intensity that allows improved metabolism on subsequent exercise rather than effecting restorative processes. In either case, the variance in individual TW outcomes may be an artifact of the procedures used to determine threshold intensities. Since, workloads during the $\dot{V}O_{2max}$ test were increased by 30 W per stage, the thresholds could only be determined accurately within a 60 W range. Therefore, it could be that some of the subjects performed better in the CAR trial because their IAT workload was on the lower end on that range, whereas others may have performed worse because the workload may have been too high. Differences in relative workload intensities may have subsequently altered the aerobic contribution to metabolism by changing the chemical environment within the muscle and hence oxidative enzyme activation levels (Balaban, 1990). Or conversely, it may be that the glycolytic contribution to metabolism was hindered via an increased acidity (Spriet et al., 1989), since mild-intensity recovery has been shown to reduce intracellular pH levels (Sairyo et al., 2003). However, neither of these suppositions can be supported of refuted from the present study

results, and further research is necessary to clarify the mechanism for improved work capacity during AR.

Two other investigations have noted improved TW with AR compared to PR (Signorile et al., 1993; Spierer et al., 2004). Interestingly, in the investigation conducted by Spierer and colleagues (2004), both sedentary and moderately trained subjects performed more TW with AR compared with PR despite no significant differences in MP output in the trained group. This is likely the result of the criteria used to determine the cut-off point for exercise bouts performed. Subjects performed bouts until they were unable to achieve 70% of the PP output from the first sprint bout. This resulted in three moderately trained subjects performing an extra sprint bout on the AR day. In contrast, a recent investigation found no significant difference in TW performed between active and PR, despite a greater power decrement and lower final PP during AR (Spencer et al., 2006). Similarly, Franchini et al. (2003) measured TW performed but did not find any significant differences between active and PR. Other investigations have used TTE trials as their performance indicator (Dorado et al., 2004; Dupont et al., 2004; Dupont et al., 2007; McAinch et al., 2004) and therefore are not relevant for a direct comparison. However, it could be argued that a longer TTE is indicative of more TW, but it is not known whether or not this would hold for a repeat sprint task.

5.4 Heart Rate and Volume of Oxygen Consumed

Heart rate and $\dot{V}O_2$ data were collected as measures of exercise intensity, to allow comparisons of the recovery workloads throughout the exercise and recovery periods as well as prior to the initiation of the sprint task. Additionally, HR was measured to provide an indirect and crude measure of blood flow, albeit total body blood flow. Despite the varying recovery intensities resulting in different metabolic demands, the final 5 min of the 14 min recovery period was performed at the same intensity in all three recovery protocols. This was an attempt to standardize the pre-sprint physiological conditions. Assessment of HR and $\dot{V}O_2$ at this sample point allowed the determination as to whether or not this goal was achieved.

5.4.1 Exercise and Recovery Heart Rate

As expected mean HR values were different among the three recovery intensities during the SE bouts and recovery periods. Mean HR was greatest during the CAR trial, followed by AR, then PR. This was to be expected since the recovery intervals performed during the CAR were at a higher intensity for 2 min of the 5 min recovery periods, and 5 min of the 14 min recovery period compared to AR, as well as for the entire recovery intervals compared to PR. Similarly, the intensity during AR recovery intervals was higher throughout the entire recovery

periods compared to PR. This demonstrates that the amount of work performed prior to each sprint task was different between recovery conditions and had a significant physiological effect.

Mean HR after the first SE bout (HR1) was highest in the PR trial, but there were no significant differences between the three recovery trials. This was to be expected since the workloads were the same across all three recovery trials and subjects performed equivalent warm-ups on all testing days. However, after the second SE bout (HR3) the effect of PR on HR became evident. The lack of activity during the PR interval resulted in a significantly lower HR after the SE2 bout (HR3) compared to the other two recoveries. The lower HR value at the start of the PR SE2 bout (HR2) prevented the HR from reaching the same peak values as in the AR and CAR protocols. Heart rate prior to SE3 (HR4) was slightly greater in the AR protocol compared to the CAR protocol, which in turn were both significantly greater than the PR protocol. It is likely that this small difference is a result of a slower pedal cadence in the CAR protocol during the higher intensity portions. Subjects were only required to keep their cadence above 60 rpm, but were allowed to cycle at whatever speed they found comfortable. Consequently, while not systematically observed, anecdotal observations suggest that subjects had lower cadences during the higher workload periods of the CAR, as several subjects were warned to maintain their cadence above 60 rpm. The slower cadences apparently allowed for a lower HR during the first 2 min of the recovery interval that carried over into a slightly lower final HR at the 5^{th} min of recovery.

During the final recovery period mean HR followed the expected trends, with the highest values occurring in the CAR trial. At the end of recovery period (HR7) HR values were not significantly different from each other. This provides some evidence that the equivalent workload during the final 5 min of the 14 min recovery period was successful in standardizing the pre-sprint conditions. Therefore it is quite likely that subjects would have began each sprint task with a comparable cardiac output and blood flow to the legs on each testing day. Thus, it may be that differences observed in performance are related to changes that occurred earlier in the recovery period. The sprint task performance was not related to HR pre-sprint. Thus, the sprint task performance did not appear to be heavily controlled by pre-exercise cardiac output or blood flow factors.

5.4.2 Sprint Task Heart Rate

During the sprint task, HR did not differ significantly between groups. Mean HR across all five sprints did however mirror the trend observed during the SE and recovery period. The CAR SHR was highest, followed by AR, and then PR. This suggests that there was some

residual effect on HR from the SE bouts and recovery period to the sprint task. Interestingly, the greater amount of TW performed in the AR trial did not result in a significant change in HR compared to the other recoveries. The differences in performance on the sprint task appeared to be independent of HR. This is likely due to the fact that energy metabolism during high-intensity short duration exercise is predicated by local muscle factors, rather than systemic attributes.

5.4.3 Volume of Oxygen Consumed

Mean $\dot{V}O_2$ throughout the SE and recovery period was greatest in the CAR trial, followed by AR, then PR. This was a result of the higher recovery intensities and hence greater amount of work performed in the two active recoveries prior to the sprint task. As expected, this again demonstrates that the amount of work performed prior to each sprint task was different between recovery conditions and had a significant physiological effect.

The first and second SE bouts caused a similar $\dot{V}O_2$ peak in all three recovery trials. After the first 5 min recovery interval ($\dot{V}O_2$2), $\dot{V}O_2$ was similar between the two active recoveries, which were both greater than the PR $\dot{V}O_2$. Despite a substantially greater workload during the initial 2 min of the recovery interval in the CAR, equivalent workloads for the final 3 min of the interval resulted in a similar $\dot{V}O_2$ value at the $\dot{V}O_2$2 sample during to the AR trial. This finding was similar to the HR data. However, contrary to the HR scores at the same sample point, $\dot{V}O_2$ scores post-SE2 ($\dot{V}O_2$3) were not significantly different between recoveries. Leading into the third SE bout ($\dot{V}O_2$4), $\dot{V}O_2$ showed the same trend as the previous bout, with the exception that $\dot{V}O_2$ was slightly greater in the AR trial compared to the CAR trial. The difference is negligible and likely due to the summation of breaths over the 20 s sample period for $\dot{V}O_2$ data. At the $\dot{V}O_2$5 sample, $\dot{V}O_2$ was significantly higher in the CAR trial compared to the other two recoveries, since subjects were still riding at their IAT workload at this time. Active recovery $\dot{V}O_2$ was in turn significantly greater than PR $\dot{V}O_2$, as subjects were working at their $IVT_{-50\%\Delta T}$ workload compared to seated rest. Although the difference was less than the previous sample, $\dot{V}O_2$ remained significantly greater in the CAR at the $\dot{V}O_2$6 sample time compared to the other recoveries, in spite of the reduced workload. Obviously there was a lag in $\dot{V}O_2$ to accommodate the new workload. At the 9th min of recovery ($\dot{V}O_2$7) the differences in $\dot{V}O_2$ between the two active recoveries were no longer significant. Fascinatingly, the addition of $IVT_{-50\%\Delta T}$ workload to the PR trial at the 9th min of recovery resulted in a substantial increase in $\dot{V}O_2$, to the point that the PR $\dot{V}O_2$ was significantly greater than the AR $\dot{V}O_2$ at the $\dot{V}O_2$8 sample. Though the difference is hardly substantial, it perhaps reflects a lack of metabolic

efficiency with the onset of exercise in the PR, compared to greater efficiency in the AR from a sustained effort.

Thus, while the HR and $\dot{V}O_2$ data demonstrate that the SE bouts provoked similar physiological responses among the three testing days, it is apparent that the overall physiological effects throughout the exercise and recovery period are different. This was to be expected, as obviously the workloads between the three recoveries were not equivalent. Interestingly, the least amount of work prior to the sprint task (i.e. PR trial) did not result in the best performance, as one might logically reason. In fact, it was the middle amount of work performed pre-sprint (i.e. AR trial) that resulted in the best performance, as measured by TW performed.

5.5 Practical Significance

Active recovery has been suggested to result in superior recovery from exercise compared to PR (Ahmaidi et al., 1996; Bogdanis et al., 1996b; Spierer et al., 2004). However, the empirical evidence of this is more equivocal than the common perception. The belief that AR is beneficial to subsequent performance appears to result from historic belief that La^- is a cause of fatigue in addition to the finding that AR does in fact clear La^- faster than PR. From a practical standpoint in performance athletics, AR would only be beneficial if it in fact did transmit some sort of improvement to performance, regardless of the effect on La^- concentration. Furthermore, the effect on performance would need to impart a significant competitive advantage. The results from this investigation showed that the AR recovery employed over the two 5 min and one 14 min recovery intervals resulted in statistically significantly more TW during five 10 s sprints interspersed by 30 s, when compared to PR. However, conditions in a laboratory setting are different from those in an athletic competition. Specifically, when dealing with sports with multiple repetitive high-intensity bouts within one competition (e.g. hockey, football, soccer, basketball), a favourable and successful outcome is rarely determined purely by total work capacity. The unpredictable nature of the activities is determined by a combination of work capacity, physical fitness, skill, and motivation, to name a few. Similarly, in more fitness driven competitions (e.g. track, cycling, swimming), work capacity is not the sole factor determining success. Though, a greater work capacity would likely increase the chances for a successful outcome and thus is a desirable attribute.

Interestingly, the data obtained in this study appear to show practical significance when the differences in mean MP between groups are converted from power outputs to velocities. Using an on-line bicycle speed and power calculator (Zorn, 2005), differences in mean MP wattage demonstrated that the average subject tested would have gone 10 $km \cdot hr^{-1}$ faster in the

AR trial compared to the PR trial and 8.1 $km \cdot hr^{-1}$ faster than the CAR trial. The CAR trial would have had a velocity 3.1 $km \cdot hr^{-1}$ faster than the PR trial. Over 50 s of total sprint time, these velocities would correspond to distances of 138.9, 112.5 and 43.1 m, respectively, assuming that the mean MP (i.e. differences in velocity) was maintained. However, these calculations are somewhat contrived as they do not consider changing conditions, but nonetheless demonstrate the potential for application to competition. For example it cannot be determined from this experiment whether these effects would hold over an event like an individual pursuit (4000m), or be relevant in the final sprint to the finish line of a longer distance event.

CHAPTER VI: CONCLUSION

6.1 Conclusions

This study adds further knowledge to the wealth of literature surrounding the use of AR and its effects on subsequent performance. The following was concluded based on the results of the study:

a. Active and CAR both cleared blood La^- significantly faster than PR.
b. Active recovery resulted in greater TW performed on the five 10 s sprints via a greater maintenance of MP in the latter sprints compared to PR.
c. Despite similar decreases in blood La^- concentration between AR and CAR, no significant performance benefits were observed in the CAR trial. Therefore the improved performance in the AR trial appears to be independent of blood La^- clearance.
d. Active recovery at a moderate intensity (i.e. approx. 50% $\dot{V}O_{2max}$) may be an effective recovery intervention for recovery intervals of approximately 15 min when subsequent high-intensity intermittent exercise requires the maintenance of power output.

Therefore, this investigation confirms that a CAR, utilizing a moderate-to-high-intensity followed by a moderate-intensity of exercise, can successfully clear blood La^-. However, the intensities used in this investigation for the CAR did not impart any significant clearance benefit above that of a single intensity moderate AR. And furthermore, did not statistically improve performance above that achieved from PR. Thus, for similar exercise requirements and recovery durations it appears that a moderate intensity AR is most beneficial. This study provides more evidence that La^- is not a causative factor in the development of fatigue and that benefit of AR to performance is not directly related to the faster clearance of La^-. Additionally, it provides support for the use of an AR to promote improved performance over moderate recovery durations (i.e. approx. 15 min). However, further research is needed to confirm the effect of AR on performance over this recovery duration as the literature is somewhat divided, as well as to determine the exact mechanism by which AR is beneficial.

6.2 Recommendations for Future Research

Future research areas for the continued acquisition of knowledge regarding AR, lactate kinetics, performance and fatigue are diverse. While it is likely that future research endeavours will remain subdivided and specific, it is important for the resulting interpretations to remain unified and generalized to facilitate further discussion and dissemination.

6.2.1 Performance Criteria

It is imperative that future research into the effects of AR incorporates some form of performance assessment. Anecdotal evidence of improved performance or logical reasoning that increased blood La^- clearance should prevent fatigue (e.g. Gisolfi et al., 1966) can longer be the basis for the notion that AR is beneficial to performance. There must be empirical evidence of a performance benefit that can only come through objective assessment of performance variables.

6.2.2 Active versus Passive Recovery and Duration Dependency

Given that recent investigations have demonstrated improved performance with PR over short duration sprint and recovery intervals (Dupont et al, 2004; Dupont et al., 2007; Spencer et al, 2006), further research is still required to elucidate the time frames as well as intensities at which active or PR is most beneficial. Specifically, active recoveries of a moderate duration (i.e. ranging from 3-20 min) have shown equivocal findings as to whether they are beneficial or detrimental to performance. Therefore, future research should focus on illuminating the circumstances and intervals over which active or PR is most appropriate. Particularly the reproducibility of some of the previous works should be tested using larger sample sizes to confirm previous results.

6.2.3 Mechanisms

The mechanisms by which AR is beneficial are still undetermined and further studies delving into the mechanisms are needed. Similarly, as the mechanisms of muscular fatigue still remain elusive, it follows that any insight into methods to maintain or improve performance will be limited. As such, it follows that further research into the mechanisms of fatigue are needed and will lead to new ideas and interpretations of the processes of recovery, specifically, how or why AR is beneficial.

The development, utilization and refinement of new technologies will play a pivotal role in new research. Particular, Magnetic Resonance Spectroscopy (MRS) looks to be a promising tool as it allows for non-invasive and real-time measurement of the working muscle intracellular metabolism (Sairyo et al., 2003). Specifically, further research focused on the role of P_i and [H+] via MRS looks to be intriguing. Additionally, MRS technology may also prove to be useful for the assessment of muscular blood flow.

6.3.3 Suggested Modifications and Additions

Further research using similar investigative models to the one employed in this study look to benefit from the addition of more blood measures as well as the inclusion of muscle

measures. Metabolites of key interest would be La^{-}, H^{+}, Pi, and Ca^{2+}, in addition to examine the effects of temperature as well as the mediation of acid-base status via P_{CO2}.

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APPENDIX I

Table 16. Daily and Overall Maximum and Minimum Blood Lactate Values

Subject		1	2	3	4	5	6	7	8	9	10	11	12
PR	Max.	14.8†	16.3	15.1	13.2†	16.7†	15.6	15.0†	15.6†	17.2†	14.3†	15.0†	11.8†
	Time	3	2	7	5	4	5, 6, 7	7	5	5	5	6	3
	Min.	7.8	7.6‡	13.2	4.7	12.9	12.8	12.1	9.3	14.3	14.7	8.8	5.3
	Time	8	7	8	7	8	8	8	8	8	8	8	8
	Final	15.6*	16.9*	15.1*	15.3*	15.9*	15.7	14.8	14.4*	15.8*	13.4	16.2*	10.7*
AR	Max.	11.1	15.6	17.6†	11.4	13.4	16.0†	14.6	12.0	17.2†	13.2	17.2	11.2
	Time	5	6	5	5	5	7	5	5	5	5	5	3
	Min.	2.8‡	9.0	10.0	7.1	6.2	9.2	8.3‡	5.1	7.6‡	6.0	5.2	4.2
	Time	8	7	7	7	8	8	8	8	8	8	8	8
	Final	11.6	15.2	13.3	14.4	13.1	16.8	15.8*	13.4	17.0	11.9	13.9	10.2
CAR	Max.	13.4	19.6†	13.8	10.0	14.4	15.2	14.9	11.	15.2	12.8	12.4	11.1
	Time	5	7	3	5	3	5	5	3	5	5	5	3
	Min.	5.6	14.0	6.6‡	3.2‡	3.8‡	6.1‡	10.3	4.0‡	10.6	5.1‡	4.8‡	4.1‡
	Time	8	8	8	8	8	8	8	8	8	8	8	8
	Final	14.6	16.9*	15.1*	12.9	10.6	16.0*	14.3	11.6	15.0	16.6*	12.8	9.2

Subjects 2 and 11 had two and one missing data point, respectively; † Overall maximum blood La^-; ‡ Overall minimum blood La^-; * Overall final maximum blood La^-

APPENDIX II

Table 17. Daily and Overall Maximum and Minimum Peak Powers

Subject		1	2	3	4	5	6	7	8	9	10	11	12
PR	Max.	1113	1105	1012	1179	1050	1359†	975	982	1055	894	942	1290
	Sprint	2	5	1	1	1	2	1	2	1	1	2	1
	Min.	991	1045	907‡	1060	936	1189	847‡	859‡	920	759‡	904	1142‡
	Sprint	3	4	2, 5	5	4, 5	5	5	5	4	4	3	3
	Initial	1068	1081	1012$^{\infty}$	1179$^{\infty}$	1050	1328$^{\infty}$	975$^{\infty}$	951	1055	894$^{\infty}$	923	1290
	Final	1023*	1105	907*	1060	936	1189	847	859	926*	817	929*	1212
AR	Max.	1010	1189†	977	1135	1057	1320	1034†	1013	1023	894	985	1342†
	Sprint	3	1	1	1	1	1	2	1	2	1	1	1
	Min.	856‡	1057	794	1048‡	969	1143	905	890	862	791	904	1228
	Sprint	1	4	3	5	4	5	5	5	5	4	4	4
	Initial	856	1189$^{\infty}$	977	1135	1057	1320	911	1013	978	894$^{\infty}$	985	1342$^{\infty}$
	Final	952	1147*	822	1048	1023*	1143	905*	890*	862	811	929*	1267*
CAR	Max.	1152†	1141	1068†	1185†	1077†	1312	901	1037†	1139†	952†	1010†	1337
	Sprint	1	1	2	2, 3	1	1	3	1	1	2	1	2
	Min.	1010	1032‡	907‡	1091	902‡	1073	850	884	888‡	785	873‡	1149
	Sprint	3	2	3	5	5	5	5	5	5	1	5	4
	Initial	1152$^{\infty}$	1141	914	1135	1077$^{\infty}$	1312	878	1037$^{\infty}$	1139$^{\infty}$	785	1010$^{\infty}$	1290
	Final	1017	1093	892	1091*	902	1073	850	884	888	843*	873	1157

† Overall maximum PP; ‡ Overall minimum PP; $^{\infty}$ Overall initial maximum PP; * Overall final maximum PP

APPENDIX III

Table 18. Daily and Overall Maximum and Minimum Mean Powers

Subject		1	2	3	4	5	6	7	8	9	10	11	12
PR	Max.	818	878	919†	814†	865†	1039	696	809	932	821	730	1014
	Sprint	1	1	1	1	1	1	1	1	1	1	1	1, 2
	Min.	792	853‡	741	668‡	734	802	586‡	615	689‡	569‡	675	913
	Sprint	5	4	5	5	4	5	5	5	5	4	5	5
	Initial	818	878	919$^{\infty}$	814$^{\infty}$	865$^{\infty}$	1039	696	809	932	821	730	1014
	Final	792	860	741	668	739	802	586	615	689	597	675	913
AR	Max.	844	933†	888	791	862	1111†	735†	822†	923	830†	775†	1033†
	Sprint	3	1	1	1	1	1	2	1	1	1	1	1
	Min.	796	875	696‡	672	759	827	610	630	709	650	669	976
	Sprint	1, 5	5	4	5	4	5	5	5	5	4	5	5
	Initial	796	933$^{\infty}$	888	791	862	1111$^{\infty}$	707$^{\infty}$	822$^{\infty}$	923	830$^{\infty}$	775$^{\infty}$	1033$^{\infty}$
	Final	796*	875	751	672*	761*	827*	610	630*	709*	652*	669*	976*
CAR	Max.	894†	921	916	757	839	1079	686	817	939†	806	743	1018
	Sprint	1	1	2	1	1	1	1	1	1	2	1	1
	Min.	786‡	885	777	676	712‡	799‡	611	592‡	708	638	635‡	872‡
	Sprint	5	5	4	5	5	5	5	5	5	5	5	5
	Initial	894$^{\infty}$	921	873	757	839	1079	686	817	939$^{\infty}$	750	743	1018
	Final	786	885*	784*	676	712	799	611*	592	708	638	635	872

† Overall maximum MP; ‡ Overall minimum MP; $^{\infty}$ Overall initial maximum MP; * Overall final maximum MP

APPENDIX IV

Table 19. Daily and Overall Maximum and Minimum Fatigue Indexes

Subject		1	2	3	4	5	6	7	8	9	10	11	12
PR	Max.	41.6	36.6	31.3	56.2	38.1	47.2†	46.4	46.9	40.3†	45.7†	47.0†	42.7
	Sprint	2	5	4	4	4	4	2	4	5	5	5	4
	Min.	35.7	27.6	13.2	47.6‡	25.3‡	32.0	40.7	24.5‡	21.3	18.0	35.8	34.9‡
	Sprint	3	2, 3	2	1	2	1	1	1	1	1	1	3
	Initial	38.0$^{\infty}$	28.5	16.0	47.6	25.6	32.0$^{\infty}$	40.7$^{\infty}$	24.5	21.3	18.0$^{\infty}$	35.8	35.2
	Final	38.4	36.6	27.9*	55.9	36.0	46.1*	44.8	44.3	40.3*	45.7*	47.0*	38.7
AR	Max.	28.4	38.9†	19.4	57.1	38.8†	46.0	47.1†	48.4	32.7	36.5	43.0	40.4
	Sprint	5	5	1	5	5	4	5	4	4	5	5	1
	Min.	11.3‡	25.6‡	11.5‡	48.4	28.0	28.1	34.0‡	32.7	9.9‡	14.4	37.5	38.3
	Sprint	1	2	3	1	1	1	1	1	1	1	2	5
	Initial	11.3	32.0$^{\infty}$	19.4$^{\infty}$	48.4	28.0	28.1	34.0	32.7	9.9	14.4	42.4	40.4$^{\infty}$
	Final	28.4	38.9*	18.8	57.1	38.8*	42.6	47.1*	46.9	30.6	36.5	43.0	38.3
CAR	Max.	47.1†	33.1	32.3†	64.6†	34.9†	41.7	41.7	54.2†	39.9	42.0	44.4	50.3†
	Sprint	4	5	4	4	2	5	4	5	3	4	1	2
	Min.	35.2	25.7	11.5‡	50.0	31.9	27.3‡	34.6	34.3	26.6	7.4‡	32.4‡	35.8
	Sprint	1	2	1	1	1	2	1	1	1	1	2	1
	Initial	35.2	28.6	22.8	50.0$^{\infty}$	31.9$^{\infty}$	28.2	34.6	34.3$^{\infty}$	26.6$^{\infty}$	7.4	44.4$^{\infty}$	35.8
	Final	40.5*	33.1	11.5	58.9*	32.1	41.7	41.2	54.2*	34.8	42.0	44.3	42.6*

† Overall maximum FI; ‡ Overall minimum FI; $^{\infty}$ Overall initial maximum FI; * Overall final maximum FI

APPENDIX V

Table 20. Individual Total Work Outputs and Maxima and Minima

Subject	PR	AR	CAR
1	39890‡	40730	41420†
2	43320‡	44920†	44790
3	40870	39500‡	41550†
4	36740†	36330	36110‡
5	39400	40010†	38440‡
6	46180‡	47240†	46750
7	32190	33720†	31990‡
8	34730‡	35320†	33500
9	39880‡	41150	41170†
10	33570‡	35600	35620†
11	35170	35720†	34640‡
12	47360	49800†	46050‡

† Overall maximum TW; ‡ Overall minimum TW

APPENDIX VI

Table 21. Daily and Overall Maximum and Minimum Exercise and Recovery Heart Rate Values

Subject		1	2	3	4	5	6	7	8	9
PR	Max.	178	180	170	170	185	160	176	172	192
	Time	1, 3	1, 3	3	3	3	1	3	3	3
	Min.	97‡	99‡	96‡	94‡	108‡	102‡	118‡	96‡	110‡
	Time	6	6	6	6	6	6	6	6	6
	Initial	178∞	180∞	167∞	166	177	160	175∞	166	188
	Final	107	102	107	104	116	112	119*	99	127
AR	Max.	179	191†	173†	174	186	168†	179	179†	201†
	Time	3	3	3	3	3	3	3	3	3
	Min.	119	150	140	141	129	130	138	138	144
	Time	2	2	2	5, 6	5, 6	2	6, 7	7	6
	Initial	170	180∞	164	169∞	179	161∞	167	167∞	193∞
	Final	96	111	110*	101	112	113*	116	115*	137*
CAR	Max.	186†	188	169	178†	190†	168†	183†	175	201†
	Time	3	3	3	3	3	3	5	3	3
	Min.	133	154	141	149	129	136	136	131	149
	Time	2, 7	2	2	4	2	2	2	8	2
	Initial	177	176	160	166	181∞	159	172	167∞	189
	Final	113*	119*	110*	105*	117*	110	115	106	124

n = 9, Subjects 10-12 were excluded; Initial, HR at HR1; Final, HR at HR9; † Overall maximum HR; ‡ Overall minimum HR; ∞ Overall initial maximum HR; * Overall final maximum HR

APPENDIX VII

Table 22. Daily and Overall Maximum and Minimum Sprint Task Heart Rate Values

Subject		1	2	3	4	5	6	8
PR	Max.	176	177	171	180	185†	169	178
	Sprint	4, 5	3	3, 4, 5	3	5	5	5
	Min.	166	167‡	162‡	174	176	157‡	169
	Sprint	1	1	1	1	1	1	1
	Initial	166	167	162	174$^{\infty}$	176$^{\infty}$	157	169
	Final	176	176	171	178	185*	169	178
AR	Max.	178	190	171	178	184	170†	184†
	Sprint	5	3, 5	3, 5	4, 5	5	5	4, 5
	Min.	165‡	178	163	171‡	169‡	162	173
	Sprint	1	1	1	1	1	1	1
	Initial	165	178	163$^{\infty}$	171	169	162$^{\infty}$	173
	Final	178	190*	171	178	184	170*	184*
CAR	Max.	181†	193†	176†	183†	185†	170†	175
	Sprint	4, 5	4	4	4	5	3, 4, 5	4, 5
	Min.	168	179	162‡	173	170	159	165‡
	Sprint	1	1	1	1	1	1	1
	Initial	168$^{\infty}$	179$^{\infty}$	162	173	170	159	165$^{\infty}$
	Final	181*	190*	173*	182*	185*	170*	175

n = 7, Subjects 7 and 9-12 are excluded; Initial, HR at SHR1; Final, HR at SHR5; † Overall maximum SHR; ‡ Overall minimum SHR; $^{\infty}$ Overall initial maximum SHR; * Overall final maximum SHR

APPENDIX VIII

Table 23. Daily and Overall Maximum and Minimum Volume of Oxygen Consumed Values

Subject		1	2	3	4	5	6	7	8	9	10	11	12
PR	Max.	67.6	73.0	52.2†	59.4	56.5†	54.7	61.0	54.9	53.6	59.7	58.5	64.0
	Time	1	3	3	3	3	3	3	1	3	1	1	1, 3
	Min.	13.1‡	9.1‡	8.7‡	6.6‡	9.7‡	10.2‡	11.1‡	12.3‡	13.9‡	15.8‡	6.6‡	16.0‡
	Time	6	7	6	6	6	6	6	6	6	6	7	6
	Initial	67.6	63.4	46.8	51.7	54.7$^{\infty}$	50.6	59.3$^{\infty}$	54.9$^{\infty}$	46.5	59.7$^{\infty}$	58.8$^{\infty}$	64.0
	Final	33.7	48.4	28.5	38.5	25.1*	30.2*	29.1	30.2	26.8	35.8*	37.7*	31.5*
AR	Max.	73.5†	75.8	50.1	60.6	56.5†	60.1†	59.2	56.5†	56.5	60.0†	55.7	65.3
	Time	3	3	3	3	3	3	1	3	1, 3	3	1	1
	Min.	31.4	47.1	30.2	38.3	24.0	29.3	26.8	28.6	23.3	31.1	29.9	29.5
	Time	8	6	8	8	8	8	6	7	7	8	8	6
	Initial	70.0$^{\infty}$	65.9	45.7	57.6$^{\infty}$	50.9	52.7	59.2	54.3	56.5$^{\infty}$	50.6	55.7	65.3
	Final	31.4	47.6	30.2	38.3	24.0	29.3	30.2*	31.1*	27.3	31.1	29.9	29.9
CAR	Max.	70.1	82.3†	51.0	68.2†	54.1	57.8	63.7†	51.8	58.3†	59.2	61.0†	65.7†
	Time	3	3	3	3	3	3	3	3	3	3	3	1
	Min.	36.4	48.6	32.3	42.4	24.6	29.6	26.5	28.4	31.0	31.3	27.1	23.8
	Time	8	8	8	7	8	8	2	8	8	8	8	8
	Initial	69.2	69.5$^{\infty}$	48.8$^{\infty}$	57.0	52.8	53.6$^{\infty}$	55.7	51.3	54.7	57.9	58.0	65.7$^{\infty}$
	Final	36.4*	48.6*	32.3*	42.5*	24.6	29.6	27.4	28.4	31.0*	31.3	27.1	23.8

Initial, $\dot{V}O_2$ at $\dot{V}O_2$1; Final, $\dot{V}O_2$ at $\dot{V}O_2$8; † Overall maximum $\dot{V}O_2$; ‡ Overall minimum $\dot{V}O_2$; $^{\infty}$ Overall initial maximum $\dot{V}O_2$; * Overall final maximum $\dot{V}O_2$

APPENDIX IX

Sprint Task Fatigue Formula

Fatigue = the percentage decrement score

$$\text{Fatigue} = 100 - [(\text{Total power output} \div \text{Ideal power output}) \times 100]$$

Where

Total power output = sum of MP outputs from all sprints

Ideal power output = the number of sprints × MP_{max}

Table 24. Sprint Task Fatigue Scores

Subject	PR	AR	CAR
1	2.5	3.5	7.3
2	1.3	3.7	2.7
3	11.1	11.0	9.3
4	9.7	8.1	4.6
5	8.9	7.2	8.4
6	11.1	15.0	13.3
7	7.5	8.2	6.7
8	14.1	14.1	18.0
9	14.4	10.8	12.3
10	18.2	14.2	11.6
11	3.6	7.8	6.8
12	6.6	3.6	9.5
$\bar{X}$	9.1 ± 5.1	8.9 ± 4.2	9.2 ± 4.1