Preparatory Strategies for Optimising an All-out Sprint Effort

Mohd Sani Madon
(Msc. Phys ed.)

**This thesis is presented for the degree of Doctor of Philosophy of the University of Western Australia
School of Human Movement and Exercise Science**

2007

Declaration

I declare that this thesis is comprised of my own research work and that it has not been previously submitted at any other University

The thesis is comprised of research that was carried out while I was an international Postgraduate Student at UWA

…………………………………

Mohd Sani Madon
24 March 2006

Abstract

The inclusion of a warm-up in the form of prior exercise (PE) is generally advocated as a preparatory strategy of choice to improve sprint performance. Although there is evidence that both increasing muscle temperature and mobilising the cardiorespiratory system prior to exercise contribute largely to the benefit of PE on sprint performance, their relative importance is unknown. Another important question relates to situations where an athlete has to engage in a sprint shortly after one or several earlier sprints. Under these conditions, is engaging in mild exercise also the most effective preparatory strategy to adopt prior to sprinting when performed after a previous sprint(s)? It was the primary aim of this thesis to address these questions.

Firstly, we hypothesised that there is a temporal shift in the mechanisms responsible for the effect of PE on power output during a maximal sprint effort, with temperature-dependent mechanisms playing a more important role at the onset of the sprint and mobilisation of the cardiorespiratory system playing a more important role later. To test this hypothesis, we compared the responses of a 30-s sprint to different PE protocols designed to control for either muscle temperature or pre-exercise $\dot{V}O_2$. A group of cyclists or triathletes performed either a 4-min PE and 10-min rest (PE_{4R}), 4-min PE (PE_4) or 20-min PE (PE_{20}) immediately before undertaking a 30-s sprint, with sprint performance evaluated by measuring peak power (PP) and mean power over 30 seconds (MP_{30}), first 6 (MP_{+6}) and 10 seconds (MP_{+10}), and last 10 (MP_{-10}) and 20 seconds (MP_{-20}). We found that core and muscle temperatures increased significantly in response to PE_{20}, but were not significantly affected by PE_4 and PE_{4R}, thus allowing us to isolate the effect of pre-sprint $\dot{V}O_2$. On the other hand, $\dot{V}O_2$ prior to sprinting increased to a similar extent in response to PE_{20} and PE_4, but not to PE_{4R}. Our findings show that MP_{30} was similar for PE_{20} and PE_4, but lowest after

PE_{4R}. On the other hand, PP, MP_6 and MP_{10} were significantly greater after PE_{20} than after PE_{4R} or PE_4, but PP was not different between the latter two PE protocols. In contrast, MP_{-10} and MP_{-20} were higher after PE_4 than after $PE_{20,}$ and lowest after PE_{4R}. Overall, these findings show for the first time that there is a temporal transition in the mechanisms responsible for the increase in power output in response to PE performed immediately before a 30-s maximal sprint effort, with increased muscle temperature playing the most important role at the onset of exercise and the mobilisation of the cardiovascular system later (>10 s).

Given that 20 min of PE performed immediately before a sprint improves sprint power, this raises the question of whether mild physical activity (< 40% $\dot{V}O_{2peak}$), or active recovery, performed between two consecutive sprints is preferable to a rest as the best preparatory strategy for the optimisation of sprint performance. In this regard, most studies have reported that the impact of active recovery is either small (< 3%) or undetectable. We hypothesised that these conflicting results might be due in part (a) to a failure to control for placebo effect and (b) to the efficacy of this recovery protocol being compared at times close to full recovery (14-20 min). In order to test these hypotheses and compare active and passive recovery when recovery is incomplete, we established the time dependency of the recovery of several indicators of sprint performance including, PP, MP_{30}, MP_{+10}, MP_{-20} and MP_{-10}. A group of exercise-trained male athletes performed two 30-s sprints on a cycle ergometer, each separated by a recovery period of either 0, 1, 2, 5, 10, 15, 20 or 40 min. Based on the results of this experiment, passive and active recovery performed at an intensity of 40% $\dot{V}O_{2peak}$ were then compared at a time when recovery of all indicators of power output was 45-75% complete (4 min) or near complete (20 min), with great care taken to minimise the possibility of a placebo effect by informing our participants that we were only concerned with the effect of recovery on blood

variables. We found that, although active recovery resulted in a faster return of blood lactate and pH to pre-exercise levels, it had no effect on the recovery of any of the indicators of sprint performance examined here, irrespective of recovery time. Our findings thus support, for the first time, the possibility, that a placebo effect might explain the results of those studies that have reported a marginal improvement in sprint performance capacity in response to active recovery.

Given strong evidence from the literature that sprint performance falls in response to several consecutive 30-s sprints, and our findings and those of others that early and late mean power during a single sprint recover at different rates, we tested for the first time the hypothesis that the early and late mean power output of each of repeated 30-s sprints respond differently to active and passive recovery, with active recovery providing the best preparatory strategy for the optimisation of repeated sprint performance. A group of trained athletes was subjected to four consecutive bouts of 30-s sprint, each separated by 20 min of either active recovery at 40% $\dot{V}O_2$ peak or passive recovery. Our results show that PP, MP_{-20} and MP_{-10} did not fall between the first and last sprints, and were not affected by active recovery. In contrast, we found that MP_{10} and MP_{30} decrease significantly between the first and last sprint of the passive recovery trial, but not when active recovery is performed between consecutive sprints. Finally, this study also showed that the fall in mean power associated with repeated 30-s sprints in the passive recovery trial resulted primarily from a fall in early, but not late power output. These findings show that the early and late mean power output of repeated sprints respond differently to active and passive recovery, with the decrease in total mean power with repeated 30-s sprints resulting primarily from a fall in early as opposed to either late power output or peak power, thus highlighting the benefit of active recovery as a favourable preparatory strategy for the performance of repeated sprints of short (<10s) or longer duration (<30s), but not for repeated peak power.

Acknowledgements

I wish to express sincere appreciation to those individuals who provided assistance and support during the course of my PhD. Without their involvement, the completion of this thesis would not have been possible. Particularly, I would like to acknowledge the following people:

Special thanks to Dr. Paul Fournier, my principal supervisor, for his scholarly expertise, passionate love for knowledge, adept advice, bountiful patience and countless hours of guidance and assistance without which this thesis would take years more to complete.

To Dr David Bishop for his excellent knowledge, experience and countless hours in the lab making sure all is perfect. Your commitment to assist me is very much appreciated.

To my willing subjects………. "its all for science, mates"…… Without your participation, this thesis would not have been submitted today. Thank you again.

A big thank you to all technical staff for always being "handy" in time of needs. Your expertise in your area of knowledge is second to none. Tony, Peter, Don and all.

A sincere thanks to HOD, Professor Dr. Brian Blanksby, also known as B^2 and all administrative staff of the Department of Human Movement and Exercise Science, UWA. My appreciation for all the assistance rendered.

Thank you to all my colleagues in Ex. Physio team; Dr. Luis, Hanz, Vanessa, Kym, Lian, Rob D, Rob M, Rob S, Tim and Stephen for sharing the "knowledge".

Finally, special thanks to my family for all the support, encouragement, patience and understanding, To my mother who didn't make it to see me through. This success is my gift to you. Your 'doa has helped me through.

March 2006-03-24

Table of contents

Declaration……………………………………………………………… i

Abstract…………………………………………………..…………..…… ii

Acknowledgments…………………………………………………..……. v

Table of Contents……………………………………….…………..……. vi

List of Figures……………………………………………………………. xii

List of Abbreviations……………………………………………………..… xvii

Chapter 1:

Review of related literature

1.1 Short intense sprint effort: a ubiquitous component of high intensity sports……………………………………………………...2

1.2 Pattern of ATP production and fuel utilisation during a maximal sprint effort…………………………………….…..……..3

1.3 Mechanical manifestation and sites of fatigue during intense muscle contraction

1.3.1 Mechanical manifestation of fatigue………………….…….…4

1.3.2 Potential sites of fatigue: central or peripheral…..………...5

1.3.3 Potential peripheral sites affected by fatigue……… ……..6

1.3.4 Impaired propogation of action potential along the sarcolemma and T-tubules…………………… …………7

1.3.5 Ca^{++} release and reuptake for the sarcoplasmic Reticulum………………………………………….………….9

1.3.6 Cross-bridge cycling…………………………….…………….9

1.4 Mechanism of fatigue associated with an intense muscle contraction

1.4.1 Lactate and fatigue……………………………….……10

1.4.2 H+ and fatigue……………………………………………11

1.4.3 Inorganic phosphate (Pi) and muscle fatigue……..……15

1.4.4 Adenylates and fatigue……………………………………18

1.5 Preparatory strategies to optimise sprint performance: Impact of "warming-up"

1.5.1 effect of prior exercise (PE) on sprint performance…….20

1.5.2 Temperature-mediated effects of PE on sprint performance of short duration…………………………..…22

1.5.3 PE-mediared mobilisation of the cardiorespiratory system prior to a sprint……………………………………..24

1.5.4 $\dot{V}O_2$ kinetics-mediated effects of PE on sprint performance of short duration……………………………..26

1.5.5 Cardiorespiratory-independent mechanism of PE effect on sprint performanceMechanism of PE effect on sprint performance……….……………………….…….28

1.5.6 Mechanism underlying the negative effect of PE on sprint performance……………………………………..28

1.5.7 Relative contributions of temperature and cardiorespiratory-dependent mechanism in determining the positive effect of PE on sprint performance………………………………………………..29

1.6 Preparatory strategies to optimise sprint performance after an earlier sprint……………………………………………29

1.7 Preparatory strategies to optimise single sprint performance after repeated sprints……………………………..…..35

1.8 Aims and Research hypothesis……………………………..……41

Chapter 2:

Preparatory strategies for optimising all-out sprint efforts I: Effect of prior exercise on subsequent sprint performance

2.1 Introduction……………………………………………………….45

2.2 Materials and Methods

2.2.1 Participants…………………………………………….…...47

2.2.2 Familiarisation session……………………………….……47

2.2.3 Experimental design………………………………………47

2.2.4 Sprint trial………………………………………………...48

2.2.5 Rectal, muscle and skin temperature……………………49

2.2.6 Determination of $\dot{V}O_2$ and $\dot{V}O_2$ peak…………………...50

2.2.7 Blood sampling…………………………………………...51

2.2.8 Statistical Analyses………………………………………. 51

2.3 Results

2.3.1 Effect of PE on muscle and rectal temperature, $\dot{V}O_2$, plasma lactate concentration and pH……………..52

2.3.2 Effect of PE conditions on power output During a 30 s sprint……………………………………..52

2.4 Discussion………………………………………………………..64

Chapter 3:

Preparatory strategies for optimising all-out sprint efforts II: Effect of active recovery post-sprint on subsequent sprint performance

3.1 Introduction……………………………………………….…….......73

3.2 Materials and Methods

3.2.1 Participants………………………………………………………76

3.2.2 Familiarisation session……………………………….………...76

3.2.3 Effect of recovery time on sprint capacity ………..……76

3.2.4 Effect of active and passive recovery on sprint Capacity……………………………….……………..……….77

3.2.5 Sprint trial………………………………………………………...77

3.2.6 $\dot{V}O_2$ peak determination………..………………….……78

3.2.7 Blood sampling…………………………………………………..79

3.2.8 Statistical analyses………………………………………...79

3.3 Results

3.3.1 Effect of recovery duration post-maximal effort on subsequent sprint performance………………..80

3.3.2 Pattern of change in blood pH and lactate Levels…………………………………………………………80

3.3.3 Effect of active and passive recovery on recovery of sprint performance…………………………..80

3.3.4 Effect of 4 and 20 min of active and passive

recovery on recovery of H+ and blood lactate levels……………………………………………………...97

3.4 Discussion…………………………………………….………........98

Chapter 4:

Preparatory strategies for optimising all-out sprint efforts III: Effect of active recovery between repeated all-out sprints on subsequent sprint performance

4.1 Introduction……………………………………………………..104

4.2 Materials and Methods

4.2.1 Participants……………………………………………106

4.2.2 Familiarisation session…………………………………106

4.2.3 Intermittent sprint protocol…………………….……..106

4.2.4 Sprint trial………………………………………………107

4.2.5 $\dot{V}O_2$ peak determination……………………………..107

4.2.6 Blood sampling………………………………………..108

4.2.7 Statistical analyses……………………………………109

4.3 Results

4.3.1 Mean and peak power…………………….…………..110

4.3.2 Lactate and pH responses……………………………110

4.4 Discussion……………………………………….……….......119

Chapter 5:

Summary, Discussion and Suggestions For

5.1 General Discussion……………………………………………125

5.2 Conclusion and direction of future research….……………132

Reference List……………………………………………………...135

List of figures

		Page
Figure 2.1	Effect of different prior exercise (PE) treatments on muscle temperature.	53
Figure 2.2	Effect of different prior exercise (PE) treatments on rectal temperature.	54
Figure 2.3	Effect of different prior exercise (PE) treatments on the rate of oxygen consumption.	55
Figure 2.4	Effect of different prior exercise (PE) treatments on lactate levels prior to a 30-s sprint.	56
Figure 2.5	Effect of different prior exercise (PE) treatments on the concentration of blood H^+ prior to a 30-s sprint.	57
Figure 2.6	Effect of different prior exercise (PE) treatments on peak power (PP) during a 30-s-sprint.	58
Figure 2.7	Effect of different prior exercise (PE) treatments on mean power over 30-s (MP_{30}).	59
Figure 2.8	Effect of different prior exercise (PE) treatments on mean power over first 6 s (MP_6).	60

Figure 2.9 Effect of different prior exercise (PE) treatments on mean power over 10 s (MP_{10}). 61

Figure 2.10 Effect of different prior exercise (PE) treatments on mean power over last 20 s (MP_{-20}). 62

Figure 2.11 Effect of different prior exercise (PE) treatments on mean power over last 10 s (MP_{-10}) 63

Figure 3.1 Effect of recovery time post-sprint on peak power (PP) generated during a subsequent 30-s sprint 81

Figure 3.2 Effect of recovery time post-sprint on mean power 30-s (MP_{30}) generated during a subsequent 30-s sprint. 82

Figure 3.3 Effect of recovery time post-sprint on mean power 10 s (MP_{10}) generated during a subsequent 30-s sprint. 83

Figure 3.4 Effect of recovery time post sprint on mean power last 20 (MP_{-20}) generated during a subsequent 30-s sprint. 84

Figure 3.5 Effect of recovery time post sprint on mean power last 10 (MP_{-10}) generated during a subsequent 30-s sprint. 85

Figure 3.6	Effect of recovery time post sprint on blood lactate generated during a subsequent 30-s sprint.	86
Figure 3.7	Effect of recovery time post sprint on blood [H+] generated during a subsequent 30-s sprint.	87
Figure 3.8	Effect of 4 and 20 min passive and active recovery On peak power generated during a subsequent 30-s sprint.	88
Figure 3.9	Effect of 4 and 20 min passive and active recovery on Mean power 30-s (MP_{30}) generated during a Subsequent 30-s sprint.	89
Figure 3.10	Effect of 4 and 20 min passive and active recovery on Mean power first 10 s (MP_{10}) generated during a Subsequent 30-s sprint.	90
Figure 3.11	Effect of 4 and 20 min passive and active recovery on mean power last 20 s (MP_{-20}) generated during a Subsequent 30-s sprint.	91
Figure 3.12	effect of 4 and 20 min passive and active recovery on mean power last 10 s (MP_{-10}) generated during a Subsequent 30-s sprint.	92
Figure 3.13	effect of 20 min passive and active recovery on Blood lactate concentrations.	93

Figure 3.14 Effect of 20 min passive and active recovery on blood H^+ concentrations. 94

Figure 3.15 Effect of 4 min passive and active recovery on blood lactate concentrations. .95

Figure 3.16 Effect of 4 min passive and active recovery on blood H^+ concentrations. 96

Figure 4.1 Effect of passive and active recovery between repeated 30-s sprints on peak power (PP) 111

Figure 4.2 Effect of passive and active recovery between repeated 30-s sprints on mean 30-s (MP_{30}). 112

Figure 4.3 Effect of passive and active recovery between repeated 30-s sprints on mean power first 10 (MP_{10}) 113

Figure 4.4 Effect of passive and active recovery between repeated 30-s sprints on mean power last 20 (MP_{-20}) 114

Figure 4.5 Effect of passive and active recovery between repeated 30-s sprints on mean power first 10 ($MP\text{-}_{\cdot 10}$). 115

Figure 4.6 Effect of passive and active recovery between repeated 30-s sprints on blood lactate levels. 116

Figure 4.7 Effect of passive and active recovery between repeated 30-s sprints on blood H^+ concentrations 117

List of Abbreviations

ADP	adenosine diphosphate
AMP	adenine monophosphate
ATP	adenosine tri-phosphate
Ca^{++}	calcium ions
h	hour
IMP	inosine monophosphate
K^{+}	potassium
min	minute
PE	prior exercise
P_i	inorganic phosphate
s	second
S.D.	standard deviation
S.E.M.	standard error of the mean
SR	sarcoplasmic recticulum
Na^{+}	sodium
$\dot{V}O_2$ max	maximum rate of oxygen consumption
$\dot{V}O_2$ peak	peak rate of oxygen consumption
Vmax	maximal velocity of muscle shortening

CHAPTER 1

Review of literature

1.1 Short intense sprint effort: a ubiquitous component of high-intensity sports

The performance of short, all-out, sprint efforts is integral to several sports. For example, a 100- or 200-m sprint is typical of this type of exercise. Likewise, 200- to 1000-m sprint cycling on the track involves an intense effort lasting between 11 and 60 s. Moreover, in most team sports such as rugby, soccer or hockey, the players engage in several short all-out sprints in the course of a game, with each sprint being interspersed with recovery periods of varying duration. Interval training, a pattern of activity characterised by successive sprint-recovery-sprint episodes, also relies heavily on short sprints.

Given that sprinting is central to such a broad range of physical activities, it is not surprising that a large volume of research has focused on the physiology and biochemistry of sprinting. One important issue that has not been as thoroughly investigated, however, is that of the optimal preparatory strategies to adopt in order to maximise all-out sprint performance. In particular, several questions remain to be answered with respect to the best pre-exercise warm-up strategies to optimise sprint performance. For instance, how long and intense should the warm-up be without jeopardising sprint performance? Another important question relates to the most effective preparatory strategies to adopt prior to a sprint performed a short time after an earlier sprint. Under these conditions, is it better for athletes to remain at rest or to exercise lightly to optimise sprint performance when recovery is of a short duration? The effects of recovery mode on subsequent sprint performance are still poorly defined. It is the primary goal of this thesis to address some of these issues and examine some of the preparatory strategies likely to optimise sprint performance. In the following sections, a brief description of the biochemistry of

sprinting and associated causes of fatigue will be provided. Then, the impact of warming-up on sprint performance will be examined, followed by a discussion of the recovery modes post-sprint that are most likely to optimise subsequent sprint performance.

1.2 Pattern of ATP production and fuel utilisation during a maximal sprint effort

During a maximal sprint effort, ATP demands can increase in excess of 100 fold and this is largely supported by the anaerobic production of ATP. During the initial few seconds of a sprint, both the stores of muscle glycogen and phosphocreatine are rapidly mobilised. These oxygen-independent ATP sources can support rates of ATP turnover as high as 10-15 mmol ATP$\cdot$kg dry muscle mass$^{-1}\cdot$s^{-1} over the initial 6 s of a maximal sprint cycling effort (Jacobs et al., 1983; Boobis et al., 1987; Gaitanos et al., 1993; Greenhaff and Timmons, 1998). Then, these oxygen-independent rates of ATP production fall progressively to 5 mmol ATP$\cdot$kg dry muscle mass$^{-1}\cdot$s^{-1} between the 6th and 30th s together with a fall in glycolysis and phosphocreatine mobilisation rates (Boobis et al., 1987; Gaitanos et al., 1993; Gastin, 2001). Despite the marked fall in oxygen independent rates of ATP regeneration, power output is reduced to a much lower extent, a finding consistent with muscles relying increasingly on aerobic ATP production. The contribution of aerobic metabolism during a 30-s maximal sprint effort has been reported to range between 18-46% (Kavanagh and Jacobs 1988; Medbo and Tababta, 1989; Bangsbo et al., 1990; Withers et al., 1991; Granier et al., 1995; Bogdanis et al., 1996), with its contribution increasing throughout the sprint (Kavanagh and Jacobs, 1988) to an extent that correlates with $\dot{V}O_{2max}$ (Bogdanis et al., 1996).

Despite the progressive increase in the contribution of aerobic ATP production as sprint duration increases, this does not prevent the total rate of ATP production in skeletal muscle to fall with time. The pattern of total ATP production rate is similar to that of power output, since in response to a single 30-s sprint cycling against a fixed load, power peaks within the first 2-3 s and thereafter declines as the sprint continues (Bogdanis et al., 1998, 1995, 1994, 1996; Neville, 1996). For this reason, close to 44% of the total work done during a maximal 30-s sprint is generated during the initial 10 s (Bogdanis et al., 1996). The subsequent rapid fall in power output during intense exercise thus raises the question of the mechanisms explaining the rapid onset of fatigue. It will be the purpose of the next section to describe briefly some of those mechanisms.

1.3 Mechanical manifestation and sites of fatigue during intense muscle contraction

1.3.1 Mechanical manifestation of fatigue

The rapid onset of muscular fatigue is an important factor limiting an all-out sprint performance, fatigue being defined here as an exercise-induced reduction in the voluntary maximal capacity to generate force or power output (Vollestad, 1997). Muscle fatigue associated with intense contraction manifests itself in a range of different ways. A decline in peak tetanic or twitch tension generally reflects the severity of fatigue (Thompson et al., 1992; Fitts, 1994). Moreover, the maximal velocity of muscle shortening (V_{max}), the peak rate of tension development, and the rate of relaxation also undergo a significant decline (Crow and Kushmerick, 1982; Thompson et al., 1992; Fitts, 1994), with the fall in the rate of relaxation being relatively greater than the fall in the rate of tension development (Fitts, 1994). Although both the fall in V_{max} and peak tension could contribute to the fall in muscle power during fatigue, muscle fatigue in response to intense contraction is, in

general, characterised by a greater fall in peak tetanic tension than V_{max} (Fitts, 1994).

1.3.2 Potential sites of fatigue: central or peripheral?

Fatigue has both central (Saltin et al., 1992; Taylor et al., 2000) and peripheral components (Nordlund et al., 2004). Central fatigue refers to the inability to fully activate a muscle voluntarily, with this muscle being capable of greater work output than that elicitated by the central nervous system (MacIntosh and Rassier, 2002; Nordlund et al., 2004). Central fatigue can result from a defect at several levels; (i) excitatory input to higher motor centres, (ii) excitatory drive to lower motoneurons, (iii) motoneuron excitability, and (iv) neuro-muscular transmission (Bigland and Ritchie, 1984). In particular, some of the possible contributory factors to central fatigue includes non-optimal facilitation from motor cortex (Taylor et al., 2000), decreased facilitation from muscle spindles and increased inhibition from afferent neurons (Garland, 1991; Garland and Kaufman, 1995), and desensitisation of the motoneurons (Kernell, 1969). In contrast, peripheral fatigue implies that the ability of the muscle to produce force or to contract or relax rapidly is reduced, irrespective of the intensity of the stimulus (Bigland-Ritchie 1984). As discussed later, several sites are targeted by peripheral fatigue, namely: (i) sarcolemmal and T-tubular excitability, (ii) excitation-contraction (E-C) coupling, (iii) contractile mechanisms, and (iv) metabolic energy supply (Nordlund et al., 2004).

Although controversies exists about the relative importance of central and peripheral factors in explaining the fatigue associated with an intense sprint effort, many studies have identified the peripheral sites as playing an important role (Fitts, 1994). Early findings by Merton, (1954), indicated that fatigue is not caused by action potential, neuromuscular junction or sarcolemma failure but rather by events within

the muscle itself, a conclusion corroborated later by the work of others (Bevan et al., 1992). Moreover, many recent studies have reported conditions where events that occur outside the muscle cell only play a complimentary role in fatigue (Bigland-Ritchie and Woods, 1984; Fitts, 1994). It is important to note that although fatigue in human muscle has been shown in some studies to be associated with a reduced motoneuron firing frequency, this has been explained on the basis that this might constitute a protective mechanism against fatigue (Bigland-Ritchie et al., 1981, 1983, 1986). However, since the fall in the maximal voluntary force of contraction does not always decline in parallel with that elicited by electrostimulation of muscle, this implies some failure of the central nervous system in fatigue (Ikai and Yabe, 1969; Bigland-Ritchie et al., 1986). This latter interpretation is supported by several studies that have reported that the loss of force production in intense activities can often be overcome by further voluntary effort (Ikai and Steinhaus, 1961; Asmussen and Mazin, 1978; Bigland-Ritchie et al., 1978). Overall, although there is evidence that central factors contribute to fatigue during intense exercise, disturbances within the muscle itself play an important role. For these reasons, the peripheral components underlying fatigue associated with an all-out effort will be examined briefly in the following sections.

1.3.3 Potential peripheral sites affected by fatigue

In theory, any of the events involved between the initiation of the action potential and contraction could be implicated as a site of peripheral fatigue. These events linking excitation-contraction coupling post-neuromuscular junction are well defined. Briefly, following its origin at the neuromuscular junction, the action potential is transmitted in both directions along the muscle fibre and then into the central regions of the muscle fibre along the transverse-tubules (T-tubules). The T-tubules, which are an extension of the plasma membrane and extra-cellular space, are in

close proximity to the sarcoplasmic reticulum (SR), where muscles store Ca^{++} at high concentrations. The action potential down the T-tubules activates several intra-membranous T-tubular proteins, referred to as dihydropyridine (DHP) receptors, which then undergo voltage-driven conformational changes that result not only in the inward movement of Ca^{++} inside the cell, but also in the release of Ca^{++} from the SR via the SR-Ca^{++} release channel (also known as ryanodine receptor). The Ca^{++}, thus released binds to troponin, which then initiates rapid cross-bridge cycling between the actin and myosin head, with the latter hydrolysing a large proportion of the ATP produced by the cell.

The intensity of peak force, the peak rate of tension development, V_{max} and twitch duration are all determined, in part, by key steps along the cross-bridge cycle. In particular, there is evidence that the conversion of the actomyosin complex from a low-force to a high-force state determines tension development, whereas V_{max} is limited by the dissociation of the actomyosin complex (Metzger et al., 1989). Disturbances in any of the aforementioned steps have the potential to be involved in fatigue, particularly in muscles rich in type II fibres as these are known to be less resistant to fatigue than type I fibres. For this reason, the next section will examine briefly the involvement of each of these sites in the development of peripheral fatigue associated with intense muscle contraction.

1.3.4 Impaired propagation of the action potential along the sarcolemma and T-tubules

In many (Stephen and Taylor, 1972; Hultman and Sjoholm, 1983; Bigland-Ritchie et al., 1983, 1986) but not all studies (Merton, 1954; Edwards and Lippold, 1956; Clamann and Broeker, 1979; Bigland-Ritchie et al., 1982), eletromyographic activity during intense muscle contraction decreases in parallel with force. These findings have been interpreted as evidence that muscle fatigue associated with intense

contraction results, in part, from a defect in the propagation of the action potential along the sarcolemma and T-tubules. Since the depolarisation of muscle membranes is accompanied by a loss of K^+ in the blood and accumulation of intracellular Na^+, it has been argued that fatigue results from these elevated extracellular K^+, reduced intracellular K^+ or increased K^+ conductance (Vyskocil et al., 1983; Sjogaard et al., 1985). These perturbations in K^+ and Na^+ balance across skeletal muscle membranes have been explained on the basis that the activity of the Na^+-K^+ATPase is not high enough to keep the membrane potential intact, thus resulting in muscle cell depolarization and reduced action potential amplitude (Sejersted, 1992; Fitts, 1994). This is probably worst at the T-tubule level, since the density of Na^+- K^+ATPase there is lower than in the sarcolemma. For this reason, it has been proposed that excessive T-tubule membrane depolarisation could lead to the inactivation of the DHP receptor (Luttgau et al., 1987; Brum et al., 1988) and inhibition of Ca^{++} flux through this protein (Brum et al., 1988; Pizarro et al., 1989), thus resulting in an impaired activation of the SR-Ca^{++} release channel, a fall in Ca^{++} availability, and the marked inhibition of force (Lindinger and Sjogaard, 1991; Sjogaard, 1991). Indeed, the morphological relationship between the SR-Ca^{++} release channel and the DHP receptor raises the possibility that a defect affecting the mechanical coupling between those proteins might be one important site of fatigue (Rios and Gonzalez, 1991; Rios and Pizarro, 1991).

Although the notion that excitation failure at the sarcolemma or T-tubule level causes fatigue is supported by some studies (Hultman and Sjoholm, 1983; Bigland-Ritchie et al., 1981, 1983, 1986), other findings support the view that it is events in the muscle itself that determine fatigue rather than sarcolemmal or T-tubules failure (Merton, 1954; Hultman and Sjoholm, 1983; Beavan et al. 1992), at least in the initial stage of fatigue (Bevan et al., 1992). In particular, Gyorke (1993) reported that despite a significant reduction in the amplitude of the Ca^{++} transient during fatiguing

stimulation, no significant drop in the T-tubular charge movement was detected. On this basis, it was concluded in agreement with others that the reduction in the Ca^{++} transient results from a direct inhibition of the SR-Ca^{++} release channel and not because of a disturbance involving T-tubular action potential or the DHP receptor (Ma and Zhao, 1994).

1.3.5 Ca^{++} release and reuptake from the sarcoplasmic reticulum

Several studies have reported that the amplitude of the Ca^{++} transient decreases as fatigue develops (Allen et al., 1989; Westerblad and Allen, 1991). Nonetheless, the question of whether this decrease is due to factors such as the blockade of the T-tubular action potential, a reduced intra-membranous T-tubular charge movement, an inhibition of the SR-Ca^{++} release channel or the depletion of releasable SR-Ca^{++} has been the subject of much debate. Nevertheless, Westerblad and Allen (1991) provided evidence that toward the end of a fatiguing stimulation *in vitro*, reduced Ca^{++} release becomes increasingly involved in the fall in force production. This finding was corroborated by another study where fatigue was shown to result initially from an impairment of the contractile apparatus, whereas the later stages of fatigue involved Ca^{++} handling (Lannergren and Westerblad, 1991). In addition, impaired Ca^{++} reuptake by the SR has been proposed to explain the delayed relaxation typical of fatigue. Overall, the importance of adequate Ca^{++} handling is crucial to muscle performance.

1.3.6 Cross-bridge cycling

Other major sites affected by fatigue are found at the cross-bridge cycling level. For instance, the observation that the sensitivity of muscle contraction to Ca^{++} is reduced in fatigue has been explained on the basis that Ca^{++} binding to troponin is impaired. Also, as discussed in Section 1.4.3, there is evidence that the transition from the low-force actomyosin.ADP.Pi complex to the high-force actomyosin.ADP state

decreases during fatigue. Finally, the fall in V_{max} associated with fatigue has been shown to involve a decrease in the dissociation of the actomyosin complex, the rate limiting step of V_{max}. Altogether, these falls in force and V_{max} result in a decrease in muscle power. The central role played by the different cross-bridge cycling steps in fatigue will be explored in more details in Section 1.4, as many of the mechanisms underlying muscular fatigue involve those sites.

1.4 Mediators and mechanisms of fatigue associated with an intense muscle contraction

The involvement of several potential intra-muscular sites (propagation of action potential, Ca^{++} release from the SR, and cross-bridge cycling) in fatigue raises the issue of the identity of the mechanisms at the origin of these defects. Since a bout of intense physical activity is associated with a rapid fall in muscle glycogen, phosphocreatine and ATP levels together with a rise in lactate, H^+, inorganic phosphate (Pi), creatine, ADP, AMP and IMP levels (Adams et al., 1990, Conley et al., 1999; Growther et al., 2002), albeit to varying extent, any of these metabolites could in theory contribute to the onset of fatigue. The following sections will examine which of these metabolites is most strongly associated with the onset of muscular fatigue during muscle contraction, and how this is achieved.

1.4.1 Lactate and fatigue

Since fatigue associated with high-intensity exercise occurs together with the accumulation of large amounts of lactate in muscle, for several years this metabolite was postulated to mediate fatigue (Sahlin, 1986). At the origin of this view were the findings that, regardless of whether *in vitro* or *in vivo* studies are performed, the reduction in force is strongly linked with the rise in lactate levels when the

stimulation of a muscle requires a high glycolytic rate. However, a role for lactate *per se* was challenged when it was reported that lactate ion has no direct effect on peak force generated by isolated fibres (Chase and Kushmerick, 1988; Posterino et al., 2001). Also, the marked dissociation between recovery of muscle force and intramuscular lactate levels reported in several studies (Sahlin et al., 1976; Sahlin, 1992) suggests that other mechanisms are involved. Finally, in conditions where a strong inverse correlation between lactate and force has been reported, this has been explained on the basis of the high correlation between lactate and free H^+ levels, which have been more strongly implicated in as a mediator of fatigue, as discussed in the next section. Nevertheless, that lactate might play a small role in mediating muscle fatigue is suggested by the recent observation that elevated lactate levels decrease marginally (<10%) Ca^{++} released from the SR in isolated muscle fibres (Dukta and lamb, 2000). Overall, lactate plays, at the very best, only a small role in fatigue.

1.4.2 H^+ and fatigue

It is noteworthy that the fall in pH associated with high-intensity exercise can be very pronounced, with muscle pH decreasing from resting level of 7.0 to 6.2, and blood pH from 7.4 down to 6.8 (Hermansen and Osnes, 1972). In support of H^+ playing a role in fatigue, is the finding that acidosis depresses tension in isolated muscle fibres (Donaldson and Hermansen, 1978; Fabiato and Fabiato, 1978; Donaldson, 1983; Metzger and Fitts, 1987; Cooke et al., 1988; Chase and Kushmerick, 1988; Metzger et al. 1992; Myburgh and Cooke, 1997), with fast twitch fibres being more sensitive. Low pH affects not only muscle tension, but also V_{max} in fast and slow twitch muscle fibres (Metzger and Moss, 1987; Cooke et al., 1988; Myburgh and Cooke, 1997). There are, however, large variations in the extent to which V_{max} is inhibited across different muscle fibre types with fatigue (Edman and Matiazzi, 1981). Overall, by inhibiting peak force and V_{max}, low pH inhibits maximal power. There is also

evidence that a fall in pH increases the levels of free cytosolic Ca^{++} required to initiate contraction (Donaldson and Hermansen, 1978; Fabiato and Fabiato, 1978; Godt and Nosak, 1989). Finally, the significant association between intramuscular pH and half relaxation time during recovery suggests that low pH increases relaxation time (Westerblad and Lannergren, 1991).

There are several mechanisms whereby low pH could decrease muscle power. Low pH can cause a fall in force by interfering with excitation-contraction coupling. In particular, the stimulatory effect of Ca^{++} on the SR-Ca^{++} release channel and DHP receptors is inhibited by excessive H^+(Rousseau and Pinkos, 1990; Ma and Zhao, 1994). This would be expected to reduce the amplitude of the Ca^{++} transient associated with fatigue and the production of force (Fitts, 1994). Moreover, the excitation–contraction coupling can be further impaired by a pH-mediated inhibition of Ca^{++} binding to troponin as there is evidence that the fall in force during fatigue results, in part, from the inhibitory effect of low pH on Ca^{++} binding to troponin C (Blanchard et al., 1984; Ball et al., 1994). In addition to affecting excitation-contraction coupling, low pH can also affect several of the cross-bridge cycling steps, with low pH inhibiting the transition from the low-force-actomyosin-ADP-Pi complex to the high-force-actomyosin-ADP state (Metzger and Moss, 1990). This is, in part, because this transition is accompanied by the release of H^+ and Pi, with a build-up in H^+ levels expected to favour the reversal of this reaction, thus inhibiting this transition, and reducing both the force per cross-bridge and the number of cross bridges (Metzger and Moss, 1990). It has also been proposed that a H^+-mediated inhibition of myosin-ATPase could be responsible for reducing the number of force-generating cross-bridges for a given Ca^{++} levels (Taylor, 1977) and for slowing the cross-bridge cycling rate, thus inhibiting V_{max} (Fitts and Holloszy, 1977; Thompson et al., 1992). This, coupled with the pH-mediated fall in force would explain the inhibition in peak power associated with fatigue. Finally, the inhibitory effect of low

pH on relaxation rate could result, in theory, from the combined effect of an altered cross bridge kinetics or an impaired ability of the SR to sequester Ca^{++}. Although Westerblab and Lennagren, (1991), reported that acidic pH does not alter the Ca^{++} transient, and, on this basis, concluded that the effect of pH on relaxation rate are mediated through cross-bridge kinetics, others have reported that H^+ compromises the functional capacity of the SR-Ca^{++}ATPase pump, thus explaining the lower relaxation time under conditions of low pH (Nakamaru and Schwartz, 1972; Byrd et al., 1989).

Since low pH inhibits glycogenolysis and glycolysis, this raises the issue of whether acidosis contributes to fatigue as a result of reducing ATP production rates (Krebs et al., 1959; Chasiotis et al., 1982; McCartney et al., 1986; Spriet et al, 1989). Thismechanism was first proposed by Hill in 1955, who showed that at a very low pH of 6.3, the formation of lactate ceases. Subsequently, low pH was shown to inhibit both glycolysis and glycogenolysis by inhibiting phosphofructokinase and glycogen phosphorylase, respectively (Krebs et al., 1959; Chasiotis et al., 1982). However, there is little evidence that limiting ATP production rates would reduce ATP to levels that limit ATP utilisation rates (Edwards et al., 1972; Sahlin et al., 1978). This is because most studies have shown that cytosolic ATP levels rarely drop below 50% of pre-fatigue levels during high-intensity exercise (Fitts, 1992; Thompson and Fitts, 1992), with these levels being above those required for optimal activity of muscles ATPase. Also, there is evidence that the inhibitory effects of H^+ on glycogenolysis and glycolysis are acted against by the rise in the levels of activators of phosphorylase and PFK, such as IMP, AMP and Pi (Chasiotis et al., 1982; Ren and Hultman, 1989). It is important to stress, however, that it is possible that ATP near muscle ATPases might fall to suboptimal levels and thus affect ATP utilisation rates. Finally, as discussed later (Section 1.4.4), a fall in ATP levels has

the potential to elicit fatigue by mechanisms other than limiting ATP provision for muscle ATPases.

Despite the aforementioned observations suggesting that low pH is an important cause of fatigue, there is compelling evidence that this might not be the case (Mainwood and Renaud, 1972; Renaud, 1986; Westerblad and Allen, 1992). Indeed, several studies have shown that, as for lactate, intracellular pH and force/tension recover at different rates following fatigue (Metzger and Fitts, 1987; Sahlin and Ren, 1989), with some reporting that the early recovery of force occurs while intramuscular pH is either stable or falling due to phosphocreatine synthesis (Sahlin and Ren, 1989; Metzger et al., 1989). Also, it has been reported that during maximal voluntary isometric contraction, the early fall in force is accompanied by an increase or no change in H^+ levels rather than a fall in pH (Vollestad et al., 1988; DeGroot et al., 1993; Saugen et al., 1997), thus suggesting that mechanisms other than H^+ mediate fatigue. More importantly, the studies that have reported an inhibitory effect of low pH on force and V_{max} in isolated muscle fibres have been performed at unphysiologically low temperature to preserve the integrity of these muscle preparations (Cooke et al., 1988; Chase and Kushmerick, 1988). When similar experiments are performed at physiological temperature, low pH has little effect on force production in intact or skinned muscle fibres as well as in whole muscle (Pate et al., 1995; Wiseman et al., 1996; Westerblad et al., 1997). Some studies have even reported that acidification results in a rise in tetanic force at physiological temperature (Ranatunga, 1987). In this regard, Pedersen and colleagues (2004) recently provided evidence that low pH protects muscle against fatigue by preserving muscle excitability. Others have reported that acidification has little or no effect on V_{max} of intact or skinned muscle fibre kept at close to physiological temperature (Pate et al., 1995; Westerblad et al., 1997). Finally, the view that low

pH mediates fatigue by inhibiting phosphofructokinase has also been challenged (Dobson et al., 1986). Overall, since low pH at physiological temperature has little effect on cross-bridge function and because of the poor temporal association between changes in pH and both force and V_{max}, this suggests that other mechanisms probably play a more important role in causing fatigue.

1.4.3 P_i and muscle fatigue

Given the many reports of a poor association between fatigue and intramuscular lactate and pH, and since intense exercise is accompanied by a marked fall in phosphocreatine levels as well as a large rise in P_i levels, and that both P_i and PCr levels show a strong correlation with fatigue during and after muscle contraction (Dawson et al., 1977, 1978; Crow and Kushmerick, 1982; Dawson, 1988; Millar and Homsher, 1990; Weiner et al., 1990), this suggests that these metabolites could be prime mediators of fatigue. On thermodynamic grounds, it is to be expected that the maximum rate of ATP production from phosphocreatine breakdown decreases as phosphocreatine levels fall. However, as discussed earlier, it is unlikely that such a decrease in ATP production rate would limit ATP utilisation, unless ATP near highly active myosin ATPases is present at very low levels (Tokiwa and Tonomara, 1965; see section 1.4.4 for further discussion on this issue). This suggests that other mechanisms play a more important role in the development of fatigue.

A strong link between fatigue and the levels of P_i has been established over the past two decades, and led many to the view that P_i levels play a more important role than pH as a mediator of fatigue (Cooke et al., 1988; Cooke and Pate, 1989; Steinen et al., 1992; Westerblad, 2004). This is supported by the findings that P_i levels recover from fatigue with a time course similar to that of recovery of muscle force (Miller et al., 1988; Thompson and Fitts, 1992). Moreover, high P_i levels inhibit the maximal force of skinned muscle fibres (Cooke and Pate, 1985; Kawai, 1986; Chase and

Kushmerik, 1988; Martyn and Gordon, 1992), the effect of P_i on tension being greater in fast than in slow twitch fibres (Stienen et al., 1992) and consistent with fast twitch fibres being more susceptible to fatigue (Barclay, 1996; Hamada et al., 2003). However, it must be stressed that it is generally the case that P_i has no effect on V_{max} (Cooke and Pate, 1985) and little or no effect on myosin ATPase (Cooke and Pate, 1985; Kawai et al., 1987; Potma and Stienen, 1996), unless ATP are below physiological levels (Pate and Cooke, 1989).

Several mechanisms have been proposed to explain how excessive P_i levels inhibit force. Elevated P_i levels have been shown to decrease Ca^{++} release from the SR during the final stages of fatigue (Fryer et al., 1995; Posterino and Fryer, 1998; Duke and Steele, 2001). This reduced Ca^{++} release has been proposed to result from the formation of a Ca^{++}- P_i precipitate in the SR, which would decrease the amount of free Ca^{++} available for release from the SR (Posterino and Fryer, 1998; Kabbara and Allen, 1999). This is supported by the observation that P_i can accumulate inside the SR when exposed to elevated levels of P_i (Laver et al., 2001). The problem with this interpretation is that it does not explain the transient rise in cytoplasmic Ca^{++} levels in the early stage of fatigue. In this regard, it has recently been shown that high P_i levels together with low ATP increase Ca^{++} release from the SR via reversal of the reaction normally catalysed by the SR Ca^{++}ATPase pump (Duke and Steele, 2000; Macdonald and Stephenson, 2001), thus causing a subsequent fall in releasable Ca^{++}. Also, the inhibition of Ca^{++} release from the SR by elevated P_i can also occur in the absence of any detectable precipitation of Ca^{++}- P_i, an effect that is enhanced by elevated free Mg^{++} levels (Duke and Steele, 2001b). Considering the effect of elevated P_i levels on the SR Ca^{++}ATPase pump, it is not surprising that P_i also inhibits Ca^{++} uptake by this pump (Duke and Steele, 2000), thus explaining the decreased relaxation rate associated with fatigue. This inhibition is explained on the basis that the high P_i levels together with the increased ADP and decreased ATP

levels that normally accompany fatigue would be expected to reduce the free energy of ATP hydrolysis to an extent that inhibits the activity of the SR- Ca^{++} ATPase pump (Dawson et al., 1980).

Irrespective of its effects on Ca^{++} handling, P_i has been implicated in the fall in force production via a direct effect on cross-bridge cycling. Indeed, normally P_i is released from the actomyosin complex during the transition of the low-force actomyosin-ADP-P_i complex to the high-force actomyosin-ADP state during cross bridge cycling (Dantzig et al, 1992). Since elevated levels of P_i favours the reversal of this transition, force is expected to decline due to an increased proportion of cross-bridges in the low-force state (West et al., 2003). Not only is this mechanism expected to reduce force production, but also Ca^{++} sensitivity due to a lower number of cross-bridges in the force-generating state for a given Ca^{++} levels. The lack of effect of P_i on V_{max} is consistent with the absence of effect of Pi on actomyosin dissociation.

Despite the aforementioned relationship between P_i and fatigue, several studies have reported a stronger relationship between the decline in force and the increase in P_i in its diprotonated form, $H_2PO_4^-$ (Miller et al., 1987; Dawson et al., 1988; Wilson et al., 1988; McCully et al., 1989; McCully et al., 1991), with many identifying $H_2PO_4^-$ as a major cause of fatigue (Wiener et al., 1990; Steele and Duke, 2003; Westerblad, 2004). One study has also shown that the altered relaxation time with fatigue is highly correlated with $H_2PO_4^-$, both during the development of and recovery from fatigue (Bergstrom and Hultman, 1988). Interestingly, since the proportion of $H_2PO_4^-$ rises with a fall in pH, this implies that H^+ plays an important indirect role in mediating fatigue (Nosek et al., 1987). However, others have reported that the effect of total P_i is the same at both low and high H^+ levels, and

that for this reason the reduction in force in response to P_i cannot be entirely attributed to $H_2PO_4^-$ (Chase and Kushmerick, 1988; Cooke et al., 1988).

It is important to stress that not all studies supports the view that $H_2PO_4^-$ or P_i is the primary cause of fatigue in skeletal muscle, since the relationship between P_i or $H_2PO_4^-$ and force is not always a close one (LeRumeur et al., 1990. For instance, Miller and colleagues (Miller et al., 1988; Weiner et al., 1990) observed that the increase in P_i during exercise is more rapid than the decline in peak force. Moreover, the depressive effect of P_i on force is also reduced as temperature rises to physiological levels; however the effect of P_i on force is still important at high temperature (Coupland et al., 2001). Finally, since high levels of P_i are without any effect on V_{max}, this implies the participation of other mechanisms of fatigue.

1.4.4 Adenylates and fatigue

During intense muscle contraction, free ADP levels can increase in excess of 10 fold despite lower relative changes in total ADP content (Sahlin and Ren, 1989). Elevated but physiological levels of free ADP have been shown to increase maximal force (Cooke and Pate, 1985; Godt and Nosek, 1989; Metzger, 1996) and to decrease V_{max} as well as myosin ATPase (Cooke and Pate, 1985; Godt and Nosek, 1989; Metzger, 1996a) and SR Ca^{++} ATPase activities (Macdonald and Stephenson, 2001), some of these effects being more pronounced in fast twitch muscle fibres (Chase and Kushmerick, 1995). The activation of muscle force by ADP is explained on the basis that the release of ADP from the actomyosin complex is a prerequisite for ATP binding to myosin and subsequent dissociation of the actomyosin complex after a power stroke. The presence of elevated ADP levels inhibits ADP release, thus preventing ATP binding. As a result of this ADP-mediated fall in the rate of cross-bridge detachment (Westerblad et al., 1998; Dantzig et al., 1991), more actomyosin remains in the high force state and maximal force increases, but V_{max} is

depressed because the dissociation of the actomyosin complex is the rate limiting step of V_{max} (Weiss et al., 2001). Although some authors have concluded that these effects are small in the presence of physiological levels of ADP (Godt and Nosek, 1989; Chae and Kushmerick, 1995), it has recently been shown that the effect of physiological levels of ADP on tension and myosin ATPase requires low concentrations of ATP (Karatzferie et al., 2003). Moreover,since intracellular ADP diffusion is limited, the levels of ADP near sites of high ATPase activities could be higher than the concentrations alleged to be physiological (Korge and Campbell, 1995).

The fall in ATP levels during intense muscle contraction is also believed to mediate fatigue. This decrease in ATP concentrations during intense exercise is in general modest (less than 50%; Bogdanis et al., 2004), but, as discussed previously (Section 1.43.), since the Km for myosin ATPase is low, even a large fall in ATP is expected to have little effect on contraction. However, if the possibility of spatial differences in ATP and ADP concentrations in muscle is taken into consideration, lower ATP and higher ADP levels are expected close to highly active ATPase sites, and thus could have an effect on ATP utilisation rates. As already alluded to in Section 1.4.3, a fall in ATP levels could also affect muscle function indirectly. Indeed, given that P_i entry into the SR is inhibited by ATP (Posterino and Fryer, 1998), low ATP levels has been proposed to be a prerequisite for P_i entry into SR and associated Ca^{++}- P_i precipitation. This finding is consistent with the observation that P_i enters the SR with a delay during intense contraction. A fall in ATP could also interfere with the SR Ca^{++} release channel. Although it has been reported that a physiological fall in ATP levels has only a small effect on SR Ca^{++} release (Blazey and Lamb, 1999) and on SR Ca ATPase activity (Korge, 1995), more recent studies have shown otherwise. What was overlooked in the initial studies on this question is that the Mg^{++} released from MgATP complex during ATP hydrolysis could also

participate in the development of fatigue. Indeed, it has recently been shown that elevated levels of Mg^{++} enhance the inhibitory effect of ATP depletion on SR Ca^{++} release (Stepheson et al., 1998; Blazev and Lamb, 1999b). Moreover, the combined inhibitory effect of reduced ATP and elevated Mg^{++} on SR Ca^{++} release channel is enhanced further by AMP and IMP binding to this channel (Duke and Steele, 1998; Blazev and Lamb, 1999a,b). Finally, elevated Mg^{++} levels have also the additional effect of increasing the inhibitory effect of Pi on the SR Ca^{++} release channel.

1.5 Preparatory strategies to optimise sprint performance: Impact of "warming-up"

The inclusion of a warm-up session prior to a sprint event is a widely accepted practice among most coaches, trainers and sprint athletes to improve sprint performance and reduce the risks of injury. In general, a warm-up takes the form of mild prior exercise (PE) performed before a sprint, but passive heating of muscle by immersion in warm water or using hot shower or heating pads is also another means for warming-up muscle. Although PE is also referred to in the literature as active warm-up, we will minimise the use of the expression "warm-up" because it is a term that is not as theory neutral as PE.

1.5.1 Effect of PE on sprint performance

Prior exercise of moderate intensity has been reported to improve sprint performance. In particular, moderate intensity PE (<60% $\dot{V}O_{2max}$), with or without a short rest before a subsequent sprint, results in an increase in cycling peak power (Bergh and Ekblom, 1979; Dolan et al., 1983; Dolan and Sargeant, 1983; McKenna and Green, 1987; Sargeant and Dolan, 1987; O'Brian et al., 1997; Racinais et al., 2005), average cycling power output (Inbar and Bar-Or, 1975; McKenna and Green,

1987; Sargeant and Dolan, 1987; O'Brian et al., 1997; Burnley et al., 2005), cycling average velocity (Inbar and Bar-Or, 1975; Bergh and Ekblom, 1979), cycling time trials (Asmussen and Boje, 1945; Hajogloo et al., 2005) and cycling acceleration (De Bruyn-Prevost and Lefevbre, 1980). Moderate intensity PE has also been reported to improve time trials in running (Andsel, 1982) and swimming (Thompson, 1958).

Despite the large body of evidence supporting the benefit of PE on sprint performance, some studies have failed to show any positive effect of PE (Hipple, 1955; Massey et al., 1961; Skubic and Hodgkins, 1957; Genovely and Stanford, 1982; Hawley et al., 1989). Indeed, there are conditions where the intensity or duration of PE as well as the length of the rest period between PE and sprinting have either no effect or are detrimental to performance. For instance, PE of short duration and low intensity has no effect on sprint performance (Skubic and Hodgkins, 1957). Moreover, PE performed at high intensity (>60% $\dot{V}O_{2max}$) decreases subsequent cycling acceleration (De Bruyn-Prevost and Lefevbre, 1980) and has either no effect (Hawley et al., 1989) or results in a fall in cycling peak power if such an intense PE is performed immediately before sprinting (Margaria et al., 1971; Dolan and Sargeant, 1983; Genovely and Stamford, 1982; Sargeant and Dolan, 1987), with an inverse relationship existing between PE intensity and subsequent sprint power (Dolan and Sargeant, 1983).

The length of time between PE and the start of sprinting is another condition that can affect sprint performance. Although, as discussed above, intense PE impairs subsequent sprint performance, this is not the case if a short rest (5-6 min) is allowed between such a PE and sprinting. Under these conditions, power output is no more impaired as it either remains at levels comparable to those attained without PE (De Bruyn-Prevost and Lefevbre, 1980; Hawley et al., 1989; Burnley et al., 2005) or increases to levels above those attained in the absence of PE (Dolan and

Sargeant, 1983; Sargeant and Dolan, 1987). Similarly, intense PE followed by a 5-min rest has no detrimental effect on running time in a subsequent sprint (Hipple, 1955). In contrast, the benefits of moderate-intensity PE on sprint performance are impaired when a short rest is allowed between PE and sprint cycling (De Bruyn-Prevost and Lefevbre, 1980).

Given the many variables affecting the impact of PE on sprint performance, a better understanding of the mechanisms whereby PE enhances sprint performance is required in order to explain why its efficacy depends on factors such as the intensity and duration of PE and the time elapsed between PE and sprinting. As will be discussed in the next sections, the benefits of PE on sprint performance have been attributed to (a) temperature-related mechanisms, (b) cardiorespiratory-related mechanisms, and (c) non-temperature/cardiorespiratory-related mechanisms.

1.5.2 Temperature-mediated effects of PE on sprint performance of short duration

It well established that exercising muscles generate considerable heat, with the rise in muscle temperature being proportional to the relative work rate (Saltin et al., 1968) and dependent on the duration of exercise. Typically, muscle temperature during exercise rises more rapidly than rectal temperature and attains stable levels within 10-20 min (Saltin et al., 1968). This increase in muscle temperature *per se* has the capacity to improve sprint performance. Indeed, passive warm-up achieved by prolonged limb immersion in hot water results in an increase in cycling peak power in proportion to muscle temperature (Berg and Ekblom, 1979; Dolan et al., 1983), with this improvement ranging from 2.7-4%/°C (Dolan et al., 1983; Sargeant, 1987). On the other hand, in response to cooling, peak power decreases in proportion to muscle temperature (Sargeant, 1987). A number of studies have also

reported that passive warm-up improves time trials for sprint running and swimming (Asmussen, 1945; Thompson, 1950; Carlile, 1956). Although the work of O'Brian and colleagues (1997) suggests that PE offers a better means to improve sprint performance than passive heating, muscle temperature was not measured post-PE in this study. For this reason, the possibility remains that the differences in effectiveness between PE and passive warm-up protocol might have resulted from differences in muscle temperature between those protocols (O'Brien et al., 1997). Overall, the findings that passive warm-up improves sprint performance raise the question of whether there were temperature-related mechanisms involved.

Given that the rates of chemical reactions increase with temperature, it is to be expected that one mechanism whereby a rise in muscle temperature enhances power output during a sprint is via a Q_{10} effect on the rates of non-oxidative and oxidative ATP generation. That this is the case with respect to non-oxidative ATP generation is suggested by the observation that a rise in muscle temperature enhances the rates of muscle phosphocreatine breakdown, glycogenolysis and glycolysis during exercise (Fink et al. 1975; Febbraio et al. 1996; Febrraio; 2000). It is less clear, however, to what extent oxidative ATP generation benefits from increasing muscle temperature. Although the rate of oxygen consumption by isolated mitochondria increases with temperature (Brooks et al. 1971), the finding that PE or passive muscle warming does not affect $\dot{V}O_2$ kinetics in young adults (Ingjer and Stromme, 1979; Koga et al., 1997; Burnley et al., 2000; Koppo and Bouckaert, 2001; Koppo et al., 2002; Burnley et al., 2002) suggests that elevated temperature might interfere with mitochondrial ATP generation. In support of this view, the poor thermal dependency of oxidative ATP generation has been explained by the uncoupling of oxidative phosphorylation in mitochondria as temperature rises (Brooks et al., 1971). For these reasons, the beneficial effect of increased muscle

temperature on sprint performance is unlikely to be mediated primarily by a rise in oxidative ATP generation.

Given that an increase in temperature results in a rise in the dissociation of oxyhaemoglobin (Barcroft and Key, 1909) and oxymyoglobin (Theorell, 1934) in addition to increasing both vasodilation of blood vessels and muscle blood flow (Barcroft and Edholm, 1943), the associated expected increase in oxygen delivery to skeletal muscle would be expected to improve $\dot{V}O_2$ kinetics by increasing oxidative ATP production, and thus delay fatigue. However, for this interpretation to hold, $\dot{V}O_2$ kinetics would have to be limited by oxygen delivery. That this is not the case in young healthy individuals is suggested by the absence of any effect of passive muscle heating or PE on $\dot{V}O_2$ kinetics during heavy exercise (Ingjer and Stromme, 1979; Koga et al., 1997; Burnley et al., 2000; Koppo and Bouckaert, 2001; Koppo et al., 2002; Burnley et al., 2002)

Other mechanisms have been proposed to explain the positive effect of increasing muscle temperature on sprint performance of short duration, but have been the subject of little research effort. An increase in muscle temperature could improve sprint performance by decreasing muscle viscous resistance (Wright and Johns, 1961) and lowering the stiffness of muscle fibres during contractions (Butchal et al., 1944). A rise in muscle and body temperature could also improve performance by increasing the transmission speed of nerve impulses (Karvonen, 1992). However, there is evidence that elevated core temperature impairs voluntary muscle activation (Thomas et al., 2006). The relative importance of these and other temperature-dependent mechanisms remains to be determined.

1.5.3 PE-mediated mobilisation of the cardiorespiratory system prior to a sprint

There is evidence that PE can improve sprint performance by mobilising the cardiorespiratory system prior to exercise if a rest period between PE and the start of the sprint is short enough for $\dot{V}O_2$ to remain above resting level. This mobilisation is consistent with a greater aerobic contribution (Ingjer and Stromme, 1979; Stewart and Sleivert, 1998; McCutcheon et al., 1999) and a lower oxygen deficit at the onset of exercise when intense exercise is performed immediately after PE (Gutin et al., 1976; Andzel, 1982; McCutcheon et al., 1999). For these reasons, such a practice is expected to increase the time to exhaustion and to improve sprint performance, since less work is completed anaerobically, leaving more of the anaerobic capacity for later in the task. In support of this view, PE has been reported to reduce both non-oxidative ATP supply (Golnick et al., 1973; Ingjer and Stromme, 1979) and oxygen deficit (di Prampero et al., 1970; Gutin et al., 1976; McCutcheon et al., 1999) during subsequent intense exercise (Gerbino et al., 1996; Burnley et al., 2000; Rossiter et al., 2001; Koppo and Bouckaert, 2001; Campbell et al., 2002), and to result in a lesser blood lactate accumulation. Moreover, PE followed by little or no rest before a sprint improves in general mean power output (Inbar and Bar-Or, 1975; McKenna and Green, 1987) or time trial performance (Andzel, 1982; McKenna and Green, 1987). However, since pre-exercise muscle temperature was not controlled in these studies, the positive effect of PE might have been mediated by a rise in muscle temperature. Better indirect support for the benefit of pre-exercise mobilisation of the cardiorespiratory system on exercise performance is suggested by the fall in sprint performance when a 5-min rest is allowed between PE and sprinting (DeBruyn-Prevost and Lefevbre, 1980; Hawley et al., 1989; Ozyener et al., 2001; Bishop et al., 2001), since this time period is long enough for $\dot{V}O_2$ to return to basal levels, but not muscle or whole body temperature (Ozyner et al., 2001). It

follows from these observations that increasing the mobilisation of the cardiorespiratory system prior to a sprint is likely to be beneficial as long as the rest interval before the subsequent effort is of short duration. However, since some studies have reported that PE followed by a rest still improves performance (Inbar and Bar-Or, 1975; Obrien et al., 1997), this suggests that mechanisms other than mobilisation of the cardiorespiratory system are involved (e.g. increase in muscle temperature).

1.5.4 $\dot{V}O_2$ kinetics-mediated effects of PE on sprint performance of short duration

During a maximal sprint effort, both non-oxidative and oxidative energy supplies contribute to intramuscular ATP turnover (Bogdanis et al., 1998; Krustrup et al., 2001). Since at the onset of an intense bout of exercise there is a lag between the sudden increased rate of ATP utilisation and mitochondrial ATP generation (Grassi et al., 1996; Bangsbo et al., 2000; Hogan, 2001), the maintenance of steady levels of ATP levels and turnover is achieved primarily by the activation of glycolysis and phosphocreatine breakdown. Arguably, the longer it takes for the rise in mitochondrial oxygen consumption to meet the increased ATP demands at the start of exercise, the larger the reliance on non-oxidative ATP provision, and thus the earlier the onset of fatigue associated with phosphocreatine depletion and accumulation of inhibitory levels of H^+ and Pi. This delay in increasing mitochondrial oxygen consumption is closely reflected by pulmonary $\dot{V}O_2$ kinetics (Barstow, 1990; Grassi et al., 1996; Rossiter et al., 1999).

It has been proposed that moderate intensity PE could result in the accumulation of vasoactive metabolites causing vasodilation and improvement in $\dot{V}O_2$ kinetics. There is, however, little evidence in support of the view that the positive effects of

moderate intensity PE on sprint performance results from improved $\dot{V}O_2$ kinetics. Indeed, several studies have reported that PE of moderate intensity has little or no effect on $\dot{V}O_2$ kinetics and acid-base response to a subsequent bout of intense exercise (Gerbino et al., 1996; MacDonald et al., 1997; Burnley et al., 2000). These findings should not come as a surprise given that oxygen delivery has been reported to be in excess of oxygen demands at the onset of intense exercise (Grassi, 1996; Grassi et al., 1998; Bangsbo et al., 2000), with oxygen delivery not limiting exercise capacity (Bangsbo et al., 2000).

Although there is little evidence that improved $\dot{V}O_2$ kinetics mediates the beneficial effects of moderate intensity PE on sprint performance, earlier studies by Gerbino and colleagues (1996) suggested that intense but not moderate intensity PE followed by a 6-min rest improves the overall $\dot{V}O_2$ kinetics of a subsequent bout of intense exercise. This finding was deemed important as it had the potential to explain the findings by others that intense PE followed by a short rest improves power output during a subsequent sprint (Dolan and Sargeant, 1983; Sargeant and Dolan, 1987). Gerbino and colleagues (1996) proposed that the residual acidosis caused by the intense PE improves oxygen delivery via an acidosis-mediated oxyhaemoglobin dissociation and a rise in the vasodilation of the vascular beds of the exercising muscles. Subsequent studies, however, showed that intense PE does not affect $\dot{V}O_2$ kinetics (Bohnert et al., 1998; Fukuba et al., 1998; Burnley et al., 2000; Burnley et al., 2002; Fukuba et al., 2002; Koppo et al., 2003) although it reduces the amplitude of the $\dot{V}O_2$ slow component (Bearden and Moffat, 2001; Koppo and Bouckaert, 2001; Scheurmann et al., 2001; Fukuba et al., 2002; Perrey et al., 2003). It is important to note, however, that there is evidence that PE improves $\dot{V}O_2$ kinetics in older humans (Scheuermann et al., 2002), possibly via an improved rate of O_2 utilisation by the muscle (Bell et al., 2001). Overall, it seems

unlikely that PE improves sprint performance in healthy young adults via alterations in VO_2 kinetics.

1.5.5 Temperature- and cardiorespiratory-independent mechanisms of PE effect on sprint performance

Mechanisms independent of muscle temperature and oxygen availability are also likely to explain the positive effect of PE on sprint performance. For instance, there is evidence that post-activation potentiation, which refers to the transient increase in muscle contractile performance following previous activity (Sale, 2002), improves performance (Vandervoort et al., 1983). The benefit of PE on sprint performance could also be the result of a placebo effect (Massey et al., 1961), but the relative importance of this mechanism relative to those described above remains to be established.

1.5.6 Mechanisms underlying the negative effect of PE on sprint performance

The series of mechanisms described above help to make sense of the observations that the positive effect of PE depends on factors such as the intensity and duration of PE and the length of the rest between PE and sprinting. As described above, given that the mobilisation of the cardiorespiratory system last only for a few minutes after PE due to the rapid return of $\dot{V}O_2$ to basal or near basal levels post-PE, a long delay between PE and sprinting would be predicted to counter the benefit of pre-exercise mobilisation of the cardiorespiratory system. As a result, little improvement in sprint performance would be predicted to occur by combining PE and a rest. On the other hand, if no delay is allowed, but the intensity of PE is high enough to result in marked phosphocreatine breakdown, sprint performance would also decrease as mentioned above, unless a short recovery is allowed for phosphocreatine

resynthesis to take place post-PE. If PE, on the other hand, were to be long enough to mobilise the cardiorespiratory system, but too short to cause a marked increase in muscle temperature, its effect on sprint performance would also be expected to be sub-optimal.

1.5.7 Relative contributions of temperature and cardiorespiratory-dependent mechanisms in determining the positive effect of PE on sprint performance

Considering the central role played by muscle temperature and mobilisation of the cardiorespiratory system in explaining the benefits of PE on sprint performance, it is noteworthy that to date there has been little research effort aimed at evaluating the relative importance of muscle temperature and pre-exercise cardiorespiratory mobilisation in explaining the positive effect of PE on power output during a sprint. Moreover, there has been no attempt at dissecting out the effect of PE on the temporal pattern of response of power output during a sprint. This latter issue is an important one, since the relative contributions of the fuel systems supporting the energy demands of skeletal muscle during a sprint change rapidly over time, with phosphocreatine contribution being at its highest at the onset of exercise and that of oxidative ATP supply increasing progressively over time (Bogdanis et al., 1998). Because the responses of these processes to temperature differ markedly (Section 1.5.2), this raises the possibility that the mechanisms whereby PE affects power output during a sprint might also differ as a sprint progresses. As mentioned later, it will be one of the primary goals of this thesis to examine the effect of PE on the temporal pattern of response of power output during a sprint by controlling either temperature or cardiorespiratory mobilisation.

1.6 Preparatory strategies to optimise sprint performance after an earlier sprint

In the previous section, it was outlined that PE provides a better preparatory strategy for optimal sprint performance than to only rest. Often, however, athletes have to engage in a sprint shortly after one or several sprints. This raises the question of whether it is preferable to rest or exercise after a single sprint or between multiple sprints in order to maximise the power output of a subsequent sprint. In other words, is it better to rest or to be physically active between consecutive sprints in order to optimise sprint performance? This section will examine whether optimal recovery of sprint performance capacity after a single sprint is better achieved with active or passive recovery between sprints.

Given that H^+ is one of the factors alleged to cause fatigue, it has been argued that the rate at which it is removed after a sprint is likely to be critical for the speed of recovery of subsequent sprint performance capacity. For this reason, many studies have focused their efforts toward developing protocols that increase the rates of blood lactate and H^+ disappearances following intense exercise (Gisolfi et al., 1966; Hermansen and Stensvold, 1972; Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Stamford et al., 1978b; Dodd et al., 1984; Signorile et al., 1993; Thiriet et al., 1993; Balsom et al., 1994; Ahmaidi et al., 1996; Monedoro and Donne, 2000; Spierer et al., 2003, Dupont et al., 2003). One such a strategy of recovery that has attracted much research effort is active recovery, as it has been found to be superior to passive recovery in enhancing lactate clearance (Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Dodd et al., 1984). Typically, active recovery involves performing exercise of low intensity (30-40% $\dot{V}O_{2max}$) during recovery from a bout of high-intensity exercise, as this results in rapid rates of lactate disposal (Weltman et al., 1977; Thiriet et al., 1993; Signorile et al., 1993; Ahmaidi et al., 1996; Bogdanis et al., 1996; Felix et al., 1997).

There are a number of possible explanations that have been put forward to explain the increased rates of lactate and H^+ disappearance during active recovery. Firstly, a rise in blood flow to muscles during this recovery mode has been suggested to facilitate lactate transport from active muscles to removal sites such as the inactive muscles, the liver and the heart (Bonen and Belcastro, 1976; Choi et al., 1994). Secondly, an increase in blood flow to muscles during active recovery provides the oxygen for lactate oxidation within the working muscles, thus resulting in high rates of lactate disposal (Brooks, 1986; Peters-Futre et al., 1987).

Although considerable information can be found in the literature concerning the effects of active and passive recovery on the rate of lactate disposal (Gisolfi et al., 1966; Hermansen and Stensvold, 1972; Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Stamford et al., 1978b; Dodd et al., 1984), most of these studies have not examined the effect of active recovery on subsequent exercise performance capacity. In those studies that have examined the effect of active recovery on exercise performance, the impact of this mode of recovery on subsequent performance is either small (<3%; Weltman et al., 1977; Thiriet et al., 1993; Bogdanis et al., 1996; Felix et al., 1996) or undetectable (Weltman et al., 1979; Bond et al., 1991; Signorile et al., 1993; Ahmaidi et al., 1996; Monedero and Donne, 2000; Dupont, 2003).

Several limitations in the experimental designs of most of the aforementioned studies may explain the reported absence of a large effect of active recovery. In the study of Weltman and co-workers, (1979), no significant improvement in work output was noted in response to active recovery despite a marked effect on lactate levels. In this study, 5 min of intense exercise was repeated after 20 min of either passive or active recovery consisting of cycling at an intensity below anaerobic threshold (40% $\dot{V}O_2max$) or above anaerobic threshold (65% $\dot{V}O_{2max}$). No improvement in

work output was reported despite faster rates of lactate disposal during active recovery. A similar study carried out by Bond and colleagues, (1991), reported that 20 min of active recovery at 30% $\dot{V}O_{2max}$ performed after a 1-min cycling at 150% $\dot{V}O_{2max}$ enhanced the rates of lactate disposal compared to passive recovery, but did not improve isokinetic muscle functions (peak torque, total work output). It is important to note that in this study, the initial bout of exercise did not increase lactate to high levels (close to 9 mM) and lactate levels fell to 3.5 and 7.1 mM after 20 min of active and passive recovery, respectively. One may propose on the basis of these low plasma lactate levels that intramuscular H^+ levels were probably too low to influence isokinetic muscle functions. Similar considerations apply to the study of Weltman and co-workers, (1979). More importantly, however, the absence of benefit of active recovery might be simply explained on the basis that 20 min of recovery was long enough for near complete recovery of sprint performance capacity, thus explaining the absence of any effect of active recovery. Had performance measures been taken much earlier during recovery, significant differences between active and passive recovery might have been observed.

The absence of significant improvements in performance in response to active recovery has also been reported for exercise bouts of shorter duration (6 seconds; Signorile et al., 1993). It is important to note that this study was concerned with comparing the effect of active recovery to passive recovery on repeated sprint performance. Signorile and colleagues (1993), reported that 30 s of active or passive recovery has a similar effect on the total work and peak power of a subsequent sprint. The findings of this study are not surprising, since the H^+ load and fall in power output associated with a single 6-s all-out sprint is small, and 30 s of passive recovery might be long enough for full recovery of power capacity. This

might explaini in part, why 30 s of active recovery after a single 6-s sprint offers no advantage.

In contrast to the above findings, a few studies have reported a slight improvement in the recovery of performance capacity when active recovery is performed after an all-out sprint (Weltman et al., 1977; Thiriet et al., 1993; Bogdanis et al., 1996). However, the reported effect of active recovery on performance in these studies is less than 3%. In particular, Weltman and co-workers (1977) reported that when an all-out 1 min effort on a cycle ergometer was followed by 20 min of active or passive recovery, the power output in the subsequent all-out exercise bout was 3.0% less with active than passive recovery. In agreement with these findings, Thiriet and colleagues, (1993), examined the effect of 20 min of active or passive recovery on subsequent performance after close to 2 min of exercise performed at 130% $\dot{V}O_{2max}$, and found that the fall in mean power was 8% less with active than passive recovery, and cycling duration also lasted longer in active recovery. One major limitation with these two studies, however, is that recovery protocols were compared at a time when recovery is likely to have been near complete. This limitation was not shared by the study of Bogdanis and colleagues, (1996), where active recovery at 40% $\dot{V}O_{2max}$ lasted only 4 min. They showed that despite this shorter recovery time, active recovery improved significantly but only marginally power output (3-4%) in the subsequent 30-s all-out sprint in comparison to passive recovery.

It is important to point out that all the studies described above were performed on a cycle ergometer. Recently, however, Felix (1997) compared the effect of 14 min of active and passive recovery swimming after a 200-m swim, and reported that swim time in a subsequent swim was only 2% faster in the active compared to the passive recovery trial. One major limitation with these findings, however, is that lactate levels

after the first sprint were low, thus suggesting that intramuscular pH might have been too high for active recovery to make a difference and 14 min of recovery might have been long enough for near complete recovery of performance capacity.

Overall, a close examination of the studies on active recovery performed after a single sprint reveals that the benefit of this mode of recovery is small, probably because of some important limitations in the design of these studies. In some studies, the bouts of high intensities exercise were too short and the levels of lactate attained were too small for active recovery to have a large impact (Signorile et al., 1993; Bogdanis et al., 1996). In other studies, the active recovery period was either too short (Signorile et al., 1993; Bogdanis et al., 1996) or so long that recovery was almost complete (Weltman et al., 1979; Bond et al., 1991; Thiriet et al., 1993). What is lacking, is a study that examines systematically the pattern of recovery of performance capacity after a sprint to use this information to compare active and passive recovery protocols at a time when recovery is only partial. Finally, in all studies where active recovery has been shown to improve recovery of exercise performance capacity, the possibility of a placebo effect and thus of an overestimation of the benefit of active recovery has not been taken into account (Weltman et al., 1977; Thiriet et al., 1993; Bogdanis et al., 1996; Felix et al., 1997). This is a limitation shared by all studies concerned with the effect of active recovery on sprint performance. This is an important limitation considering that the participants recruited in several studies have been athletes (Signorile et al., 1993; Thiriet et al., 1993; Felix et al., 1996; Bogdanis et al., 1996; Monedero et al., 2000; Connolly et al., 2003) who are highly likely to have been aware of the alleged benefits of active recovery. As discussed, later one of the primary purposes of this study will be to re-examine the proposed benefit of active recovery.

1.7 Preparatory strategies to optimise single sprint performance after repeated sprints

In the previous sections, the benefits of using mild exercise as a preparatory strategy to optimise sprint performance in individuals being either inactive for an extended period of time or recovering from a maximal sprint effort was discussed. Although there is strong evidence that the performance of PE in preparation for a sprint in previously inactive individuals generally increases sprint performance (Section 1.5), it is unclear whether this is also the case in individuals recovering from a single bout of intense exercise (Section 1.6). This raises the question of whether this is also the case following repeated maximal sprint efforts. Indeed, if repeated sprints were to result in a more pronounced fall in performance as opposed to a single sprint, the benefit of active recovery between consecutive sprints as opposed to only resting might be more apparent.

The question addressed here is an important one given that the performance of repeated sprints typifies the sequence of movement pattern of sports such as soccer, basketball, and hockey (Balsom et al., 1992). Indeed, these sports are characterised by the performance of several short bouts of intense activity interspersed with periods of lower intensity exercise or rest. The energy demands to support these short bouts of intense exercise rely primarily on phosphocreatine breakdown and glycolysis, whereas the energy for recovery appears to be derived aerobically (Colliander et al., 1988). Arguably, any practice that favours faster recovery rate between consecutive sprints is likely to be well received by athletes. In this regard, there is evidence that active as opposed to passive recovery between consecutive sprints reduces the magnitude of the fall in sprint power output that typically occurs in association with successive sprints.

Several studies have compared the effect of active and passive recovery performed between repeated bouts of intense exercise on sprint performance (Signorile et al., 1993; Thiriet et al., 1993; Ahmaidi et al., 1996; Dupont et al., 2003; Spierer et al., 2003; Monedero and Donne, 2000). In most of these studies, the indicators of sprint performance adopted to compare recovery protocols were peak power, mean power or total work, and different indices of time to fatigue. For these reasons, most studies have been performed on a cycle ergometer to obtain measures of power output and most studies have reported that performance falls progressively with repeated bouts of intense exercise, but to a lesser extent if each sprint is separated by active recovery. However, it is important to note that recently it has been reported that active recovery of short duration between consecutive running or swimming sprints impairs rather than improve performance (Toukebis et al., 2005) One difficulty in comparing studies, however, is the fact that the protocols of intermittent high-intensity exercise vary so much across studies that it might be inappropriate, on physiological grounds, to group them under the general banner of intermittent high intensity exercise. Indeed, for the same reasons that sprinting and moderate intensity exercises are considered as distinct activity patterns on physiological and biochemical grounds, similar considerations may apply with different types of intermittent high-intensity exercise. This is best illustrated by the varying intensities and durations of the exercise bout, the recovery time between consecutive bouts, and the type of activity performed during recovery across the different studies on intermittent, high-intensity exercise. For instance, the bout of intense exercise in some studies is performed at intensities corresponding to 110-130% $\dot{V}O_2$ (Thiriet et al., 1993; Dorado et al., 2004), whereas, in others, the intensity of the exercise bouts increases progressively (Ahmaidi et al., 1996) or consisted of a maximal sprint effort (Signorile et al., 1993; Spierer et al., 2003; Connolly et al., 2003). The duration of

each exercise bout varies also markedly across studies, ranging from 6 s (Ahmaidi et al., 1996; Signorile et al., 1993) to more than 120 s (Thiriet et al., 1993). Similarly, the length of the recovery periods between consecutive bouts ranges from 15 s (Dupont et al., 2003) to 20 min (Thiriet et al., 1993). For these reasons, the studies that have examined the effect of passive and active recovery on performance in intermittent high-intensity exercise will be described individually in this section.

In general, the studies concerned with the effect of active recovery on repeated all-out sprint performance show that this mode of recovery improves sprint performance. As alluded to earlier, Signorile and colleagues, (1993) adopted a protocol where trained athletes were required to either sit passively or cycle at 60 rpm with 1 kg resistance during a 30-s recovery from a series of eight 6-s all-out sprints. Under these conditions, the fatiguing effect of intermittent exercise bouts occurred cumulatively, irrespective of recovery protocol, but the decline in peak power, total work and fatigue rate was less pronounced (5-6%) in the active recovery group. These findings were corroborated recently by the work of Connolly and colleagues (2003), who compared the effect of sitting passively to cycling at 80 rpm with 1 kg resistance for 3 min during recovery from each of six consecutive 15-s all-out sprint in trained cyclists. As reported by Signorile and colleagues, (1993), the peak power of each sprint fell progressively, irrespective of recovery protocol. However, this fall in peak power was less when active rather than passive recovery was performed between consecutive sprints. These findings, however, are not supported by those of Spierer and colleagues (2004), who subjected trained athletes to repeated 30-s all-out sprint efforts, each separated by 4 min of either a rest or mild exercise performed at 28% $\dot{V}O_{2max}$. The patterns of response of peak and mean powers were not affected by the protocol of recovery. However, when sedentary subjects were subjected to the same experimental protocol, active

recovery was beneficial to mean power, but not peak power. The source of the discrepancies between this and other studies on active recovery might be tentatively explained on the basis that the participants involved in this study were ice hockey players, and the protocol of intermittent exercise adopted in that study reflects closely that experienced by these players during a game (Spierer et al., 2004).

Active recovery has a positive effect not only on repeated all-out sprint efforts, as described above, but also on repeated sub-maximal bouts of high-intensity exercise. The first study to show this was that of Thiriet and colleagues, (1993), who compared the effects of 20 min of passive recovery to active recovery at 30% $\dot{V}O_{2max}$, each performed between four consecutive exhaustive exercise bouts at 130% $\dot{V}O_{2max}$. Although performance fell between the first and last sprints across recovery protocols, this fall was less pronounced when active recovery was performed between consecutive bouts, and active recovery improved the duration of each exercise bout. These findings were corroborated by those of Dorado and colleagues, (2004), who compared the effect of 5 min of passive recovery to active recovery at 20% $\dot{V}O_{2max}$ performed between consecutive exhaustive exercise bouts performed at 110% $\dot{V}O_{2max}$. In agreement with the findings of others, the total work output fell markedly in response to successive sprints, and irrespective of recovery protocols, but this fall was of a lesser magnitude with active recovery.

Active recovery is also beneficial under conditions where the repeated bouts of sub-maximal intensity exercise are not performed to exhaustion but for a set duration. For instance, in a study investigating the effects of 5 minute of active recovery at 32% $\dot{V}O_{2max}$ on repeated 6-s bouts of intense exercise performed against increasing

braking force, mean power output with the last exercise bout was higher with active recovery as opposed to passive recovery (Ahmaidi et al., 1996).

Although this brief survey of the literature suggests that the benefits of active recovery are easier to demonstrate with intermittent high-intensity exercise than with a single maximal sprint effort, this raises the question of whether there are conditions where active recovery is detrimental to sprint performance. Recently, Dupont and colleagues, (2003), compared the effect of active and passive recovery on intermittent run time to exhaustion, with each exercise bout consisting of a 15-s run at 120% $\dot{V}O_{2max}$, and with active recovery lasting only 15 s. They showed that running time was longer for intermittent run if interspersed with passive rather than active recovery. These findings were explained on the basis that 15 s of active recovery might have been detrimental to the resynthesis of phosphocreatine stores and reloading of myoglobin with oxygen. Similarly, Toukebis and colleagues, (2005), reported that passive recovery is preferable to active recovery when recovery duration is short during repeated swimming sprints of short duration

There are other conditions where active recovery might impair prematurely sprint performance in comparison to passive recovery. As discussed previously, the benefit of active recovery with intermittent high-intensity exercise is generally explained on the basis that by favouring the oxidative disposal of lactate and H^+ after each exercise bout, the rise in H^+ is expected not to be as pronounced, thus delaying the onset of H^+-mediated fatigue. However, the problem with this pattern of recovery, in theory, is that lactate is a major carbon source for the synthesis of glycogen in skeletal muscle following high intensity exercise, particularly when food is not available (Hermansen and Vaage, 1977; Astrand et al., 1986; Peters-Futre et al., 1987; McDermot and Bonen, 1992), and given that depleted muscle glycogen

stores may also cause fatigue in sprint activity, it is possible that an earlier depletion of these stores would arise if active recovery were performed between consecutive bouts of exercise due to associated increased oxidative disposal of lactate. For this reason, it is possible that active recovery might be beneficial early during intermittent high intensity exercise, but detrimental later as the stores of muscle glycogen are depleted. This untested prediction is consistent with the observation that the replenishment of the stores of glycogen from endogenous carbon sources is reduced during active as opposed to passive recovery (Fairchild et al., 2003).

It is important to note that there are some limitations with the studies concerned with the effect of active recovery on intermittent high-intensity exercise performance. Although most studies conclude that active recovery is beneficial, the possibility of a placebo effect and thus of an overestimation of the benefit of active recovery has not been addressed in the design of these studies (Weltman et al., 1977; Thiriet et al., 1993; Bogdanis et al., 1996, Felix et al., 1997). As discussed in the previous section, this is an important issue considering that the participants recruited in many of those studies were athletes who are highly likely to have been aware of the alleged benefits of active recovery. Another experimental limitation that might have led to overestimating further the benefit of active recovery is the fact that in most study, the bout of intense sprinting after active recovery was not initiated from a dead stop position, but while the subjects were already pedalling, thus adding the confounding variable of determining the effect of active recovery *per se* on recovery of sprint performance capacity. It is important to note, however, that in the study of Ahmaidi and colleagues, (1996), their participants were required to cease cycling at the end of active recovery so as to initiate the bout of intense exercise from a stationary position, and they reported that active recovery improves performance of intermittent high-intensity exercise. Finally, most but not all studies on the effect of active

recovery on power output have been concerned with measuring peak power, mean power or different fatigue indices. Given the complex interplay of mechanisms involved in the temporal development of fatigue during an all-out sprint, and earlier findings that peak power, early power output and mean power recover at different rates following a maximal sprint effort (Bogdanis et al., 1994) and respond differently to active recovery from a single sprint (Bogdanis et al., 1996), this raises the possibility that early and late power output in response to repeated sprints might be affected differently by active recovery, an issue that remains to be examined.

1.8 Aims and research hypotheses

Despite all the findings described above on the benefit of PE as a preparative strategy for optimising sprint performance, a number of questions remain without answers. In particular, the response of sprint performance to PE duration as well as to the time elapsed between PE and sprinting has received little attention. Also, what still remains poorly understood is the relative importance of pre-sprint muscle temperature and cardiorespiratory mobilisation to sprint performance post-PE. Another important question relates to the most effective preparatory strategy to be adopted prior to a sprint when performed after an earlier sprint. Under these conditions, is it better for athletes recovering form a sprint to rest and face the risk of “cooling down” or to exercise lightly throughout recovery to optimise subsequent sprint performance when recovery is of short duration. Although, this mode of recovery has been shown, at the very best, to only have a marginal effect of sprint performance, this might be because the length of recovery was not optimal for full benefit of active recovery to become apparent. Finally, although there is strong evidence that intermittent high-intensity exercise interspersed with active recovery between consecutive bouts improves sprint performance, it is not clear whether this

would also be the case if the possibility of a placebo effect were to be minimised. Also, it remains to be established whether active recovery affects power output to a similar extent over the whole duration of each sprint or whether it affects differently early and late power output. It is the primary goal of this thesis to address these questions, many of which for the first time in the literature. Specifically, the aims of this thesis are:

1) to evaluate the relative contributions of muscle/body temperature and cardiorespiratory mobilisation in determining the effects of PE on subsequent sprint performance.
2) To determine the temporal pattern of recovery of sprint power capacity after a 30-s sprint in order to compare the effect of active and passive recovery on sprint performance at a time when recovery of power is either partial or complete.
3) To determine whether the benefit of active recovery on repeated sprint power output will occur even when the possibility of a placebo effect is minimised, and whether peak power and early and late mean power output of consecutive sprints will be affected to a similar extent by fatigue and active recovery.

The hypotheses underlying these aims are as follows:

1) Since an increase in muscle temperature affects primarily non-oxidative metabolism, and that the relative contribution of non-oxidative metabolism to total ATP production decreases with time during an all-out sprint effort, it is expected that the relative contributions of temperature and cardiorespiratory mobilisation in mediating the effect of PE on power output during a sprint will

change over time, with temperature effects playing a more important role at the start of sprinting.

2) The positive effect of active recovery in comparison to passive recovery will be large if compared at a time when recovery is partial and minimal when recovery is complete, and active recovery will be beneficial even if the possibility of a placebo effect is minimised.

3) Active recovery under conditions where the possibility of a placebo effect is minimised will also be beneficial to sprint performance, and peak power and early and late mean power output of each of repeated 30-s sprints will be affected to a different extent by active recovery.

CHAPTER 2

Preparatory strategies for optimising an all-out sprint effort I:
Effects of prior exercise on a subsequent all-out sprint

2.1 Introduction

Prior exercise (PE), or warm-up, is a common practice before most sporting activities, including those involving a short maximal sprint effort, and is considered essential for optimising performance by many coaches and athletes (Karvonen, 1992; Bishop, 2003a, 2003b). In particular, passive heating or moderate-intensity PE (< 60% $\dot{V}O_2max$) performed with or without a rest before a cycling sprint (10-60 s) improves peak power (PP) (Bergh and Ekblom, 1979; Dolan and Sargeant, 1983; Dolan et al., 1983; McKenna and Green, 1987; Sargeant and Dolan, 1987; Obrien et al., 1997), but not if intense PE (>60% $\dot{V}O_2$ max) is performed immediately before sprinting (Margaria et al., 1971; Dolan and Sargeant, 1983; Sargeant and Dolan, 1987). Also, most studies have reported that moderate-intensity PE improves mean power if little or no rest is allowed between PE and the subsequent sprint (Inbar and bar-Or, 1975; McKenna and Green, 1987; Sargeant and Dolan, 1987; Obrian et al., 1997).

Several studies have associated the potential benefit of PE on power output during an all-out sprint with either temperature-related effects on the working muscle or with a mobilisation of the cardiorespiratory system that increases the aerobic contribution to exercise (Karvonen, 1992; Jones et al., 2003; Bishop, 2003a). For instance, the passive warming up of muscles by immersion in hot water bath has been reported to improve peak power (Dolan et al., 1983; Sargeant, 1983, 1987), whereas the cooling of muscle results in a fall in power output (Sargeant, 1983, 1987). Besides acting via increasing muscle temperature, there is evidence that PE can also improve sprint performance by mobilising the cardiorespiratory system prior to exercise if a rest period between PE and the start of the sprint is short enough for $\dot{V}O_2$ to remain above resting level (DeBruyn-Prevost and Lefevbre, 1980), otherwise PE has no effect (Hipple, 1955). As a result, the greater aerobic

contribution (Ingjer and Stromme, 1979; Stewart and Sleivert, 1998; McCutcheon et al., 1999) and lower oxygen deficit at the onset of exercise (Gutin et al., 1976; Andzel, 1982; McCutcheon et al., 1999) would be expected to increase the time to exhaustion and improve sprint performance.

To date, no study has attempted to evaluate the relative importance of an increase in muscle or core temperature and pre-exercise cardiorespiratory mobilisation in explaining the effect of PE on power output during a sprint. Furthermore, there has been no attempt at separating out the effect of PE on the temporal response of power output during subsequent sprinting. This latter issue is an important one, since the relative contributions of the fuel systems that support the energy demands of skeletal muscle during a sprint change rapidly over time, with phosphocreatine contribution being at its highest at the onset of exercise and that of oxidative ATP supply increasing progressively over time (Bogdanis et al., 1998). This raises the possibility that the mechanisms whereby PE affects power output during a sprint might also differ as a sprint progresses. For these reasons, our goal was to compare the effect of PE on the temporal response of power output during a sprint by controlling either temperature or pre-exercise $\dot{V}O_{2peak}$ in order to test the hypothesis that there might be a temporal shift in the mechanisms whereby PE affects sprint performance.

2.2 Materials and Methods

2.2.1 Participants

Twelve male cyclists and triathletes for whom sprinting was part of their training programmes were recruited for this investigation. Their age, height, body mass and $\dot{V}O_2$ peak (Mean ± SD) were 28 ± 6.1 y, 179.5 ± 5.8 cm, and 77.0 ± 10.1 kg and 57.60 ± 9.60 $mL.kg^{-1}.min^{-1}$, respectively. All participants were informed about the purpose of the study and risks associated with the methods and procedures involved for data collection. Each participant was then asked to sign an informed consent form and was informed that he could withdraw from the study at any time. This study was approved by the Human Rights Committee of the University of Western Australia.

2.2.2 Familiarisation session

The participants completed a familiarisation session for the determination of their $\dot{V}O_{2\ peak}$, and then performed a 30-s sprint in order to become acquainted with this exercise protocol, the equipment, and the researchers involved in the study. During this session, anthropometric data (height, body mass) were also obtained.

2.2.3 Experimental design

Each participant attended the laboratory on three occasions at the same time of day to minimise diurnal variations in the sprint response to PE (Atkinson et al., 1989). Tests were performed at least 4 h after the last meal, with at least 1 week between consecutive visits. Participants were also asked to observe the same pre-testing diet and to refrain from any intense physical activity for 24 h before testing, and for these reasons were asked to complete a training and dietary diaries. The participants in this study were told that we were primarily concerned with the effect of PE on blood metabolites profiles, and no mention was made about our focus on sprint

performance. Each sprint session consisted of a 30-s sprint on a front-access cycle ergometer (Model Ex-10, Repco Australia) following one of three randomly assigned PE treatments performed at an intensity of 40% $\dot{V}O_{2\ peak}$ on the same ergometer as that used for the sprint trials and administered following a counterbalanced design. These PE treatments were administered so as to control for either pre-sprint temperature or $\dot{V}O_2$. Given that, in response to moderate-intensity exercise, $\dot{V}O_2$ reaches stable levels within 3 min (Jones et al., 2003), but that a longer time period is required for muscle and core temperature to stabilise (Saltin et al., 1968), the effect of a PE-mediated increase in temperature independently of $\dot{V}O_2$ was examined by comparing the maximal sprint response to two PE protocols of different duration that resulted in similar $\dot{V}O_2$ but different temperatures (4 and 20 minutes PE with no subsequent rest before sprinting). In contrast, the temperature-independent effect of $\dot{V}O_2$ on sprint performance was examined by comparing the effect of a short PE that increases $\dot{V}O_2$ to stable levels, but not too long to cause significant changes in temperature, with a PE of similar duration but followed by a rest long enough for $\dot{V}O_2$, but not temperature, to return to basal level (4 min of PE followed by either a 10-min rest or no rest before sprinting). The three PE conditions thus examined were i) 4 min of PE (PE_4) with no subsequent rest prior to sprint, ii) 20 min of PE (PE_{20}) performed just before sprinting, and iii) 4 min of PE followed by 10 min of rest before sprinting (PE_{4R}), with all PE performed at an intensity of 40% of $\dot{V}O_{2\ peak}$.

2.2.4 Sprint trial

In order to perform the 30-s sprint test, each participant was asked to mount a front-access cycle ergometer (Repco, NSW), with each foot secured using a foot strap. Immediately before sprinting, each participant was asked to stand-up on the ergometer while placing both hands on the handles and remaining still. The trial was

initiated with the foot of the dominant leg starting at the “two o’clock” position after a three-second count down. All participants were instructed to cycle in an “all-out” fashion without pacing themselves for 30 s. During each 30-s sprint, the following information was recorded; peak power output (PP), mean power output over 30 s (MP_{30}), mean power output over the first 6 s (MP_6) and 10 s (MP_{10}), and mean power output over the last 20 (MP_{-20}) and 10 s (MP_{-10}).

2.2.5 Rectal temperature, muscle and skin temperature (T_{re})

A rectal probe (RECT-1, Type T, Copper-Constantan Thermocouple, Physitemp instruments inc., New Jersey, USA) was prepared about 90 min before testing. A piece of cotton elastic bandage was tied on the cable 10 cm from the tip of the probe. The bandage was then wrapped around the waist of the participant with the attached cable at the back. Slowly, the temperature sensitive tip of the probe was then inserted in the rectum by the participant to a depth of 10 cm marked by the knot of the elastic bandage. The other end of the cable was attached to the computer adaptor terminal.

Muscle temperature was measured with a temperature sensitive muscle probe (MUSCLE-1, Type T, Copper-Constantan Thermocouple, Physitemp Instruments Inc, New Jersey, USA) that was inserted at a depth of 4 cm in the *vastus lateralis*. To this end, the skin was first cleaned using an alcohol swab and was shaved around the area designated for the probe insertion. A local anaesthetic cream (Emla, AstraZeneca, Sweden) was then applied on the insertion site an hour before insertion. The probe, a thin copper wire, was then placed inside a 16G x 11/2 inch intravenous catheter (Surflo, Radiopaque, Netherland). Once the needle was inserted at a depth of about 4 cm, the catheter was slowly pulled out leaving the probe in the muscle, and the catheter was safely taped to a piece of cotton gauze underneath and securely strapped on to the thigh muscle. The probe was then

connected to the computer adaptor terminal for the recording of temperature. The probe remained in the muscle throughout the testing session. A skin temperature sensor (Physitemp Insruments Inc, New Jersey, USA) was also placed about 2 cm from the muscle probe site and connected to the computer adaptor terminal. All temperature readings were automatically saved using *Thermes* computer software (Physitemp Instruments Inc, New Jersey, USA) and retrieved for later analysis.

2.2.6 Determination of $\dot{V}O_2$ and $\dot{V}O_{2peak}$

In order to determine the baseline $\dot{V}O_2$ associated with the different PE protocols, each participant was asked to breathe through a Hans Rudolph valve connected to a gas analysing system with Colin hoses. Ventilation was recorded continuously using a turbine ventilometer (Morgan, Model 096, Kent, England) and expiratory gas composition was determined using oxygen and carbon dioxide analysers (Ametek gas analysers SOV S-3A and COV CD3A, respectively, Pittsburg, Pa., USA). These gas analysers were calibrated immediately before and verified after each test using three certified gravimetric beta-gas mixtures (BOC gases, Chatswood, Australia). The ventilometer was calibrated pre-exercise and verified post-exercise using a one litre syringe in accordance with the manufacturer's instructions.

$\dot{V}O_{2peak}$ was determined on the same cycle ergometer as that used for the experimental testing. The $\dot{V}O_{2peak}$ test consisted of graded exercise steps using an intermittent protocol (1-min break between stages). The test commenced at 50 W and, thereafter, the intensity was increased by 30 W every 4 min until volitional exhaustion. Participants were required to maintain the set power output, which was displayed on a computer screen in front of them. The test was stopped when the participants could no longer maintain the required power output or when respiratory exchange ratio was above 1.15. Strong verbal encouragement was provided to each

participant, particularly towards the end of the test. All inspired and expired air was collected through a mouthpiece connected to a Hans-Rudolf valve attached via Collins tubing to an on-line gas analysis system comprised of a Morgan Ventilation Monitor. All expired air was continuously monitored for the analysis of O_2 and CO_2 concentrations (Ametek gas analysers SOV S-3A and COV CD3A respectively, Pittsburg, Pa., USA). Data were averaged over 30-s intervals. Ventilation was also recorded every 30 s using a turbine ventilometer (Morgan, Model 096, Kent, England).

2.2.7 Blood Sampling

In order to provide indirect evidence that the PE protocols adopted here has no effect on pH and lactate levels blood was sampled before and after each PE treatment from the earlobe using a 125 µL capillary tube and analysed for pH and lactate levels with a blood-gas analyser (ABL 625 series, Radiometer, Copenhagen). The blood gas analyser was routinely calibrated using precision standard solutions.

2.2.8 Statistical analyses

All results were analysed using a one way ANOVA with repeated measures followed by Fisher LSD posteriori test. All data are expressed as mean standard error of means (± S.E.M), except for the descriptive characteristics of the participants which were expressed as means ± standard deviation (S.D.) The level of statistical significance was set at $p<0.05$. All statistical analyses were conducted using the SPSS 12.0 for Windows Statistical Package (version 12.0, SPSS, Chicago, IL).

2.3 Results

2.3.1 Effect of PE on muscle and rectal temperature, $\dot{V}O_2$*, plasma lactate concentration and pH*

Both muscle (Fig. 2.1) and rectal (Fig. 2.2) temperature were not significantly different between trials before all PE treatments. Both 4-min PE conditions did not result in any significant change in rectal or muscle temperature (Fig. 2.1, 2.2). However, just before sprinting, muscle and rectal temperatures were significantly higher following PE_{20} compared to PE_4 and PE_{4R} ($p<0.05$; Fig. 2.1, 2.2). There was a significant increase in $\dot{V}O_2$ in response to all three PE conditions ($p<0.05$; Fig. 2.3), but, prior to sprinting, $\dot{V}O_2$ fell to resting levels ($p<0.05$) in PE_{4R}, whereas $\dot{V}O_2$ for PE_4 and PE_{20} were not different (Fig. 2.3). Plasma lactate concentrations and pH before and after all PE treatments were not significantly different (Fig. 2.4, 2.5).

2.3.2 Effect of PE conditions on power output during a 30-s sprint

Peak power after PE_{20} was higher than after both PE_4 and PE_{4R} ($p<0.05$), with no significant difference between these latter two PE protocols (Fig. 2.6). Mean power output during the 30-s sprint was significantly greater after PE_{20} and PE_4 compared to PE_{4R} ($p<0.05$), with no significant differences between PE_{20} and PE_4 (Fig. 2.7). MP_6 and MP_{10} were highest after PE_{20} and lowest after PE_{4R} ($p<0.05$; Fig. 2.8, 2.9). Finally, MP_{-20} and MP_{-10} were higher after PE_4 than PE_{20} and lowest after PE_{4R} ($p<0.05$; Fig. 2.10, 2.11).

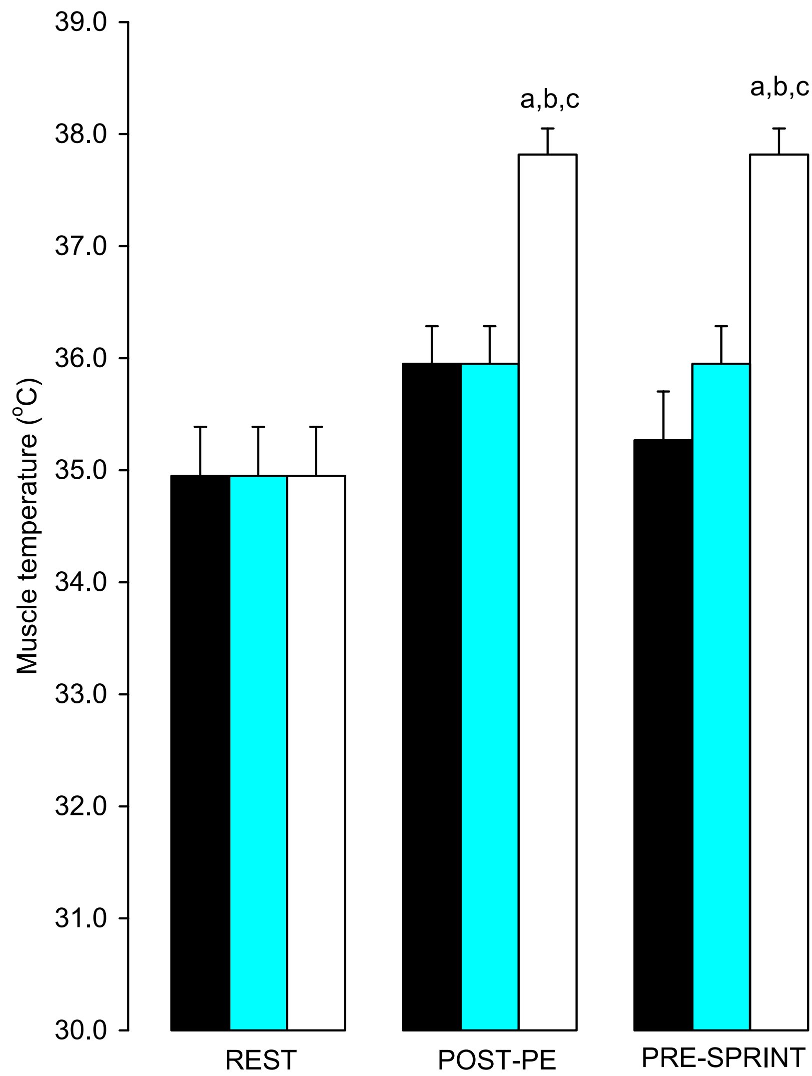

Figure 2.1: Effect of different prior exercise (PE) treatments (■, PE_{4R}; ■, PE_4; □, PE_{20}) on muscle temperature. a, significantly different from REST; b, significantly different from PE_{4R} and c, significantly different from PE_4 ($p<0.05$).

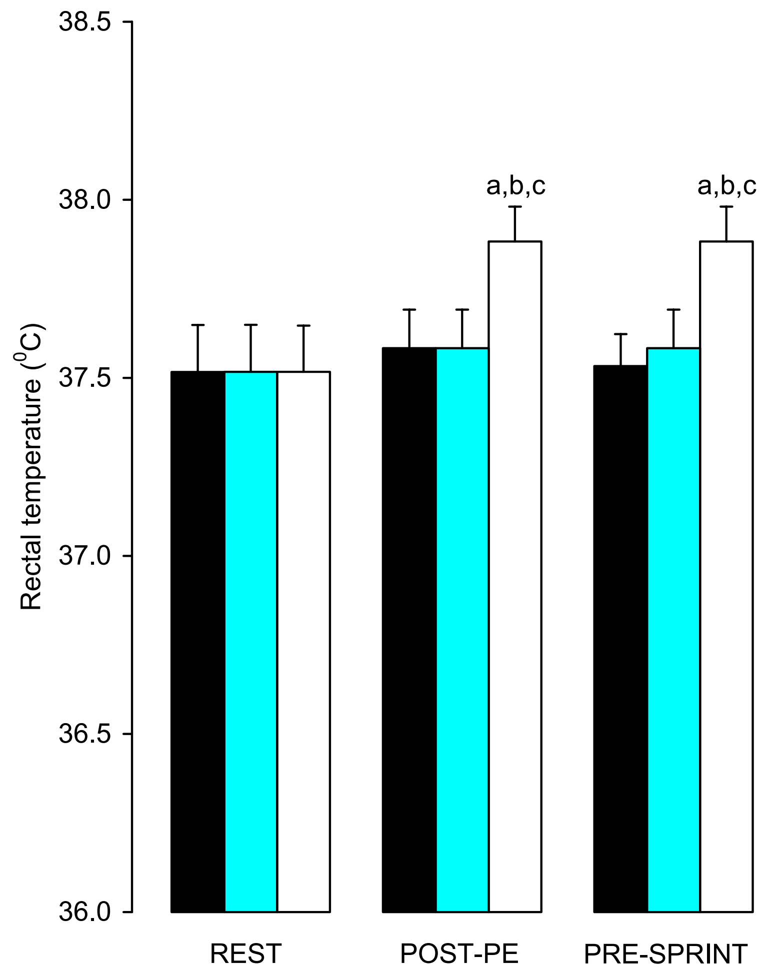

Figure 2.2: Effect of different prior exercise (PE) treatments (■ , PE_{4R}; ■ , PE_4; □, PE_{20}) on rectal temperature. a, significantly different from REST; b, significantly different from PE_{4R} and c, significantly different from PE_4 ($p<0.05$).

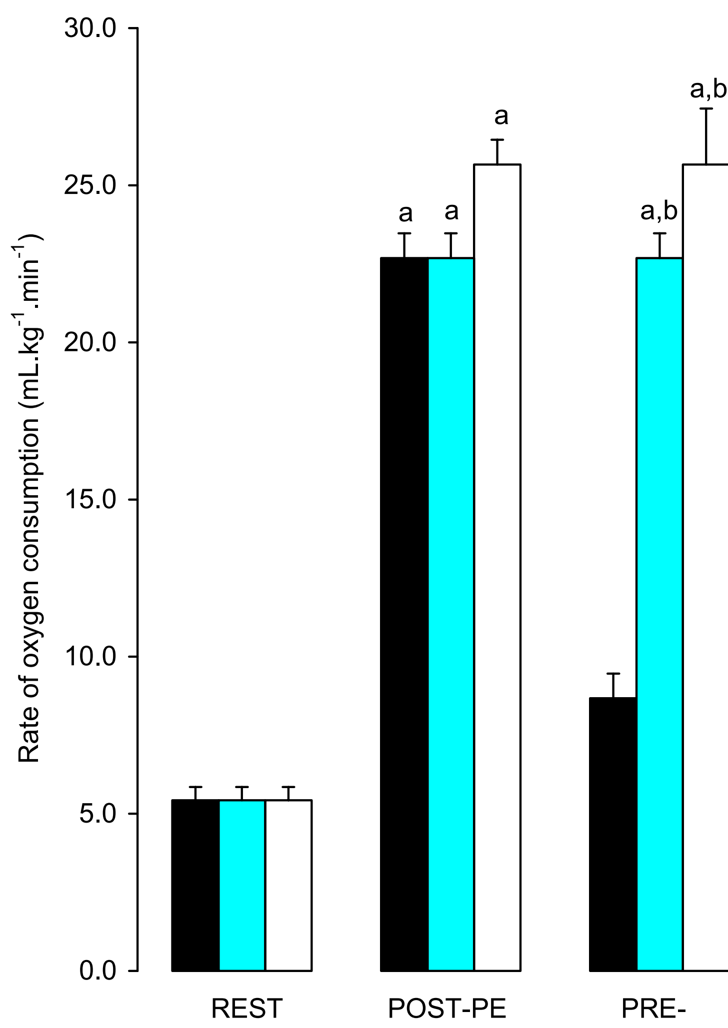

Figure 2.3: Effect of different prior exercise (PE) treatments (, PE_{4R}; , PE_4; , PE_{20}) on the rate of oxygen consumption. a, significantly different from REST; b, significantly different from PE_{4R} ($p<0.05$).

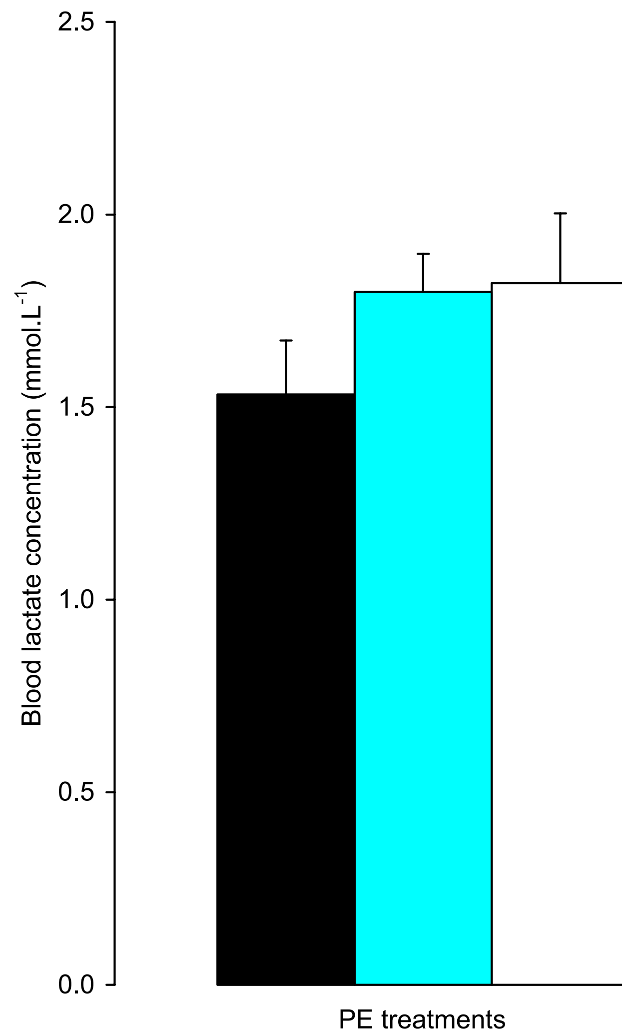

Figure 2.4: Effect of different prior exercise (PE) treatments (■, PE_{4R}; ■, PE_4; □, PE_{20}) on lactate levels prior to a 30-s sprint. No significant differences between treatments (p>0.05).

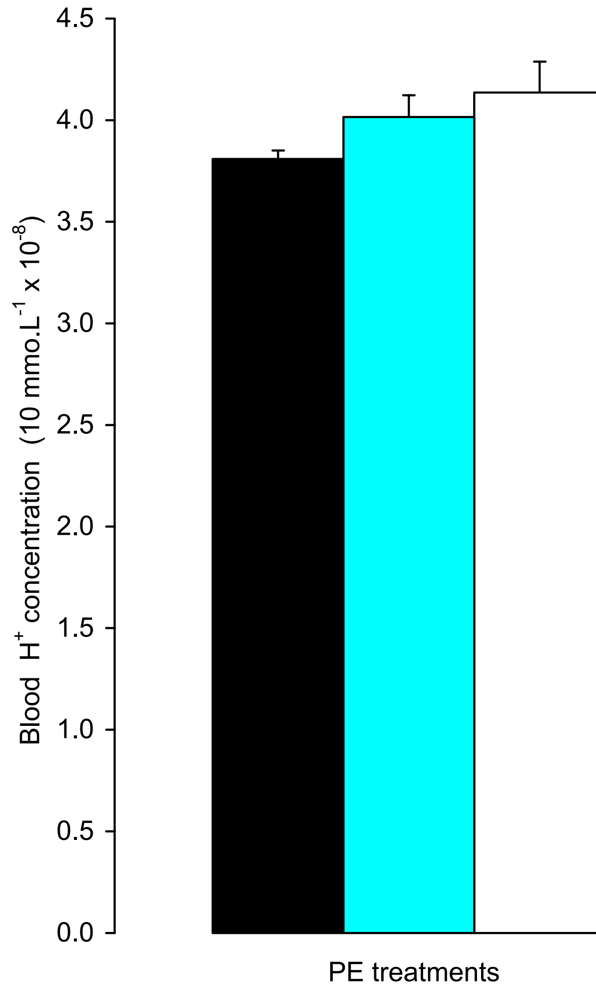

Figure 2.5: Effect of different prior exercise (PE) treatments (■, PE_{4R}; ■, PE_4; □, PE_{20}) on the concentration of blood H^+ prior to a 30-s sprint. No significant differences between treatments ($p>0.05$).

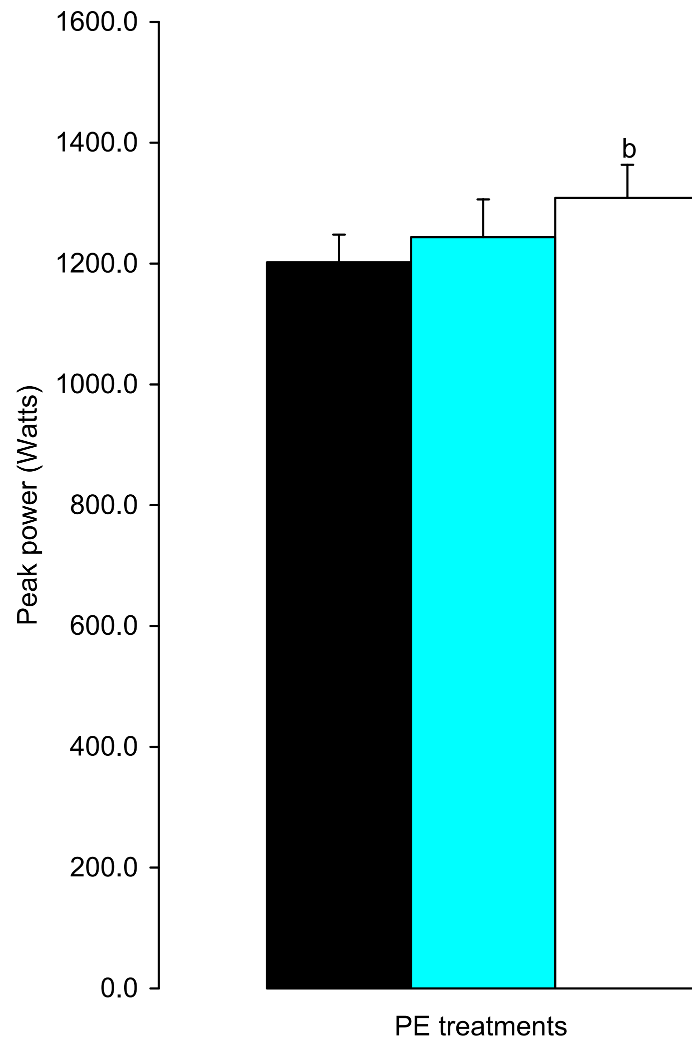

Figure 2.6: Effect of different prior exercise (PE) treatments (■ , PE_{4R}; □ , PE_4; □, PE_{20}) on the peak power (PP) during a 30-s sprint. b, significantly different from PE_{4R} ($p<0.05$).

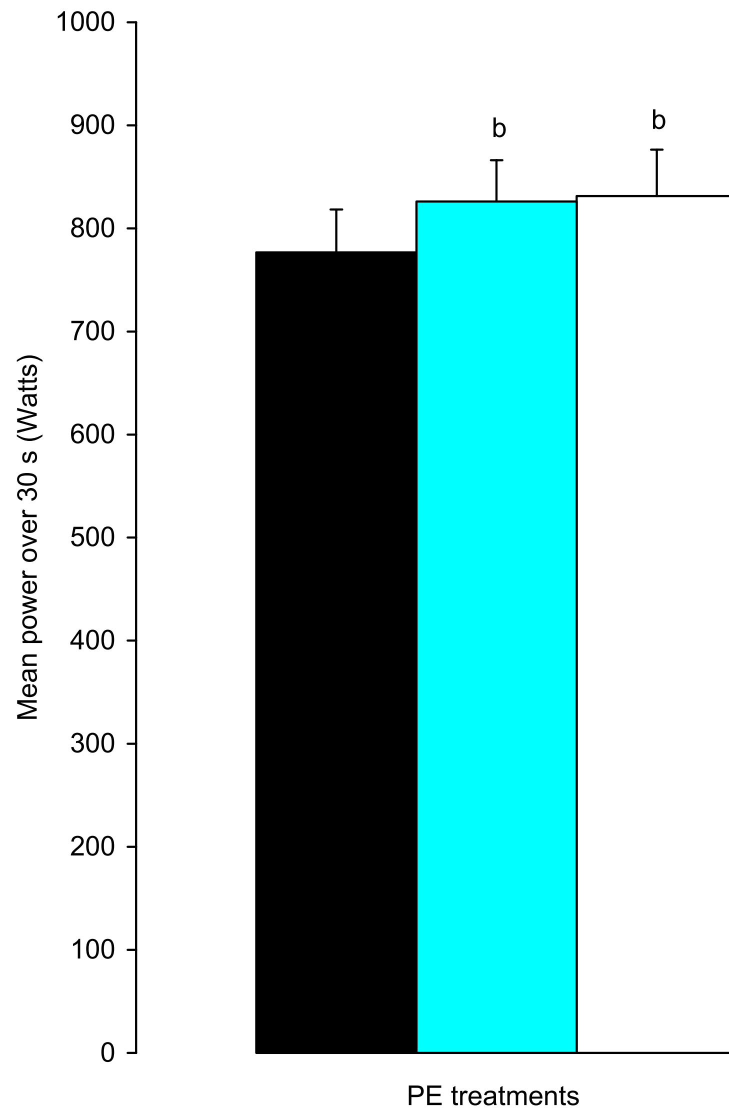

Figure 2.7: Effect of different prior exercise (PE) treatments (■ , PE_{4R}; ■ , PE_4; □, PE_{20}) on mean power over 30s (MP_{30}). b, significantly different from PE_{4R} ($p<0.05$).

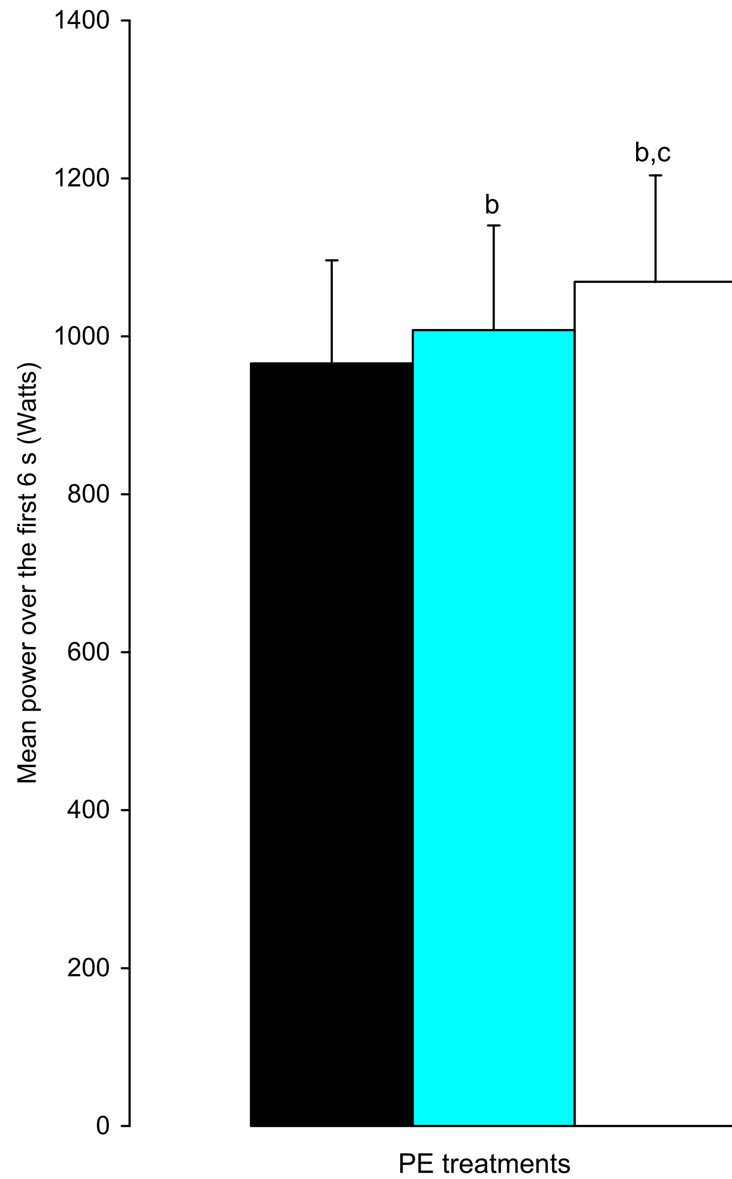

Figure 2.8: Effect of different prior exercise (PE) treatments (, PE_{4R}; , PE_4; , PE_{20}) on mean power over the first 6s (MP_6). b, significantly different from PE_{4R}, c, significantly different from PE_4 ($p<0.05$).

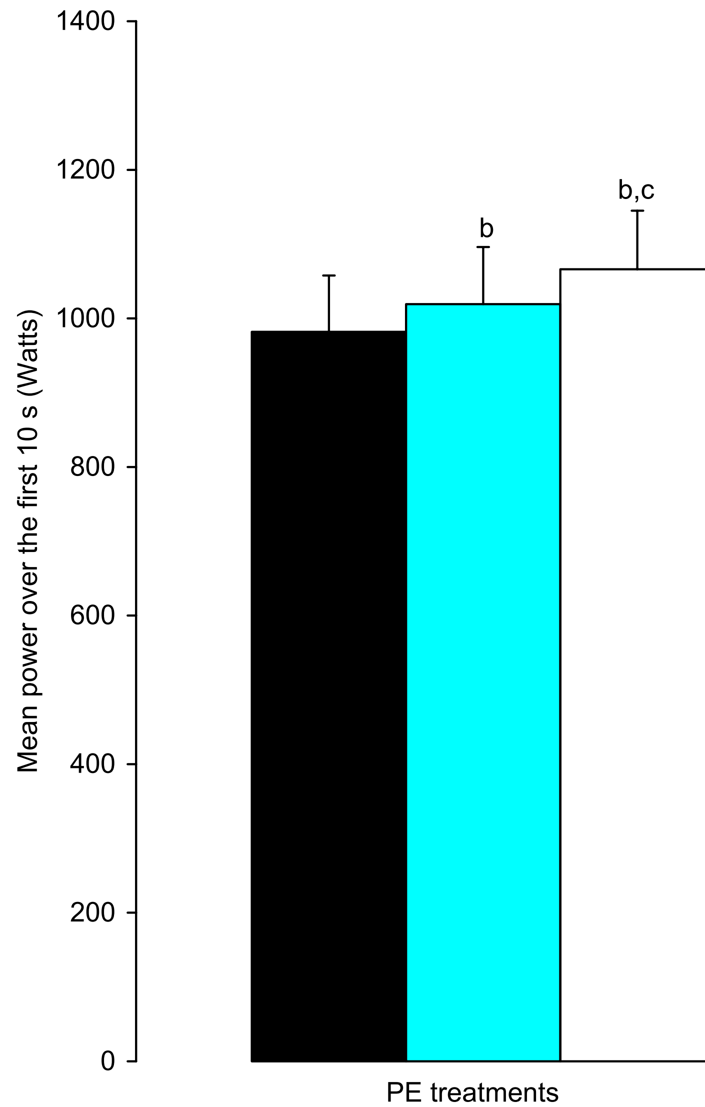

Figure 2.9: Effect of different prior exercise (PE) treatments (■ , PE_{4R}; □ , PE_4; □, PE_{20}) on mean power over the first 10s (MP_{10}). b, significantly different from PE_{4R}, c, significantly different from PE_4 ($p<0.05$).

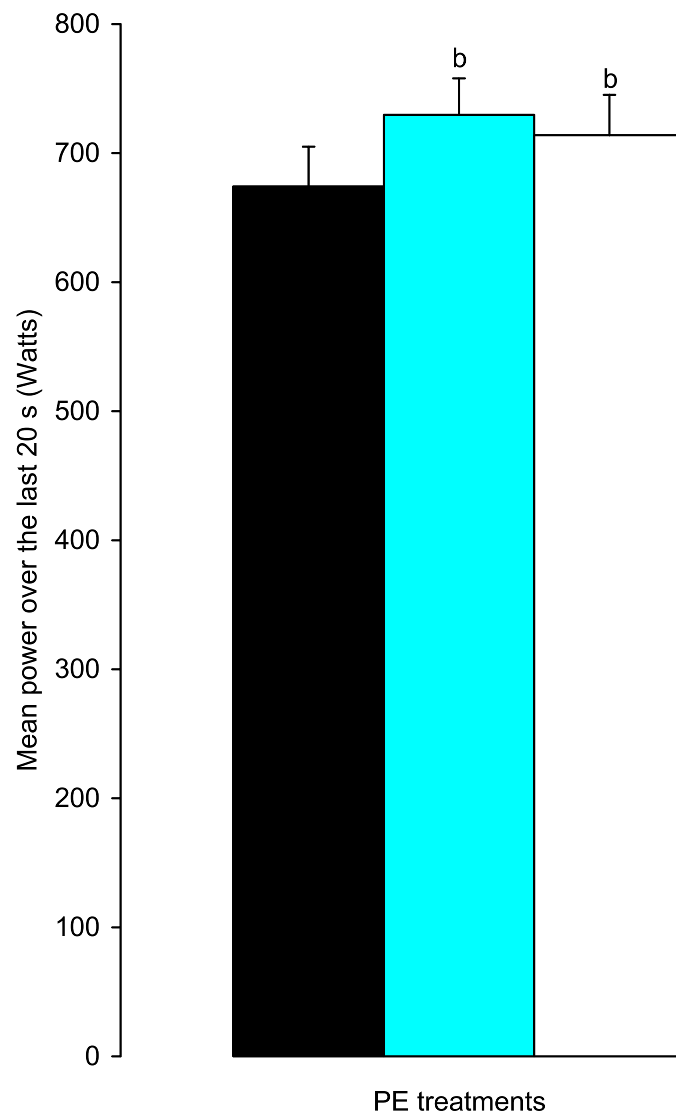

Figure 2.10: Effect of different prior exercise (PE) treatments (■ , PE_{4R}; ■ , PE_4; □, PE_{20}) on mean power over the last 20s (MP_{-20}). b, significantly different from PE_{4R} ($p<0.05$).

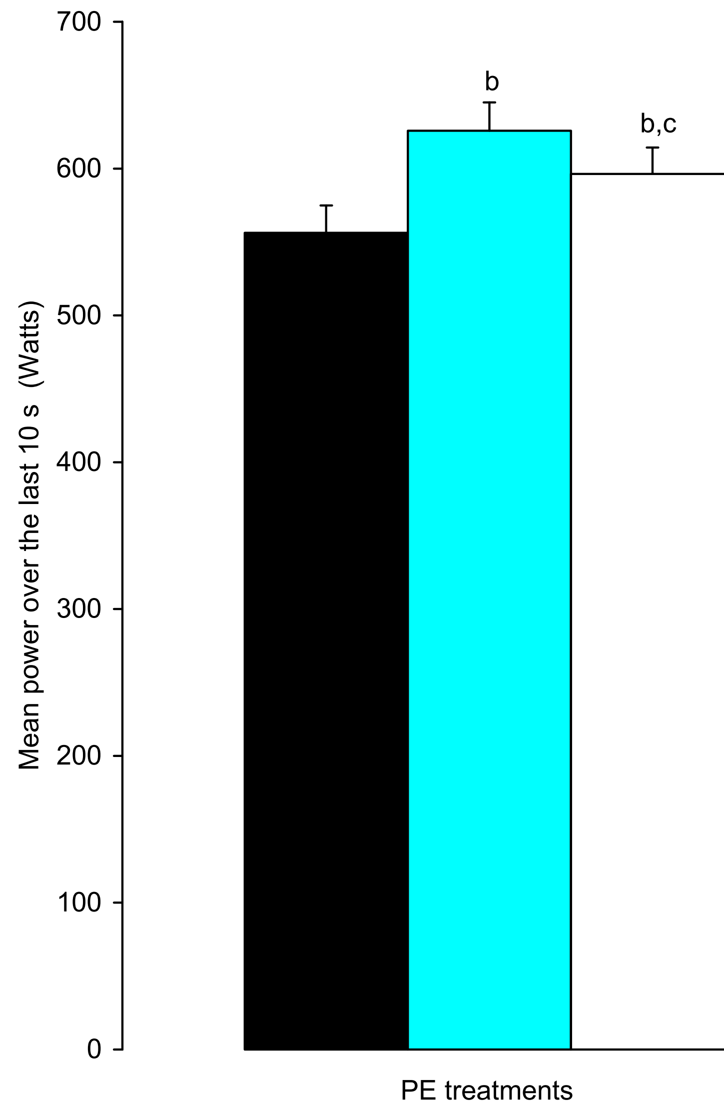

Figure 2.11: Effect of different prior exercise (PE) treatments (■, PE_{4R}; ■, PE_4; □, PE_{20}) on mean power over the last 10s (MP_{-10}). b, significantly different from PE_{4R}, c, significantly different from PE_4 ($p<0.05$).

2.4 Discussion

The extent to which the benefits of PE on sprint performance are attributable to mechanisms related to increased muscle and body temperature or mobilisation of the cardiorespiratory system have not been previously examined. In order to examine the effect of PE-mediated increase in muscle temperature alone on sprint performance, two PE conditions, PE_{20} and PE_4, were chosen to generate similar pre-sprint $\dot{V}O_2$, but different muscle temperatures. Under these conditions, muscle and core temperatures associated with PE_{20} were higher than with PE_4, whereas both PE treatments resulted in similar pre-sprint $\dot{V}O_2$. Such a matching of $\dot{V}O_2$ between PE_4 and PE_{20} is not surprising, since only a few minutes are required for $\dot{V}O_2$ to reach a steady level in response to moderate-intensity exercise (Jones et al., 2003). The higher muscle temperature in the face of comparable pre-sprint $\dot{V}O_2$ after PE_{20} in comparison to PE_4 resulted in higher PP, MP_6 and $MP_{10,}$ but lower MP_{-20} and MP_{-10}. This together with the finding that PE_4 and PE_{4R} were associated with no difference in PP despite marked differences in $\dot{V}O_2$ but similar muscle and core temperature prior to sprinting suggests that temperature-related mechanisms play an important role in mediating the effect of PE on power output at the onset of an all-out sprint. In contrast, the findings that MP_6, MP_{10}, MP_{-20} and MP_{-10} after PE_4 were significantly higher than after PE_{4R} despite similar muscle temperature but large differences in pre-sprint $\dot{V}O_2$ together with the lesser MP_{-20} and MP_{-10} when $\dot{V}O_2$ but not muscle and rectal temperature is matched (PE_4 and PE_{20}) suggest that cardiorespiratory-dependent rather than temperature-dependent mechanisms explain the benefit of PE on late power output (>10 s) during an all-out sprint. Overall, these results suggest for the first time that there is a temporal shift in the mechanisms responsible for the increase in power output in response to PE performed before a maximal 30-s sprint effort, with muscle temperature playing an

important role in enhancing power at the onset of sprinting, and increased mobilisation of the cardiorespiratory system playing the most important role later.

Our findings show that PE conditions resulting in similar increases in pre-sprint $\dot{V}O_2$, but different muscle and rectal temperatures, are associated with a rise in peak power and early power output during a sprint. Others have also reported that PE at a moderate intensity performed with little or no rest before a sprint improves PP (Bergh and Ekblom, 1979; Dolan and Sargeant, 1983; McKenna and Green, 1987; Sargeant and Dolan, 1987; Obrian et al., 1997), but not if PE is intense (Margaria et al., 1971; Dolan and Sargeant, 1983; Sargeant and Dolan, 1987). The limitation with these studies is that they did not exclude the possibility that elevated $\dot{V}O_2$ pre-exercise might be a major factor contributing to the improvement in PP because pre-sprint $\dot{V}O_2$ was not controlled for in these studies. However, in studies where 3-8 min PE of moderate intensity is followed by a rest long enough (2-6 min) for $\dot{V}O_2$ to return to basal or near basal level, PP improves in most (McKenna and Green. 1987; Sargeant and Dolan, 1987) but not all (Hawley, 1989), thus supporting our finding that an increase in temperature contributes to the beneficial effect of PE on early power output during a sprint. This interpretation is further corroborated by the findings that passive warming of muscle by immersion in hot water increases PP (Dolan et al., 1983; Sargeant, 1983, 1987), whereas a decrease in muscle temperature results in a fall in PP (Sargeant, 1987).

The aforementioned evidence that a rise in muscle and core temperature improves power output at the onset of an all-out sprint raises the question of the mechanisms underlying these effects of muscle temperature. The most obvious ones relate to the Q_{10} effect of temperature on the rates of phosphocreatine breakdown, glycolysis, cross bridge cycling and mitochondrial oxidative phosphorylation (Brooks et al.,

1971; Bennett, 1984; Febbraio et al., 1996), the latter of which, however, being less efficient as muscle temperature rises (Brooks et al., 1971; Willis and Jackman, 1994). Indirect support for such a Q_{10} effect of muscle temperature is the association between passive pre-heating of muscle and higher rate of phosphocreatine breakdown and glycolysis at the onset of a sprint (Edwards et al., 1972; Febbraio et al., 1996). A rise in muscle temperature could also improve sprint performance by decreasing muscle viscous resistance (Butchel et al., 1944), improving nerve conduction (Young et al., 1998) and by enhancing the contribution of oxygen-dependent ATP supply via increasing oxygen delivery by a temperature-mediated increased dissociation of oxyhaemoglobin and oxymyoglobin and via peripheral vasodilation (Theorell, 1934; Barcroft and Edholm, 1943). These latter mechanisms, however, are unlikely to play an important role here, since muscle temperature has been shown not to have any effect on the $\dot{V}O_2$ kinetics associated with intense exercise (Koga et al., 1997; Burnley et al., 2000; Koppo et al., 2002). Overall, it is still not clear which of the above mentioned mechanisms best explains the beneficial effect of muscle temperature on early power output during an all-out maximal sprint effort.

Although PE treatments resulting in a similar pre-sprint $\dot{V}O_2$ but greater muscle and rectal temperatures were associated with higher early power output at the onset of an all-out sprint, they were not associated with differences in MP_{30}. This is in contrast to the findings of others who have reported that PE followed by a rest long enough for $\dot{V}O_2$ to return to rest level but not too long for muscle temperature to fall markedly improves total work output during sprinting (Inbar and Bar-Or, 1975; Obrian et al., 1997; Bishop et al., 2001). These discrepancies can be reconciled by examining the power output profile of our participants over time. Although an increased muscle temperature after PE_{20} was associated with a higher power output

at the onset of the all-out 30-s sprint, this was not the case over the last 20 s (MP_{-10} and MP_{-20}), at which time PE_{20} was detrimental to performance compared to PE_4. It follows from this observation that had a shorter sprinting time been adopted here, MP over the whole sprint would have been better in response to PE_{20}, but worst had the sprint lasted for longer. Such a time-dependent pattern of response of power output to PE has never been reported before and highlights the importance of breaking down the power output of a sprint into its early and late components for more meaningful analyses to be performed.

Our finding that PE-mediated increase in muscle temperature improve early power output while being detrimental to late power output during a maximal sprint effort is in agreement with earlier reports that passive warming of muscle by immersion in hot water increases peak power (Dolan et al., 1983; Sargeant, 1983, 1987), but is accompanied by a faster rate of fatigue (Dolan et al., 1983, 1987). Since the rate of glycolysis and phosphocreatine breakdown are higher at the onset of a sprint in pre-heated muscle (Febbraio et al., 1996), the benefit of increasing muscle temperature would thus be expected to be countered by a faster rate of fatigue as the sprint progresses (Sargeant et al., 1983; Sargeant, 1987) due to the premature depletion of phosphocreatine stores, earlier accumulation of inorganic phosphate, and acidosis (Febbraio et al., 1996). In support of this, Gaitanos 1993 have previously reported a strong negative correlation between H^+ accumulation and sprint decrement. Altogether, these unfavourable metabolic changes might counteract the potential benefit of increased muscle temperature during late sprint. However, this interpretation remains to be reconciled with the growing evidence that pH plays little role in the mediation of peripheral fatigue (see section 1.4.2)

Our findings suggest that one approach to improve power output late during an all-out sprint is to increase the mobilisation of the cardiorespiratory system prior to

sprinting. Indeed, our results show that when low-intensity PE lasting 4 min is followed by a rest period long enough for $\dot{V}O_2$ to return to baseline levels (~ 5 min), MP, MP_6, MP_{10}, MP_{-10} and MP_{-20} are lower than when such a PE is performed immediately before a sprint. The absence of significant differences in muscle and rectal temperature between PE_4 and PE_{4R} treatments, but differences in pre-sprint $\dot{V}O_2$ suggest that differences in the mobilisation of the cardiorespiratory system in muscle are likely to explain the benefit of PE under these conditions, a conclusion further supported by our findings that increasing muscle temperature does not enhance MP_{-10} and MP_{-20}. Although, as discussed above, others have reported that PE followed by little or no rest before a sprint improves mean power output (Inbar and Bar-Or, 1975; Bergh and Ekblom, 1979; McKenna and Green, 1987; Obrian et al., 1997) or time trial performance (Andzel, 1982; McKenna and Green, 1987; Asmussen and Boje, 1995), pre-exercise muscle temperature was not controlled in these studies and the time-dependent impact of PE on power output was not examined. A better indirect support for the benefit of pre-exercise mobilisation of the cardiorespiratory system is suggested by the fall in sprint performance reported in some studies when a 5-min rest is allowed between PE and sprinting (DeBruyn-Prevost and Lefevbre, 1980; Hawley et al., 1989). It must be stressed, however, that others have reported that moderate PE (Inbar and bar-Or, 1975) followed by a 5 min rest improves mean power output during a 60-s cycling sprint (Obrien et al., 1997), thus suggesting that mechanisms other than mobilisation of the cardiorespiratory system are involved.

Our evidence that the mobilisation of the cardiorespiratory system improves power output late but not early during an all-out sprint raises the question of the mechanisms whereby such a mobilisation differentially affects power output throughout a sprint. The absence of any effect of elevated pre-sprint $\dot{V}O_2$ on power

output during the early stage of an all-out sprint can be explained on the grounds that the rate of anaerobic and aerobic ATP production are most probably high enough at this stage to support muscle energy demands, irrespective of whether oxygen availability increases or not (Bogdanis et al., 1998). In support of this view, hyperoxia has been reported not to affect peak power during a sprint (Burnley, et al., 2000) and oxygen delivery not to be limiting (Bangsbo et al. 2000). However, since the contribution of aerobic ATP production increases progressively during a sprint and that the onset of fatigue occurs more rapidly under conditions of increased reliance on anaerobic ATP production (Bogdanis et al., 1998), this suggests that PE favouring increased oxidative ATP production throughout a sprint should delay the onset of fatigue and improve performance in the later stage of an all out sprint effort. For this reason, increased cardiorespiratory mobilisation to meet muscle oxygen demands would be expected to allow the participants to reach more rapidly high levels of aerobic metabolism, thus reducing the initial O_2 deficit at the onset of a sprint and leaving more of the anaerobic capacity for later during the sprint. An increase in oxidative ATP generation could result not only from PE-mediated mobilisation of cardiorespiratory system, but also from the priming of mitochondrial fuel oxidation via pre-sprint PDH activation (Gibala and Saltin, 1999) or increased acetylcarnitine concentrations (Campbell et al., 2002). Also, although it has been proposed that an increase in H^+ and CO_2 levels in response to PE could increase oxygen delivery by promoting increased vasodilation and dissociation of oxyhaemoglobin (Gerbino et al., 1996), this is unlikely to be the case in this study, given the absence of significant changes in plasma lactate levels and pH across all the PE conditions examined here, which corroborate the findings of many previous studies that light exercise at ≤ 40% $\dot{V}O_2$ peak does not alter metabolic acidaemia (De Bryun and Lefebvre, 1980; Genoveley and Stamford, 1982; Bogdanis, 1996).

Overall, our findings show for the first time that the mechanisms whereby PE affects power output vary throughout a sprint. This has been overlooked by all studies concerned with the effect of PE on sprint power output, in part, because most of these studies have focused on PP alone or in combination with mean power/total work (Margaria et al., 1971; Inbar and Bar-Or, 1975; Bergh and Ekblom, 1979; Dolan and Sargeant, 1983; Dolan et al., 1983; Sargeant and Dolan, 1987; McKenna and Green, 1987; Hawley et al., 1989; Obrian et al., 1997). The problem with this latter indicator of power output is that mean power incorporates the effect of early as well as late power output, both of which relying on different fuel systems and which have been shown here to respond differently to PE. Another important confounding variable that was considered in this and only some other studies (Inbar and Bar-Or, 1975; Hawley et al., 1989) is the possibility that the benefit of PE might be psychological as opposed to physiological. This is an important issue given evidence from previous studies that a placebo effect could contribute to the effect of PE on exercise performance (Malarecki, 1954; Massey et al., 1961). In order to minimise such a placebo effect, the participants in this study were deceived by being informed that we were primarily concerned with the effect of PE on blood profile, and no mention was made about our focus on sprint performance.

In summary, our findings suggest for the first time that there is a temporal transition in the mechanisms responsible for the effect of PE on power output during a maximal 30 s sprint effort, with temperature-dependent mechanisms playing a more important role at the onset of the sprint and mobilisation of the cardiorespiratory system playing a more important role later. On practical grounds, this suggests that in order to improve sprint performance of short distance a prolonged PE (20 min) is better than shorter one, where as for sprint like 30 s, a shorter PE (4 min) is just as effective as the long one (20 min) as long as no rest is allowed between PE and the sprint. This latter condition is likely to be more difficult to achieve because $\dot{V}O_2$ falls

rapidly to resting levels if PE is followed by a short rest period. Since, it is expected that several minutes might elapse between the end of PE and the beginning of a competitive sprint event, increased mobilisation of the cardiorespiratory prior to sprinting might be difficult if not impossible to achieve in comparison to increasing muscle temperature.

CHAPTER 3

Preparatory strategies for optimising an all-out sprint effort II:

Effect of active recovery post-sprint on subsequent sprint performance

3.1 Introduction

The rate of recovery following a bout of intense exercise is an important factor limiting subsequent maximal sprint performance, with the accumulation of H+ associated with that type of exercise being one of many factors contributing to fatigue. For this reason, it has been argued that any recovery protocol increasing the rate of blood lactate and H+ disappearance should also increase the rate of recovery of sprint performance (Newman 1937; Gisolfi et al. 1966; Hermansen and Stensvold, 1972; Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Stamford et al., 1978b; Dodd et al., 1984; Fairchild et al., 2003). One such a strategy consists of performing exercise at a low intensity (30%-60% $\dot{V}O2$ peak) following high-intensity exercise, a recovery protocol referred to as active recovery. Since this protocol of recovery has been found to be superior to a complete rest (passive recovery) in enhancing the rate of blood H+ disposal post-exercise together with the early views that H+ is a mediator of fatigue (Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Dodd et al., 1984; Fairchild et al., 2003), this has generally been taken as the basis to suggest that active recovery also speeds up recovery of maximal sprint capacity.

Unfortunately, most of the studies on the effect of active recovery on lactate and H^+ disposal have not examined the effect of active recovery on subsequent exercise performance (Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Dodd et al., 1984; Fairchild et al., 2003). However, those that have addressed this issue have reported that the impact of active recovery following one sprint or the first of several consecutive sprints on subsequent single sprint performance is in general either small (<3%; Weltman et al., 1977; Thiriet et al., 1993; Bogdanis et al., 1996, Felix et al., 1996) or not significant (Weltman, et al, 1979, Bond et al., 1991; Signorile et al., 1993; Ahmaidi et al., 1996; Monedero and Donne, 2000; Connolly et al., 2003). A

close inspection of these studies, however, reveals that some aspects of their experimental designs might have led to an underestimation or overestimation of the impact of active recovery. In particular, the underestimation of the benefits of active recovery reported by some might have resulted from the duration of the exercise bout being too short (5-6 seconds) to affect subsequent sprint performance (Signorile et al., 1993; Ahmaidi et al., 1996; Connolly et al., 2003). In other studies with longer exercise duration leading to fatigue, passive and active recovery have been compared 14-20 min post-sprint (Weltman et al., 1977, 1979, Bond et al., 1991; Thiriet et al., 1993; Felix et al., 1996; Monedero and Donne, 2000) because the differences in lactate levels between these two recovery protocols are close to maximal at this time. The difficulty here is that this recovery duration might be inappropriate as suggested by the observation that 20 min of passive recovery is long enough to recover the ability to perform exercise at a high intensity (Weltman et al., 1977, 1979). It is not surprising, therefore, that prolonged active recovery has been reported to offer little or no additional benefit under these conditions (Weltman et al., 1979; Bond et al., 1991; Thiriet et al., 1993; Felix et al., 1996; Done et al., 2000).

It must be stressed that in the studies where active recovery has been shown to improve recovery of exercise performance capacity, albeit to a minor extent, the possibility of a placebo effect and thus of an overestimation of the benefit of active recovery has not been taken into account (Weltman et al., 1977; Thiriet et al., 1993; Bogdanis et al., 1996, Felix et al., 1997); a limitation shared by all studies concerned with the effect of active recovery on sprint performance. This is an important issue considering that the participants recruited in several studies have been athletes (Signorile et al., 1993; Thiriet et al., 1993; Felix et al., 1996; Bogdanis et al., 1996; Monedero et al., 2000; Connolly et al., 2003) who are highly likely to have been aware of the alleged benefits of active recovery.

Given that it is still unclear whether active recovery can improve recovery of sprint performance following a bout of intense exercise, it was our primary goal to address this issue by adopting the following precautions to avoid the aforementioned limitations. Firstly, the duration and intensity of the sprint should result in a marked fall in power output, and secondly, active and passive recovery should be compared at a time when recovery of exercise performance is incomplete. To this end, we aimed to determine the temporal pattern of recovery of power-output post-sprint to identify the most suitable time to compare recovery protocols, and special care was taken to minimise the possibility that a placebo effect might explain different responses to active and passive recoveries. We hyphothesised that active recovery performed when recovery of sprint performance capacity is incomplete will result in a better restoration of sprint power than passive recovery, even when the possibility of a placebo effect is minimised, but not if both protocols are compared when recovery is almost complete. Hopefully, the information thus gathered will help in determining the efficacy of active recovery as a means of accelerating recovery from an intense sprint effort.

3.2 Materials and Methods

3.2.1 *Participants*

Seven competitive cyclists and triathletes for whom sprinting was part of their training programmes volunteered to participate in this study. Their mean (Mean ± SD) age, height, body mass and $\dot{V}O_{2\ peak}$ were 29 ± 4 y, 177.2 ± 3.0 cm, 77.7 ± 10.2 kg and 58.1 ± 11.2 $mL.kg^{-1}.min^{-1}$, respectively. All participants were informed about the purpose of the study and possible risks associated with the methods and procedures adopted during the testing sessions. They were also asked to sign an informed consent form and told that they could withdraw from the study at any time. This study was approved by the Human Rights Committee of the University of Western Australia.

3.2.2 *Familiarisation session*

Each participant completed a familiarisation session prior to testing in order to become accustomed with the equipment, the exercise protocols, and researchers involved in the study. During this session, anthropometric data (height, body mass) as well as $\dot{V}O_{2\ peak}$ were also determined.

3.2.3 *Effect of recovery time on sprint capacity*

Each participant visited the laboratory on eight distinct mornings, with a one-week period between consecutive visits. Participants were also asked to observe the same pre-testing diet and to refrain from any intense physical activity for 24 h before testing, and for these reasons were asked to complete a training and dietary diary. On each visit, participants performed a 5-min warm-up on a front-access cycle ergometer (Repco, NSW, Australia), which consisted of light cycling at 40% $\dot{V}O_{2\ peak}$ followed by 5 min of recovery. This warm-up protocol was adopted because as shown in Chapter 2, it does not result in changes in muscle and rectal temperature

as well as changes in mobilisation of cardio-respiratory system. Afterwards, each participant was asked to stand still on the ergometer just prior to cycling at maximal intensity for the next 30 s as described in more details later. Following exercise, they were allowed to recover for either 0, 1, 2, 5, 10, 15, 20 or 40 min. The duration of the recovery protocol was assigned at random in order to avoid any order effect. After recovery, all participants were required to initiate the next 30-s sprint. During the first and second sprint, PP, MP_{30}, MP_{10}, MP_{-20} and MP_{-10} were recorded, and blood was sampled as described below.

3.2.4 Effect of active and passive recovery on sprint capacity

The experimental design adopted here was similar to that described above, with the difference that the recovery periods between the two 30-s maximal sprint efforts on the cycle ergometer lasted either 4 or 20 min. The 4-min recovery period was chosen because full recovery of all indicators of sprint performance occurred within 20 min. After sprinting, all participants were asked to either remain in a rested state or to actively recover by pedalling at an intensity corresponding to 40% of their $\dot{V}O_{2\,peak}$, with these two recovery protocols being administered in a randomised, counterbalanced order. The following 30-s all-out sprint after active or passive recovery was initiated from a still standing position. It is important to stress that all participants were partially deceived in that they were not informed that our primary goal was to examine the effect of active recovery on sprint performance, but led to believe that the main objective of this study was to examine the effect of the recovery protocol on blood variables.

3.2.5 Sprint trial

In order to perform the 30-s sprint test, each participant was asked to mount a front-access cycle ergometer (Repco, NSW), with each foot secured using a foot strap. Immediately before sprinting, participants were asked to stand-up on the ergometer

while placing both hands on the handles and remaining still. The trial was initiated with the foot of the dominant leg starting at the "two o'clock" position after a three-second count down. All participants were instructed to cycle in an "all-out" fashion without pacing themselves for 30 s. During each 30-s sprint, the following information was recorded; peak power output (PP), mean power output over 30 s (MP_{30}), mean power output over the first 6 s (MP_6) and 10 s (MP_{10}), and mean power output over the last 20 (MP_{-20}) and 10 s (MP_{-10}).

3.2.6 $\dot{V}O_{2\,peak}$ determination

$\dot{V}O_{2peak}$ was determined on the same cycle ergometer as that used for the experimental testing during the familiarisation session. The $\dot{V}O_{2peak}$ test consisted of graded exercise steps using an intermittent protocol (one-minute rest between stages). The test commenced at 50 W and, thereafter, the intensity was increased by 30 W every 4 min until volitional exhaustion. Participants were required to maintain the set power output, which was displayed on a computer screen in front of them. The test was stopped when the participants could no longer maintain the required power output or when the respiratory exchange ratio was above 1.15. Strong verbal encouragement was provided to each participant, particularly towards the end of the test. All inspired and expired air was collected through a mouthpiece connected to a Hans-Rudolf valve attached via Collins tubing to an on-line gas analysis. All expired air was continuously monitored for the analysis of O_2 and CO_2 concentrations (Ametek gas analysers SOV S-3A and COV CD3A respectively, Pittsburg, Pa., USA). Data was averaged over 30 s intervals. Ventilation was also recorded every 30 s using a turbine ventilometer (Morgan, Model 096, Kent, England). The gas analysers were calibrated immediately before and verified after each test using three certified gravimetric gas mixtures (BOC gases, Chatswood,

Australia). The ventilometer was calibrated pre-exercise and verified post-exercise using one litre syringe in accordance with the manufacturer's instruction.

3.2.7 Blood Sampling

Capillary blood (120 µL) was obtained from an ear lobe. A cutaneous vasodilator cream (Finalgon®, Boehringer Ingelheim, Atarmon, Australia) was applied 10 min prior to collection of the initial sample, and a sterile lancet (SoftTouch®, Roche Diagnostic Australia) was used to puncture the skin at the margin of the earlobe before blood sampling. All blood samples were analysed for pH and lactate levels with a blood-gas analyser (ABL 625 series, Radiometer, Copenhagen).

3.2.8 Statistical analyses

All data, with the exception of the descriptive characteristics of the participants which were reported as means ± standard deviation (S.D.), are reported as means ± standard error of mean (S.E.M.). Statistical analyses were performed using a one-way ANOVA with repeated measures. A pair-wise comparison using Fisher LSD test was adopted to compare means between treatments. Significance was set at $p < 0.05$. All statistical analyses were conducted using the SPSS 12.0 for Windows Statistical Package (version 12.0, SPSS, Chicago, IL).

3.2 Results

3.3.1 Effect of recovery duration post-maximal sprint effort on subsequent sprint performance

There were no significant differences in any of the measured indicators of sprint power output recorded for the first sprints across the eight testing sessions used to determine the temporal pattern of recovery of sprint performance, with an average PP of 1192 ± 58 W, MP_{30} of 751 ± 33 W, MP_{10} of 967 ± 47 W, MP_{-20} of 644 ± 31 W and MP_{-10} of 527 ± 23 W. During recovery from the 30-s maximal sprint effort, a rapid rate of restoration of sprint performance took place across all indicators of power output, with the time required to recover 50% of PP (Fig. 3.1), MP_{30} (Fig. 3.2), MP_{10} (Fig. 3.3), MP_{-20} (Fig. 3.4), and MP_{-10} (Fig. 3.5) of 1.96 ± 0.38, 2.03 ± 0.78, 1.36 ± 0.76, 2.47 ± 0.42 and 4.93 ± 0.61 min, respectively. Full recovery of all indicators of sprint performance took place within 15-40 min ($p < 0.05$).

3.3.2 Pattern of change in blood pH and lactate levels

In response to 30 s of maximal sprint effort, blood lactate increased to reach maximum levels 5 min post-sprint ($p<0.05$; 16.9 ± 0.8 $mmol.L^{-1}$), and decreased progressively thereafter over the whole duration of the recovery period (Fig. 3.6). In response to exercise, blood H^+ levels increased from resting levels of 3.845 ± 10^{-8} $mmol.L^{-1}$ (pH, 7.42 ± 0.01) to a maximum of 6.532 ± 10^{-8} $mmol.L^{-1}$ (pH, 7.18 ± 0.10) after 5 min of recovery, and fell progressively thereafter (Fig. 3.7). Blood lactate and H^+ levels were inversely correlated throughout recovery ($r = - 0.97$; $p< .005$).

3.3.3 Effect of active and passive recovery on recovery of sprint performance

There were no significant differences in any of the indicators of sprint power output for all the initial sprints across the four recovery trials (passive and active recovery of

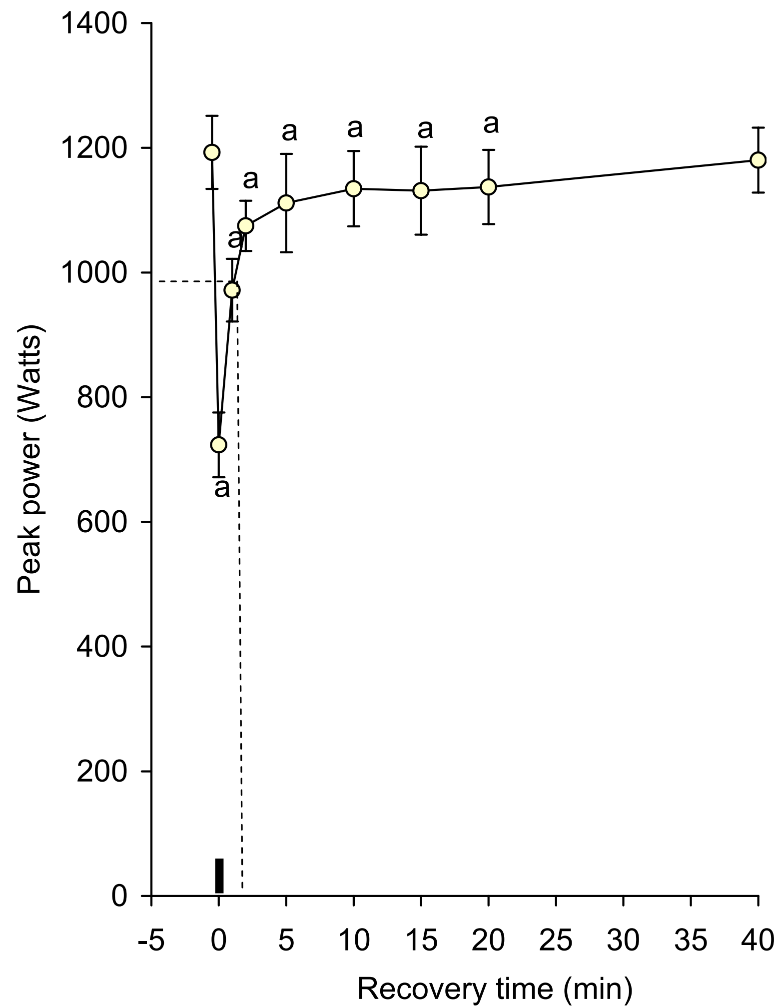

Figure 3.1: Effect of recovery time post-sprint on peak power (PP) generated during a subsequent 30-s sprint. (▪▪▪▪▪) represents 50% peak power recovery. (▌) represents initial sprint. a, significantly different from initial power ($p<0.05$).

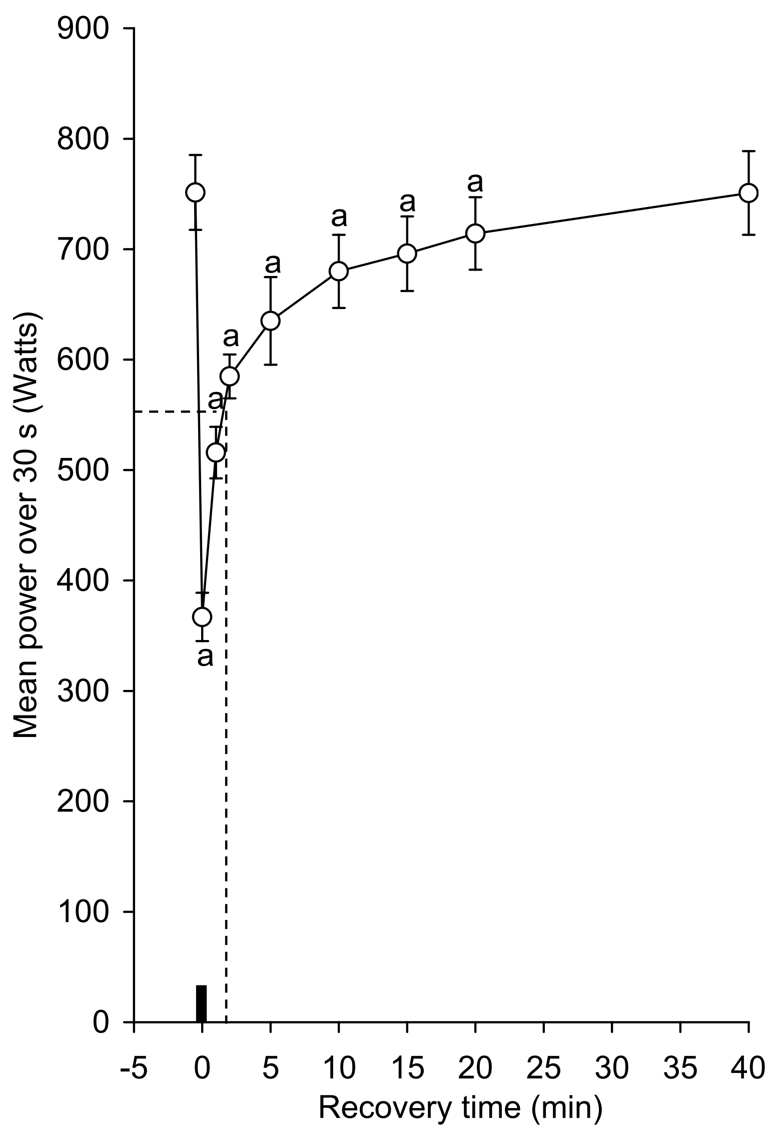

Figure 3.2: Effect of recovery time post-sprint on mean power over 30s (MP_{30}) generated during a subsequent 30-s sprint. (▪▪▪▪▪) represents 50% peak power recovery. (▮) represents initial sprint. a, significantly different from initial power ($p<0.05$).

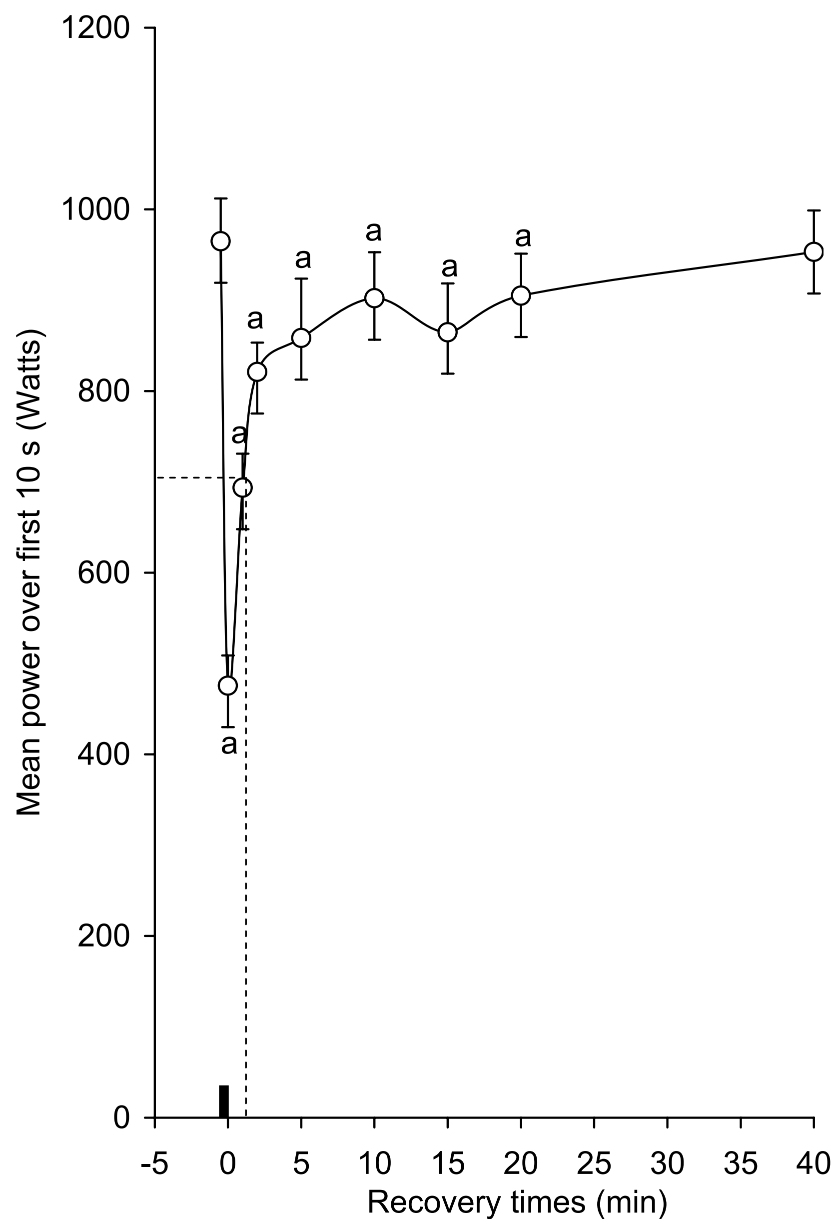

Figure 3.3: Effect of recovery time post-sprint on mean power over the first 10 s (MP_{10}) generated during a subsequent 30-s sprint. (▪▪▪▪) represents 50% peak power recovery. (▌) represents initial sprint. a, significantly different from initial power

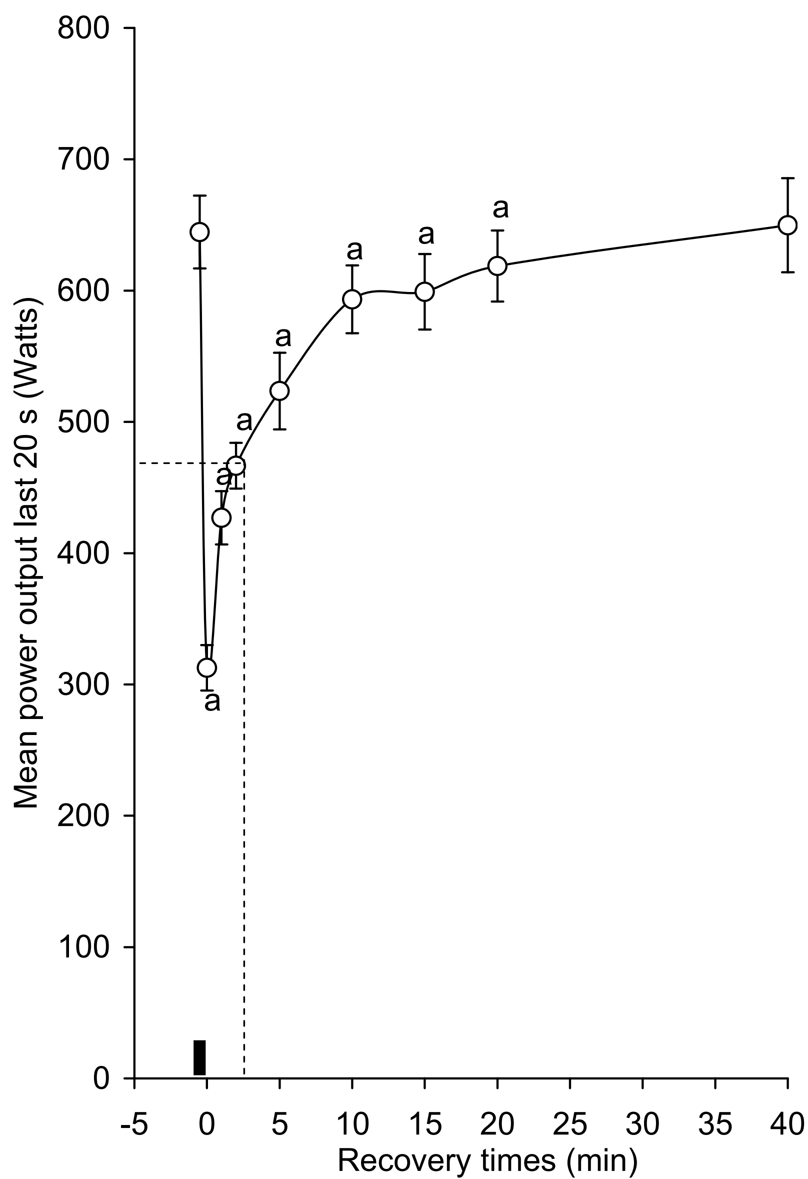

Figure 3.4: Effect of recovery time post-sprint on mean power over the last 20 s (MP_{-20}) generated during a subsequent 30-s sprint. (▪▪▪▪) represents 50% peak power recovery. (▌) represents initial sprint. a, significantly different from initial power

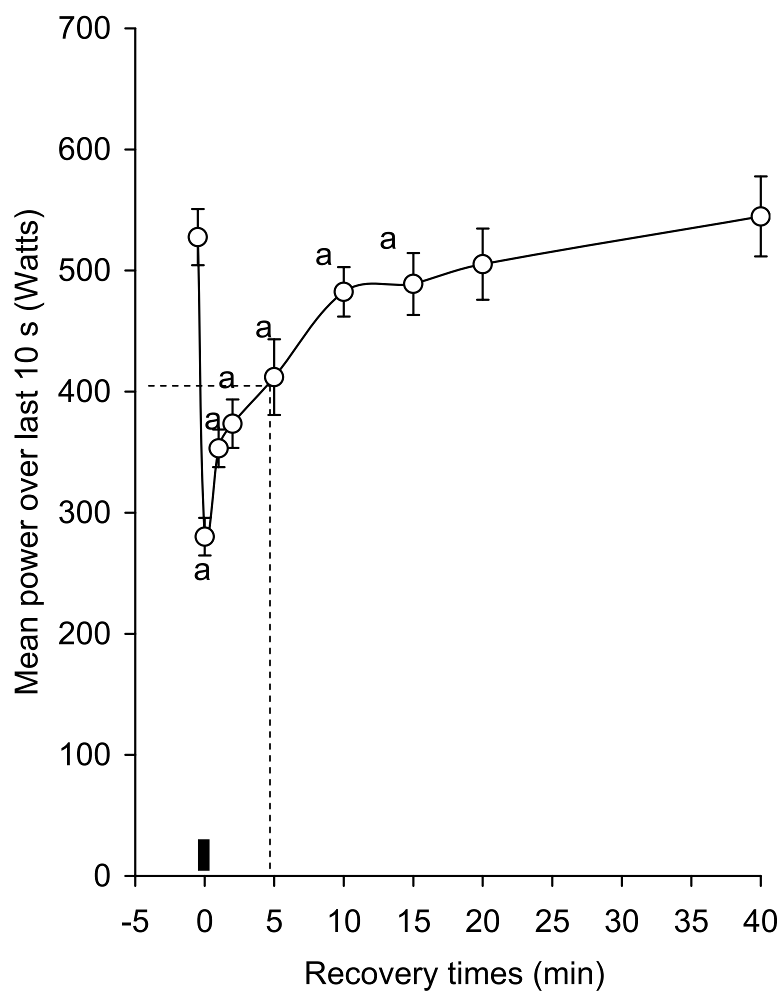

Figure 3.5: Effect of recovery time post-sprint on mean power over the last 10 s (MP_{-10}) generated during a subsequent 30-s sprint. (▪▪▪▪) represents 50% peak power recovery. (▌) represents initial sprint. a, significantly different from initial power

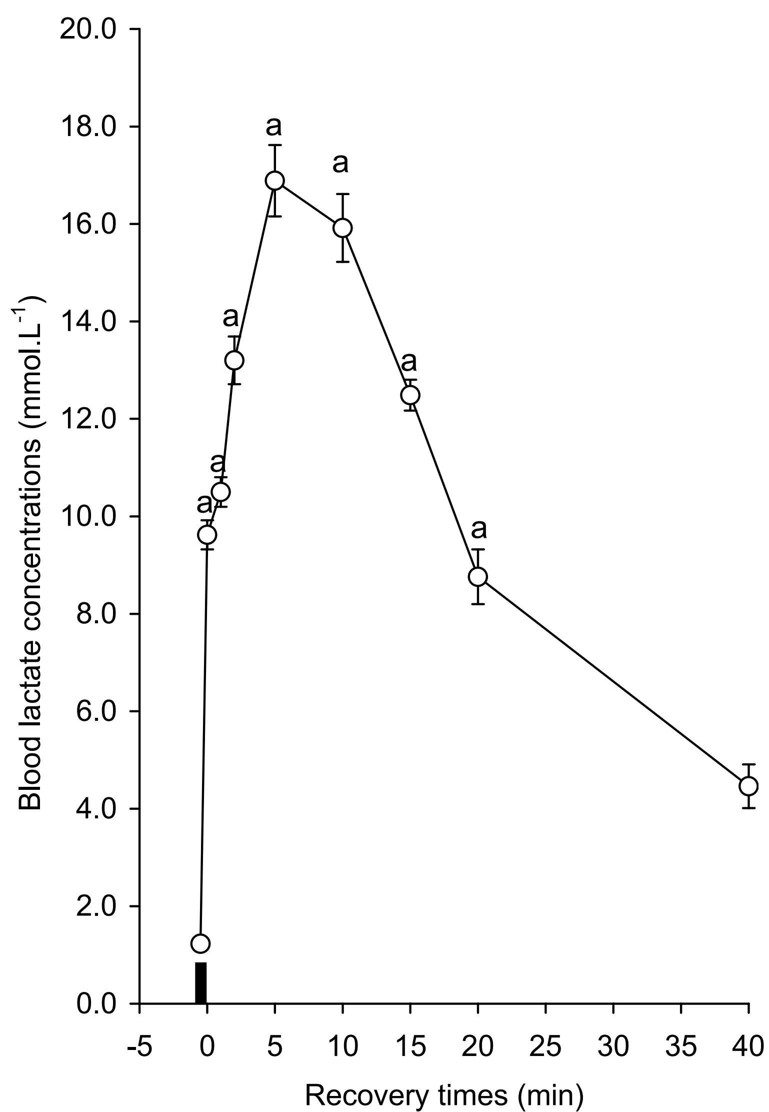

Figure 3.6: Effect of a 30-s sprint and recovery on blood lactate concentrations. (▌) represents initial sprint. a, significantly different from baseline levels ($p<0.05$).

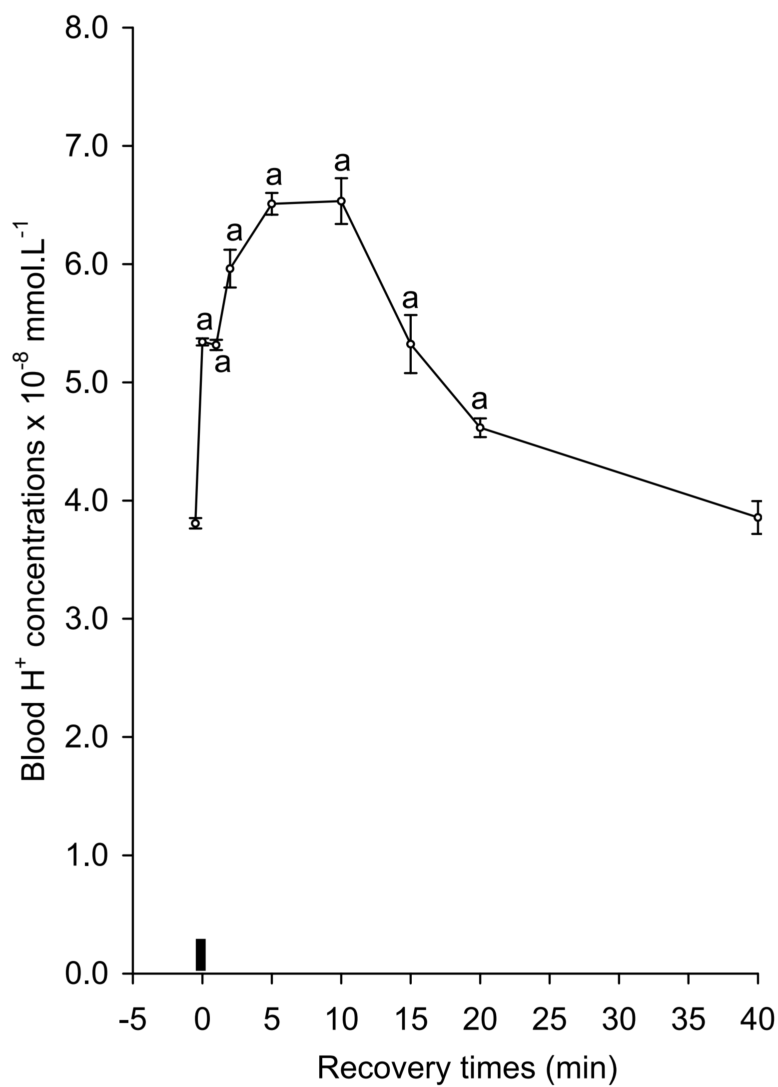

Figure 3.7: Effect of a 30-s sprint and recovery on blood H^+ concentrations. (▌) represents initial sprint. a, significantly different from baseline levels ($p<0.05$).

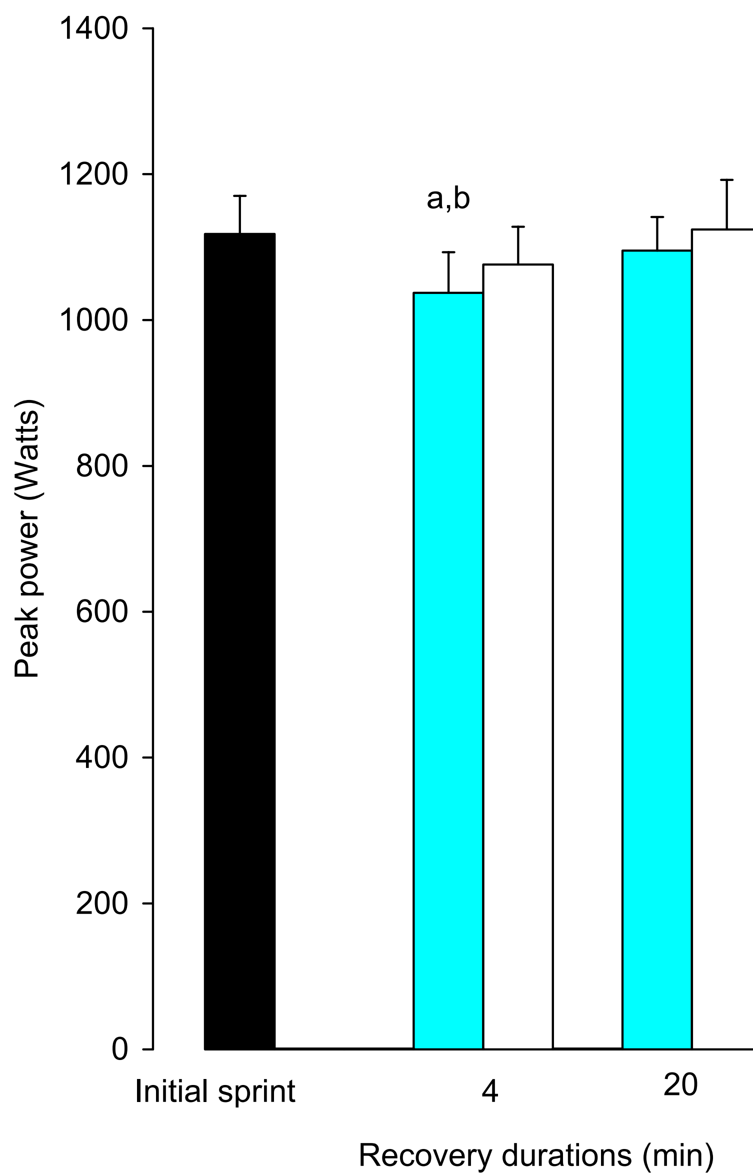

Figure 3.8: Effect of 4 and 20 min of passive ( ) and active ( ) recovery on peak power (PP). a, significantly different from initial power, b, significantly different from 20 min of recovery ($p<0.05$).

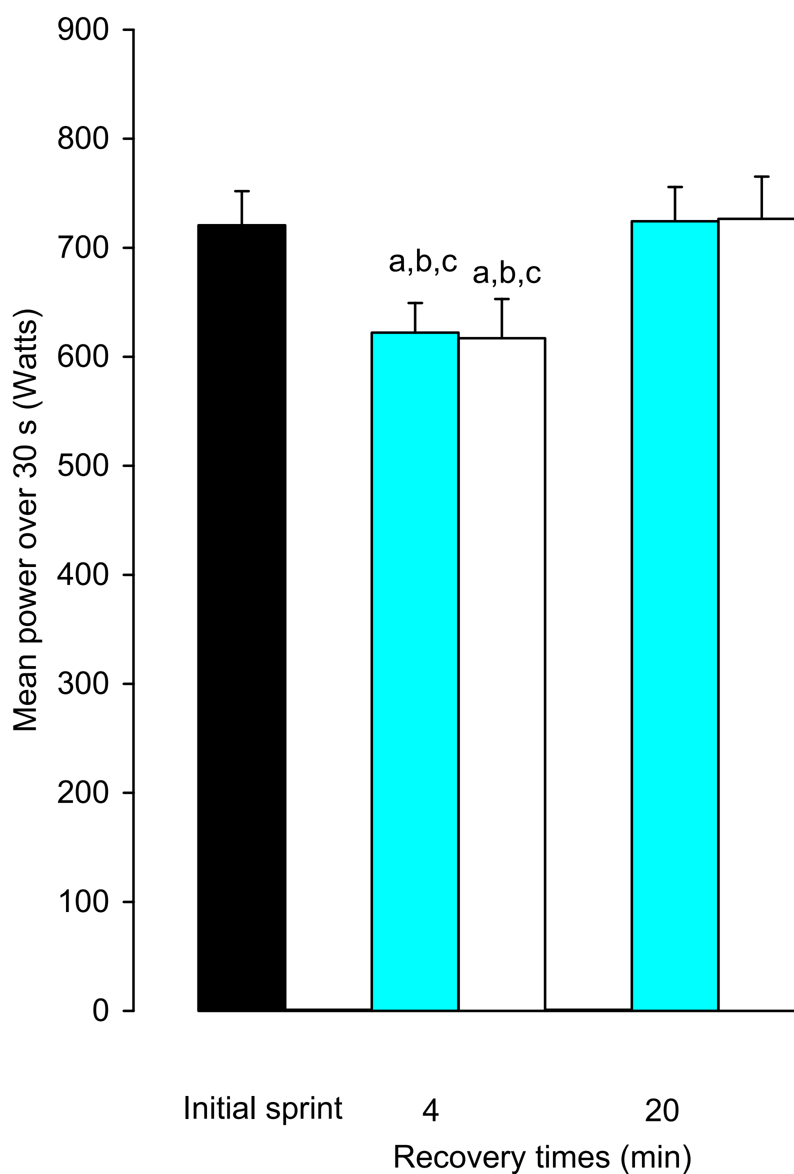

Figure 3.9: Effect of 4 and 20 min of passive ( ) and active ( ) recovery on mean power over 30s (MP_{30}) generated during a 30-s sprint. a, significantly different from initial power, b, significantly different from 20 min of passive recovery and c, significantly different from 20 min of active recovery ($p<0.05$).

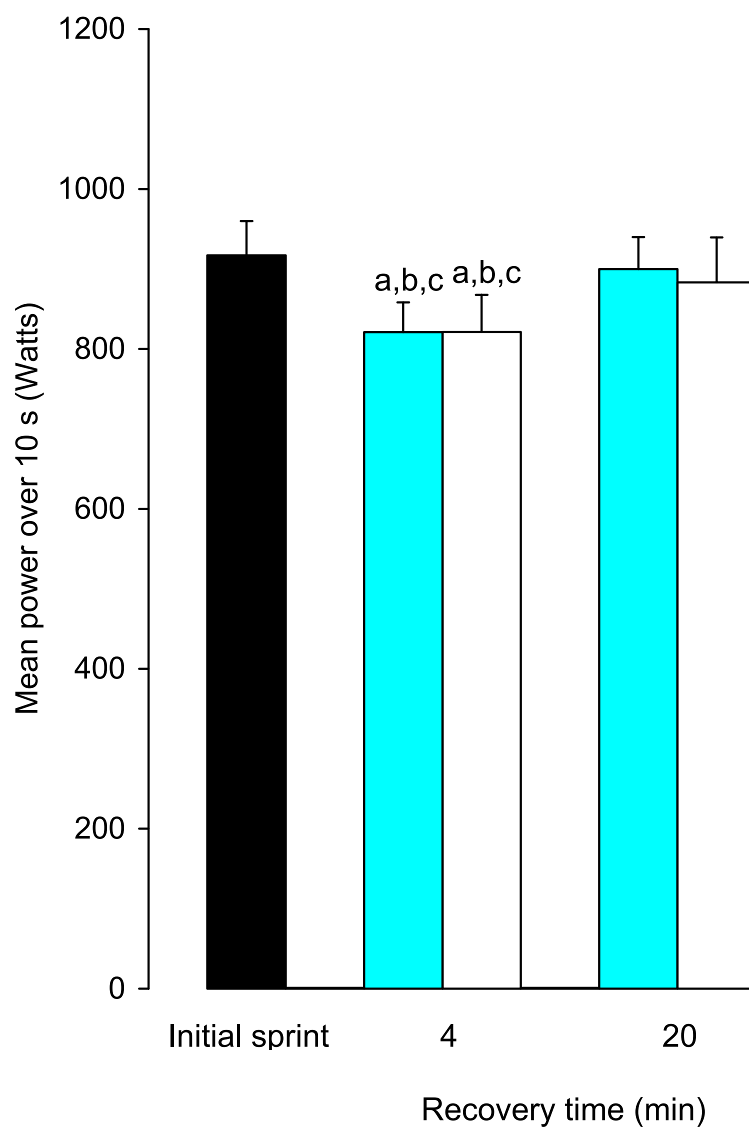

Figure 3.10: Effect of 4 and 20 min of passive ( ) and active ( ) recovery on mean power over the first 10 s (MP_{10}) generated during a 30-s sprint. a, significantly different from initial power, b, significantly different from 20 min of passive recovery and c, significantly different from 20 min of active recovery ($p<0.05$).

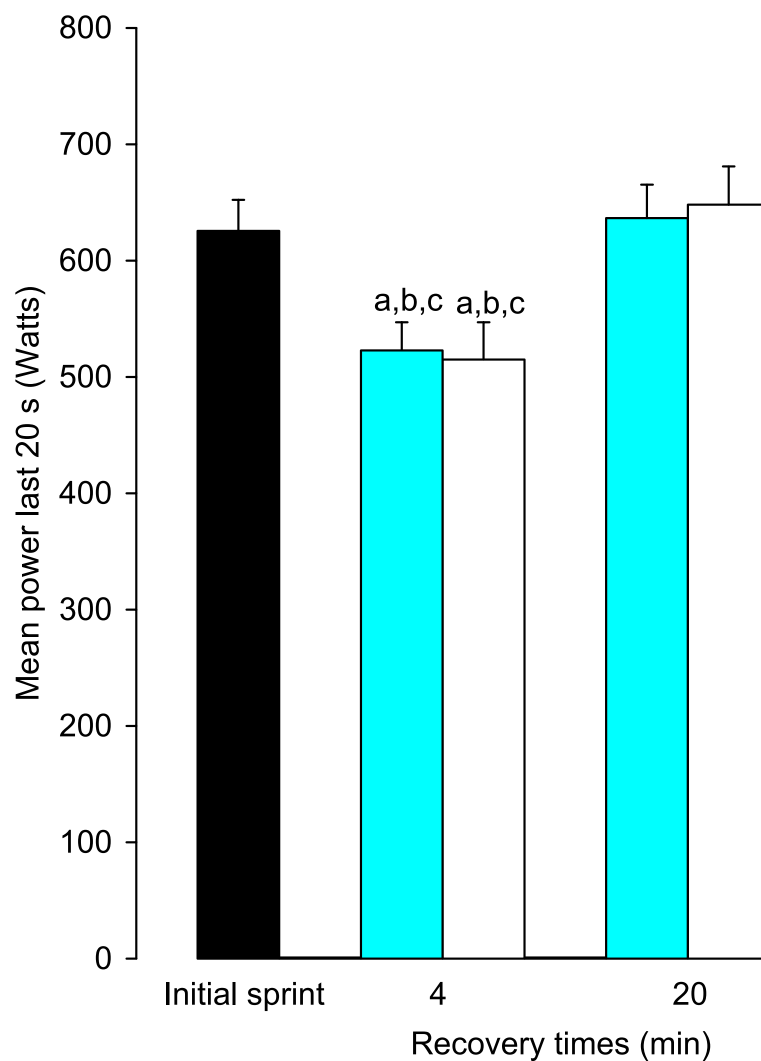

Figure 3.11: Effect of 4 and 20 min of passive ( ) and active ( ) recovery on mean power over the last 20 s (MP_{-20}) generated during a 30-s sprint. a, significantly different from initial power, b, significantly different from 20 min of passive recovery and c, significantly different from 20 min of active recovery ($p<0.05$).

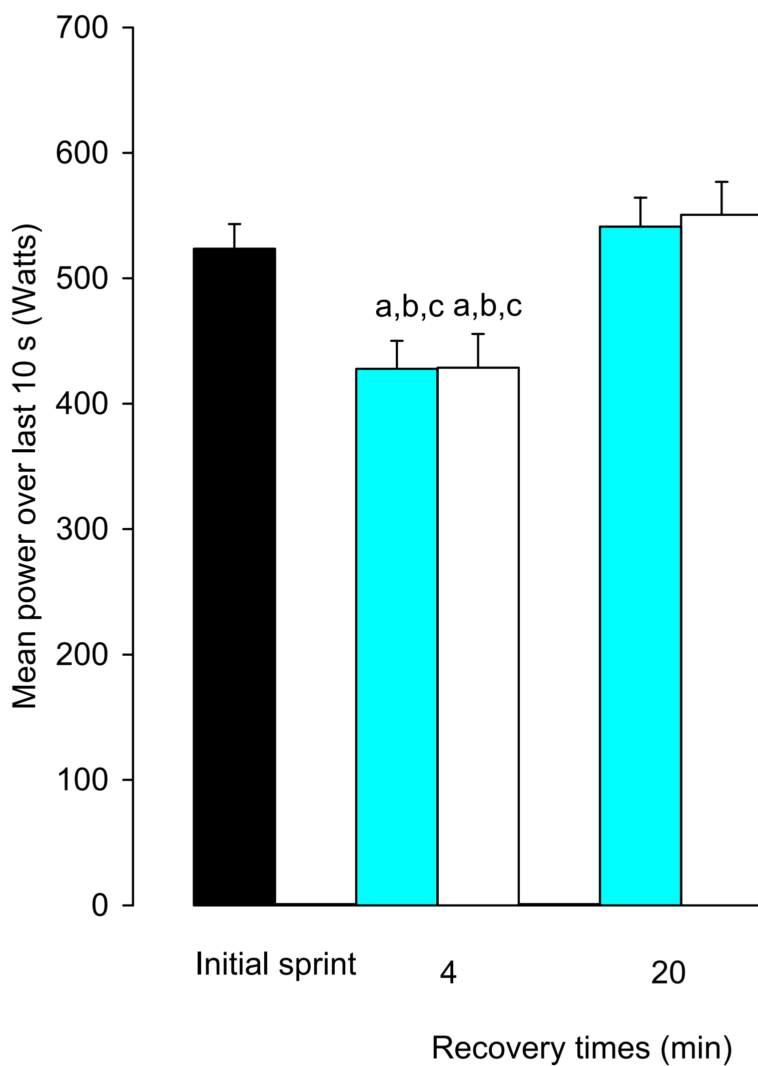

Figure 3.12: Effect of 4 and 20 min of passive ( ) and active ( ) recovery on mean power over the last 10 s (MP_{-10}) generated during a 30-s sprint. a, significantly different from initial power, b, significantly different from 20 min of passive recovery and c, significantly different from 20 min of active recovery ($p<0.05$).

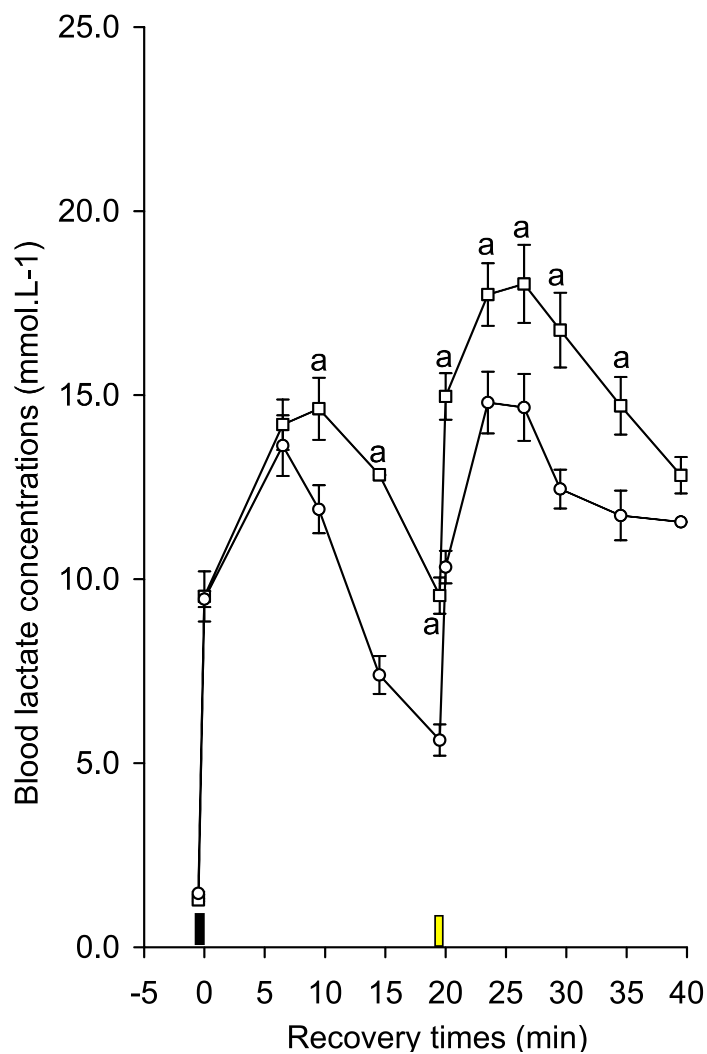

Figure 3.13: Effect of 20 min of passive ( ) and active ( ) recovery on blood lactate concentrations. ( ) represents initial and ( ) represents subsequent sprint. a, significantly different from active recovery (p<0.05).

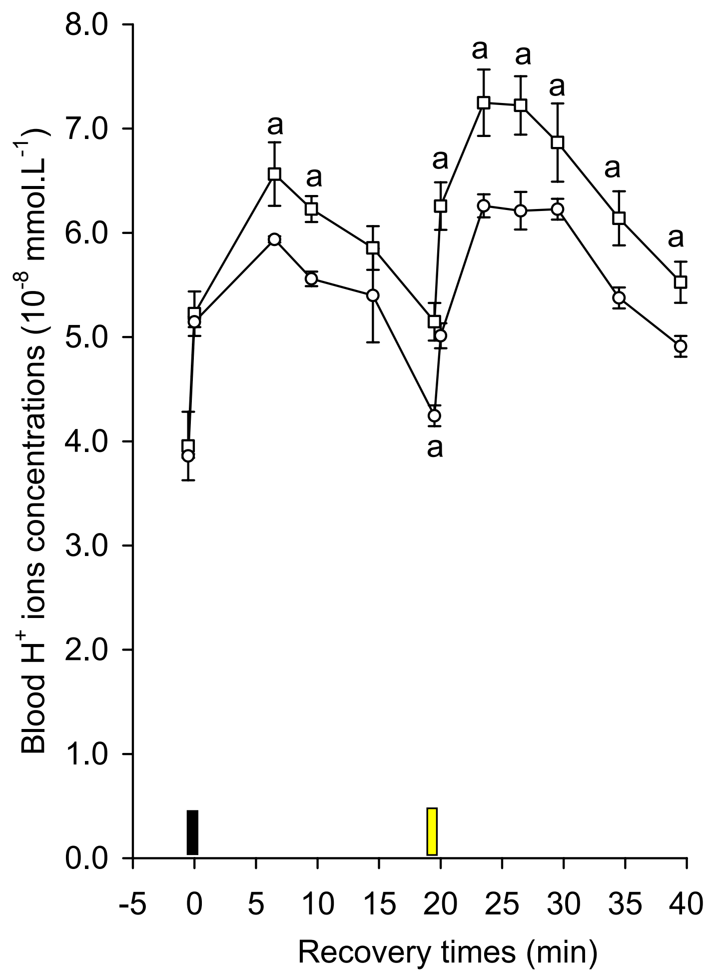

Figure 3.14: Effect of 20 min of passive ( ) and active ( ) recovery on H^+ concentrations. ( ) represents initial and ( ) represents subsequent sprint. a, significantly different from active recovery ($p<0.05$).

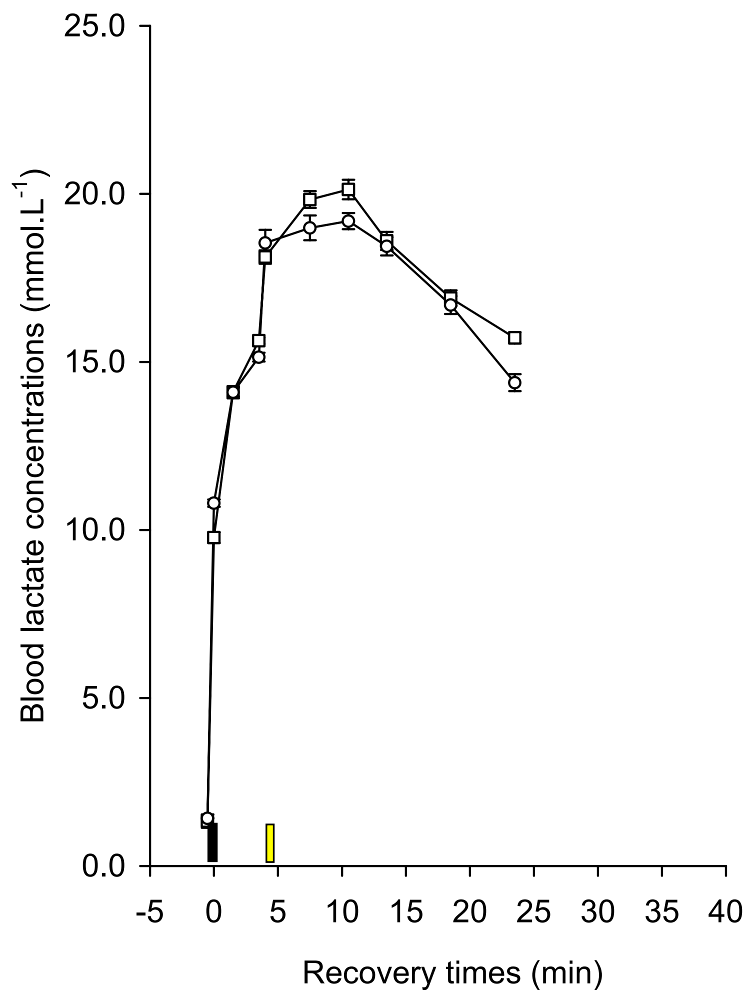

Figure 3.15: Effect of 4 min of passive (□) and active (○) recovery on blood lactate concentrations. (▮) represents initial and (▯) represents subsequent sprint. No significant differences between treatments (p>0.05).

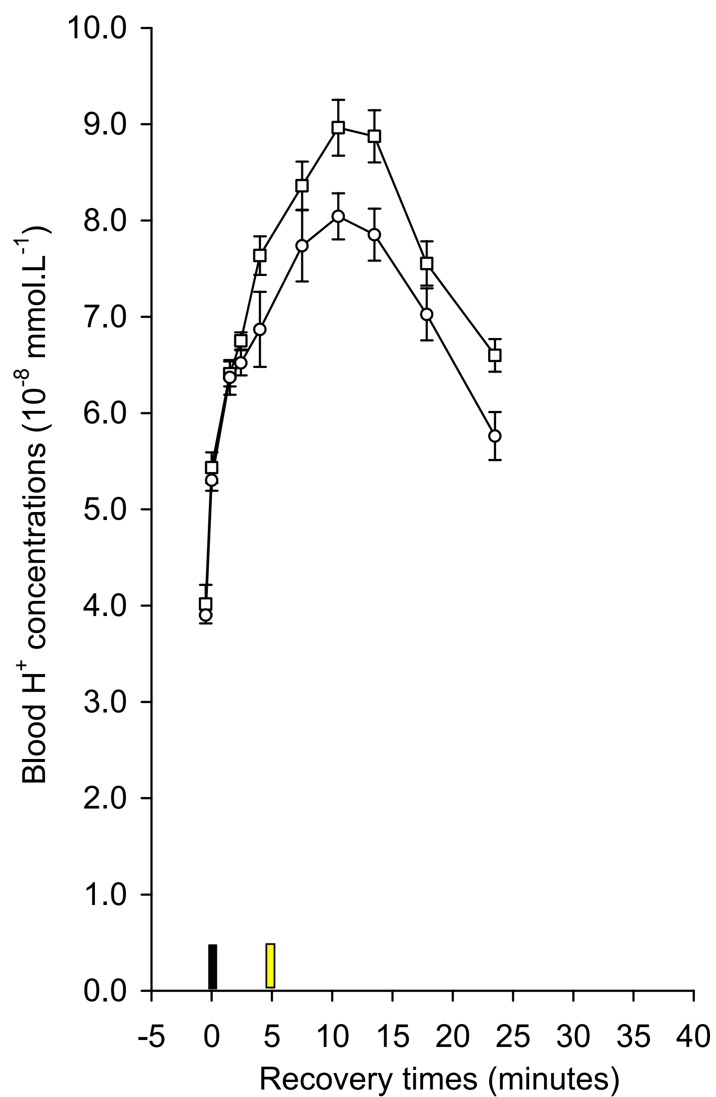

Figure 3.16: Effect of 4 min of passive (□) and active (○) recovery on H^+ concentrations. (▮) represents initial and (▯) represents subsequent sprint. No significant differences between treatments ($p>0.05$).

4 and 20 min), with PP, MP_{30}, MP_{10}, MP_{-20}, and MP_{-10} being 1118 ± 52 W (Fig, 3.8), 720.6 ± 31.3 W (Fig. 3.9), 917.1 ± 42.8 W (Fig. 3.10), 625.5 ± 26.7 W (Fig. 3.11), and 523 ± 19.7 W (Fig. 3.12), respectively. After 20 min of either passive or active recovery, PP, MP_{30}, MP_{10}, MP_{-20}, and MP_{-10} returned to levels not different from the first sprint (Fig. 3.8-3.12), whereas recovery was incomplete after 4 min of either passive or active recovery ($p<0.05$; Fig 3.8-3.12). After 4 or 20 min of recovery, PP, MP_{30}, MP_{10}, MP_{-20}, and MP_{-10} were not significantly different between active and passive recovery ($p<0.05$; Fig. 3.8-3.12).

3.3.4 Effect of 4 and 20 min of active and passive recovery on recovery of H^+ and blood lactate levels

In response to the 30-s maximal sprint effort, the increase in blood lactate and H^+ levels was higher in the passive as opposed to the active recovery trial (Fig. 3.13, 3.14). Over the next 20 min, blood lactate and H^+ levels reached lower levels in response to active than passive recovery ($p<0.05$; Fig. 3.13, 3.14). In response to the second sprint performed after 20 min of recovery, lactate and H^+ levels increased to a similar extent in both trials, but attained higher concentrations in the passive recovery trial ($p<0.05$) because of higher pre-sprint lactate and H^+ levels (Fig. 3.13, 3.14). In contrast, the extent of the rise and levels of lactate and H^+ attained when the second sprint was initiated after 4 min of recovery did not differ between trials (Fig. 3.15, 3.16).

3.4 Discussion

It is well established that active recovery after a single sprint accelerates the return of blood pH and lactate to basal pre-exercise levels (Newman 1937; Gisolfi et al. 1966; Hermansen & Stensvold, 1972; Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Weltman et al., 1977, 1979; Stamford et al., 1978b; Dodd et al., 1984; Bond et al., 1991, Thiriet et al., 1993; Fairchild et al., 2003). However, there are still some doubts as to whether it can improve recovery of sprint performance after a single sprint, since most studies have reported that the impact of active recovery after either one sprint or the first of several consecutive sprints is either small (<3%; Weltman et al., 1977; Thiriet et al. 1993; Bogdanis et al., 1996; Felix et al., 1996) or undetectable (Weltman et al., 1979; Bond et al., 1991; Signorile et al., 1993; Ahmaidi et al., 1996; Monedero and Donne, 2000; Connolly et al., 2003). It is possible that the effectiveness of active recovery might have been underestimated due to of the short duration of the exercise bout in some studies (6-15 s; Signorile et al., 1993; Ahmaidi et al., 1996; Connolly et al., 2003) or the long recovery duration (14-20 min) adopted so that full recovery had already occurred (Weltman et al., 1977, 1979; Bond et al., 1991; Thiriet et al., 1993; Felix et al., 1996; Monedero and Donne, 2000). On the other hand, those studies that have reported a beneficial effect of active recovery might have overestimated the effect of active recovery as a result of a placebo effect (Weltman et al., 1977; Thiriet 1993; Felix et al., 1996; Bodganis et al., 1996). This is an important confounding variable given that the alleged benefits of active recovery have been extensively promoted to athletes since the demonstration nearly 40 years ago that active recovery accelerates the normalisation of blood pH and lactate levels after intense exercise. For these reasons, we undertook to compare active and passive recovery at a time when recovery from a 30 s maximal sprint was either incomplete or complete in trained athletes previously deceived about our goals. To this end, the time-dependent

pattern of recovery of several indicators of sprint power output were determined, and we found that they ranged between 1.4-4.9 min with nearly full recovery takes place within 20 min. This is in agreement with the findings of others who have reported that sprint performance recovers rapidly (Cherry et al, 1998, Sahlin and Ren, 1989; Bogdanis et al., 1995). They have chosen to compare recovery protocols 4-min after the end of the sprint since by that time the indicators of sprint power adopted here had recovered by 45 - 75%. Despite active recovery resulting in a faster return of blood lactate and H^+ to pre-exercise levels, neither 4 nor 20 min of active recovery had a beneficial effect on the recovery of any of the indicators of sprint power output examined here.

Although our results corroborate those of many others who have reported that active recovery after a single sprint offers no significant benefit on recovery of sprint performance, previous findings are largely inconclusive (Weltman et al., 1979; Bond et al., 1991; Signorile et al., 1993; Monedero and Donne, 2000; Connolly et al., 2003). This is because, as mentioned earlier, the absence of a benefit of active recovery after a single sprint in some of these studies is best explained on the basis that they adopted a sprint duration that was too short (6-15 s) to cause a large fall of power output and a recovery duration long enough for full recovery of sprint power capacity. (Signorile et al., 1993; Connolly et al., 2003). It must be stressed, however, that these studies were primarily concerned with the effect of active recovery on repeated short sprint performance, and although they reported that active recovery improved sprint performance after several consecutive exercise bouts, it had no effect after the first sprint. Those who have examined the effect of active recovery following intense exercise of longer duration (1-6 min) at 100-150% $\dot{V}O_{2peak}$ have also reported the absence of any improvement in sprint performance in response to active recovery, but have compared passive and active recovery 20 mins after

exercise (Weltman et al., 1979; Bond et al., 1991, Monedero and Donne, 2000). As shown in this and previous studies, this recovery duration is likely to have allowed full recovery to take place despite lactate and H^+ remaining elevated (Fig. 3.2-3.6; Weltman et al., 1977, 1979).

The aforementioned limitations do not hold for all studies on active recovery, since two studies have investigated the effect of active recovery of short duration (3-4 min) on all-out sprint performance (Bogdanis et al., 1996; Connolly et al., 2003), with the study of Connolly and colleagues (2003) corroborating our findings but not those of Bogdanis and colleagues (1996) who reported a positive but small effect of active recovery. In this latter study, 4 min of active recovery after a 30-s sprint resulted in a significant 2–3 % improvement in MP_{30} and $MP_{\mathbf{10}}$ (Bogdanis et al., 1996). It is possible that the differences between our findings and those of Bogdanis result from the training status of our participants probably exceeding that of theirs, as suggested by the much higher $\dot{V}O_{2\ peak}$ of our participants (58.1 ± 11.2 $mL.kg^{-1}.min^{-1}$). Indeed, 4 min of active recovery from several 30-s bouts of intense exercise has been shown to have a more pronounced effect on recovery of sprint power output capacity in untrained than trained individuals (Spierer et al., 2004). Another factor likely to explain the marginal improvement in sprint performance capacity (<3%) reported by Bogdanis and colleagues (1996) is that a placebo effect might have accounted for their findings, a limitation shared by most studies on active recovery (Weltman et al., 1977, 1979; Bond et al., 1991; Signorile et al., 1993; Thiriet et al., 1993; Bogdanis et al., 1996, Felix et al., 1996; Ahmaidi et al., 1996; Monedero and Donne, 2000; Connolly et al., 2003; Dupont et al., 2003; Spierer et al., 2004). This is an important issue considering that the participants recruited in the study of Bogdanis and colleagues (1996) were recreational athletes, thus highly likely to have been informed about the alleged benefits of active recovery. Given the

importance of minimizing the possibility of a placebo effect, the participants in this study were deceived by being led to believe that we were only concerned with the effect of active recovery on blood variables.

Another important issue raised by our findings is the identity of the factors causing fatigue during sprinting and affecting the rate of power recovery post-sprint. The pattern of change in power output during the bout of maximal sprint cycling examined here is comparable to that reported by others (Lakomy 1986; Boobis et al., 1987; Bogdanis et al., 1994, 1995, 1996; Nevil et al., 1996), with the early fall in power shown by others to correlate well with the depletion of phosphocreatine stores (Bogdanis et al., 1994). Consistent with these observations, the short half-time to recovery of PP and MP_{10} reported here is similar to that reported for phosphocreatine repletion after an all-out 30 s maximal sprint effort (Bogdanis, 1995). Moreover, given that plasma lactate and H^+ levels were at maximal or near maximal levels during that time for both recovery protocols and the findings by others that intramuscular pH and lactate levels correlates poorly with sprint performance recovery during that time (Bogdanis, 1995), this suggests that the extent of recovery of PP and MP_{10} after a 30-s sprint is not determined by changes in intramuscular H^+ levels.

The longer half-time for MP_{-20} (≈ 2.5 min) and MP_{-10} (≈ 5 min) recovery reported here in comparison to that of phosphocreatine repletion suggests that phosphocreatine-independent mechanisms also mediate the fatigue of these indicators of power output during a sprint. This is to be expected given that the energy demands for ATP regeneration 10 to 20 sec after the initiation of a maximal cycling sprint depends primarily on glycolysis and oxidative metabolism and little on phosphocreatine because phosphocreatine stores are at low and stable or near stable levels at that

time (Jones et al., 1983; Bogdanis et al. 1995). One might propose that the recovery of MP_{-20} and MP_{-10} results from a rise in intramuscular pH post-exercise. Indeed, this is supported indirectly by the observation that the late fall in power output during a sprint occurs with little or no change in phosphocreatine, inorganic phosphate or H_2PO_4, while correlating better with the additional increases in H^+ levels (Jones et al., 1983; Boobis et al., 1987). However, since intramuscular pH has been reported to change little during the first 6 min of recovery after a 30s cycling sprint (Bogdanis et al., 1995), this suggests that the rapid and large extent of recovery of MP_{-20} and MP_{-10} occurs independently of changes in intramuscular H^+ concentrations. This interpretation is also in agreement with recent evidence that intramuscular pH plays a role of secondary importance in muscle fatigue (Sahlin 1992; Posterino et al., 2001). Arguably, since intramuscular H^+ levels have not been measured in the present study, the possibility still remains that they might be involved in the recovery of sprint performance. Nevertheless, if one accepts the view that H^+ concentrations have little impact on recovery of PP, MP_{10}, MP_{-20} and MP_{-10}, this could explain, in part, why active recovery has little effect on the recovery of sprint performance capacity despite its effect on H^+ disposal.

In conclusion, this study does not support the view that active recovery following an intense sprint effort improves recovery of sprint performance, irrespective of recovery duration. These findings together with the mismatch between the rapid recovery of sprint power capacity and blood pH suggest that the rationale underlying the adoption of active recovery as a means to speed up recovery from a single sprint by facilitating Lactate and H^+ disposal is questionable.

CHAPTER 4

Preparatory strategies for optimising an all-out sprint effort III: Effect of active recovery between repeated all-out sprints on subsequent sprint performance

4.1 Introduction

In preparation for a maximal sprint effort performed shortly after an earlier sprint, it is generally recommended that both sprints be separated by a period of mild exercise in order to speed up recovery of sprint performance. The rationale underlying such a recovery protocol, referred to as active recovery, is that by accelerating the disposal of blood and muscle H^+, an alleged mediator of muscle fatigue, active recovery should shorten the length of time required for a subsequent sprint to be performed unimpaired. However, as mentioned in Chapter 3, all previous studies have reported that active in comparison to passive recovery has little (<3%; Weltman et al., 1977; Thiriet et al., 1993; Bogdanis et al., 1996, Felix et al., 1996) or no effect (Weltman, et al, 1979, Bond et al., 1991; Signorile et al., 1993; Monedoro and Donne, 2000; Connolly et al., 2003) on the rate of recovery of sprint performance despite faster rates of lactate and H^+ disposal. As discussed in Chapter 3, the small benefits of active recovery reported in those studies might also be attributed, at least in part, to a placebo effect.

Although active recovery following a single all-out sprint provides little or no benefit to subsequent sprint performance, there is evidence from most (Signorille et al., 1993; Thiriet et al., 1993; Ahmaidi et al., 1996; Monedero and Donne, 2000; Spierer et al., 2003; Dorado et al., 2004) but not all studies (Spierer et al., 2003; Dupont et al., 2003; Spencer et al. 2006) that active recovery between several consecutive all-out sprints provides superior recovery of sprint performance compared to passive recovery, unless recovery duration is too short for phosphocreatine repletion to take place (Dupont et al., 2003; Spencer et al. 2006). The benefit of active recovery under these conditions is, however, generally small and explained on the basis that by favouring a more rapid fall in H^+ concentration between exercise bouts, it results in a smaller cumulative increase in H^+ concentrations after each consecutive sprint,

thus delaying the onset of H^+-mediated fatigue. However, if the duration of active recovery is so short (< 15 s) that little replenishment of phosphocreatine takes place between consecutive sprints, active recovery can have a detrimental effect on recovery of a prolonged sprint (Dupont et al., 2003).

Although our findings in Chapter 3 showed that all indicators of sprint performance recovered completely within 20 min of either passive or active recovery, with no differences between recovery protocols, Thiriet and colleagues, (1993), found that mean power falls progressively in response to several consecutive 30-s sprints separated by 20 min of active or passive recovery, but to a lesser extent with active recovery. Given our findings in Chapter 3 and the work of others that early and late mean power during a 30-s sprint recover at different rates (Bogdanis et al., 1994) and respond differently to active recovery from a single sprint (Bogdanis et al., 1996), this raises for the first time the issue of whether this might also be the case in response to repeated sprints. However, it must be stressed that the experimental design in the study of Thiriet and colleagues, (1993), did not address the possibility that a placebo effect might account for the alleged benefit of active recovery. For these reasons, our primary objective was to compare the effect of 20 min of active and passive recovery on repeated sprint performance in order to determine if active recovery offers some benefit. We predicted that active recovery performed between consecutive sprints affects differentially early and late power during a sprint.

4.2 Materials and Methods

4.2.1 Participants

Nine competitive male cyclists and triathletes for whom sprinting was part of their training programmes volunteered to participate in this study. Their mean (± SD) age, height, body mass and $\dot{V}O_{2\ peak}$ were 24.8 ± 2.2 y, 179.5 ± 7.4 cm, 76.4 ± 9.4 kg and 59.7 ± 10.2 $mL.kg^{-1}.min^{-1}$, respectively. All participants were informed about the purpose of the study and any known risks associated with each procedure; they signed an informed consent form and were informed that they could withdraw from the study at any time. This study was approved by the Human Rights Committee of the University of Western Australia.

4.2.2 Familiarisation session

Each participant completed a familiarisation session prior to testing in order to become acquainted with the equipment, the exercise protocols and researchers involved in the study. During this session, maximal rate of oxygen consumption and anthropometric data (height, body mass) were also obtained.

4.2.3 Intermittent sprint protocol

The experimental protocol adopted here was similar to that described in Chapter 3, except that each participant was asked to perform four consecutive all-out 30-s cycling sprints, with 20 min of either active or passive recovery between each sprint. The two trials were administered in a randomised, counter balanced order. Before each visit, the participants were advised to maintain the regular diet both for the content and amount taken and to abstain from heavy exercise for 48 h before testing. On each testing day and before executing the intermittent sprint protocol, participants performed a 5-min warm up on a front-access cycle ergometer (Repco, NSW, Australia) which consisted of cycling at 40% $\dot{V}O_{2\ peak}$ followed by 5 min of

passive recovery. Afterwards, the participants cycled at maximal intensity for the next 30 s on the same cycle ergometer, and, after each sprint, were asked to either remain seated on the ergometer in a rested state or to actively recover by pedalling at an intensity corresponding to 40% $\dot{V}O_{2\ peak}$ until just before the next sprint, at which point they stoped cycling to enable them to initiate subsequent sprinting from a stand-still position. For each sprint, PP, MP_{30}, MP_{10}, MP_{-20} and MP_{-10} were recorded, and blood was sampled immediately before every sprint and at the start of each recovery period. It is important to stress that, as mentioned in Chapter 3, the participants were deceived with respect to the goal of this study and were informed that this study was only concerned with the effect of recovery on blood variables.

4.2.4 Sprint trial

In order to perform each 30-s sprint test, participants were asked to mount the front-access cycle ergometer (Repco, NSW), with each foot secured using a foot strap. Immediately before sprinting, each participant was asked to stand-up on the ergometer while placing both hands on the handles and remaining still. The trial was initiated with the foot of the dominant leg starting at the “two o’clock” position after a three-second count down. All participants were instructed to cycle in an “all-out” fashion without pacing themselves for 30 s. During each 30-s sprint, the following information was recorded; peak power output (PP), mean power output over 30 s (MP_{30}), mean power output over the first 6 s (MP_6) and 10 s (MP_{10}), and mean power output over the last 20 (MP_{-20}) and 10 s (MP_{-10}).

4.2.5 $\dot{V}O_{2\ peak}$ determination

$\dot{V}O_{2\ peak}$ was determined on the same cycle ergometer as that used for all experimental trials. The $\dot{V}O_{2\ peak}$ test consisted of graded exercise steps using an intermittent protocol, with one-min break between stages. The test commenced at

an intensity of 50 W and, thereafter, the intensity was increased by 30 W every 4 min until volitional exhaustion. Participants were required to maintain the set power output, which was displayed on a computer screen in front of them. The test was stopped when the participants could no longer maintain the required power output. Strong verbal encouragement was provided to each subject as they approached the end of the test. All inspired and expired air was collected through a mouthpiece connected to a Hans-Rudolf valve attached via Collins tubing to an on-line gas analysis system comprised of a Morgan Ventilation Monitor. All expired air was continuously monitored for the analysis of O_2 and CO_2 concentrations (Ametek gas analysers SOV S-3A and COV CD3A, respectively, Pittsburg, PA., USA). Data was averaged over 30-s intervals. Ventilation was also recorded every 30-s using a turbine ventilometer (Morgan, Model 096, Kent, England). The gas analysers were calibrated immediately before and verified after each test using three certified gravimetric standard-gas mixtures (BOC gases, Chatswood, Australia). The ventilometer was calibrated pre-exercise and verified post-exercise using a one-litre syringe in accordance with the manufacturer's instructions.

4.2.6 Blood Sampling

Capillary blood (120 µL) was obtained from an ear lobe. A cutaneous vasodilating cream (Finalgon®, Boehringer Ingelheim, Atarmon, Australia) was applied 10 min prior to the collection of the initial sample, and a sterile lancet (SoftTouch®, Roche Diagnostic Australia) was used to puncture the skin at the margin of the earlobe. All blood samples were analysed for pH and lactate levels with a blood-gas analyser (ABL 625 series, Radiometer, Copenhagen).

4.2.7 Statistical analyses

All data are reported as means and standard errors of the mean (± S.E.M), with the exception of the descriptive characteristics of the participants which were expressed as means ± S.D. Statistical analyses were performed using a two-way ANOVA with repeated measures. A Fisher LSD pair-wise comparison between means was adopted to compare recovery protocols. Significance was set at $p < 0.05$. All statistical analyses were conducted using the SPSS 12.0 for Windows Statistical Package (version 12.0, SPSS, Chicago, IL).

4.3 Results

4.3.1 Mean and peak power

The PP, MP_{30}, MP_{10}, MP_{-20}, and MP_{-10} of the initial 30-s sprint were not significantly different between active and passive recovery trials. In comparison to the first sprint, PP, MP_{-20}, and MP_{-10} of each of the following sprints were not significantly different for both recovery protocols (Fig. 4.1, 4.4, 4.5), and no differences were detected between active and passive recovery trials (Fig. 4.1, 4.4, 4.5). In contrast, MP_{10} and MP_{30} during the fourth sprint in the passive recovery trial were lower than during the first sprint, whereas MP_{10} and MP_{30} did not change significantly across the four sprints in the active recovery trial.

4.3.2 Lactate and pH responses

Immediately after 30 s of maximal cycling, blood lactate levels increased to a similar extent in both recovery groups, and within 5 min reached maximal levels during passive recovery, but not when active recovery was performed during that time (Fig. 4.6). During the next 15 min, blood lactate fell to lower levels during active than passive recovery, with the differences in lactate levels between protocols being maximal after 15-20 min of recovery ($p<0.05$; Fig. 4.6). In response to the subsequent sprints, the absolute increase in lactate levels after each sprint in the passive recovery group was less pronounced than after the first sprint ($p<0.05$), but the pattern of change was similar between consecutive sprints, with identical blood lactate levels attained after the second, third and fourth sprints and 20 min of recovery (Fig. 4.6). In contrast, the levels of lactate attained after the second and

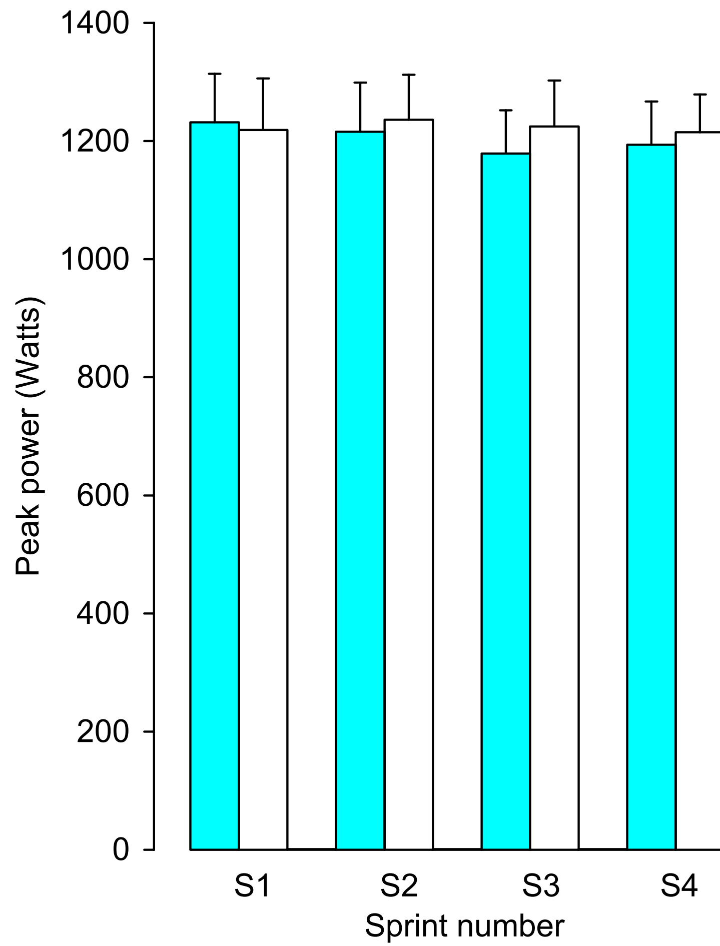

Figure 4.1: Effect of passive ( ) and active ( ) recovery between repeated 30-s sprint on peak power (PP). No significant differences between treatments (p>0.05).

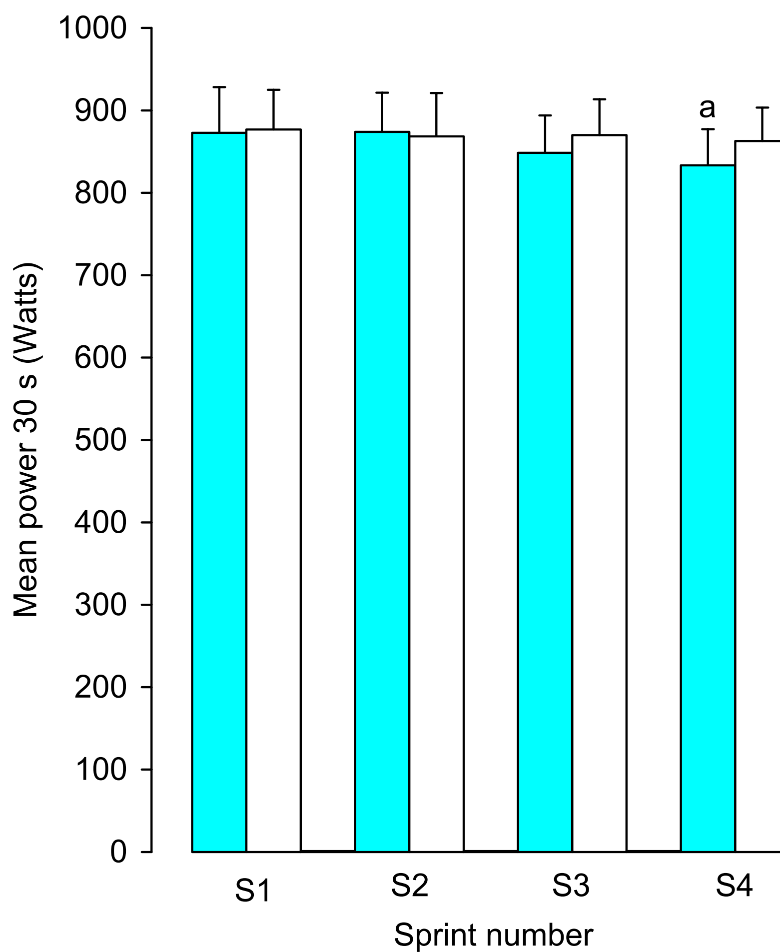

Figure 4.2: Effect of passive ( ) and active ( ) recovery between repeated 30-s sprint on mean power over 30 s (MP_{30}). a, significantly different from initial sprint ($p<0.05$).

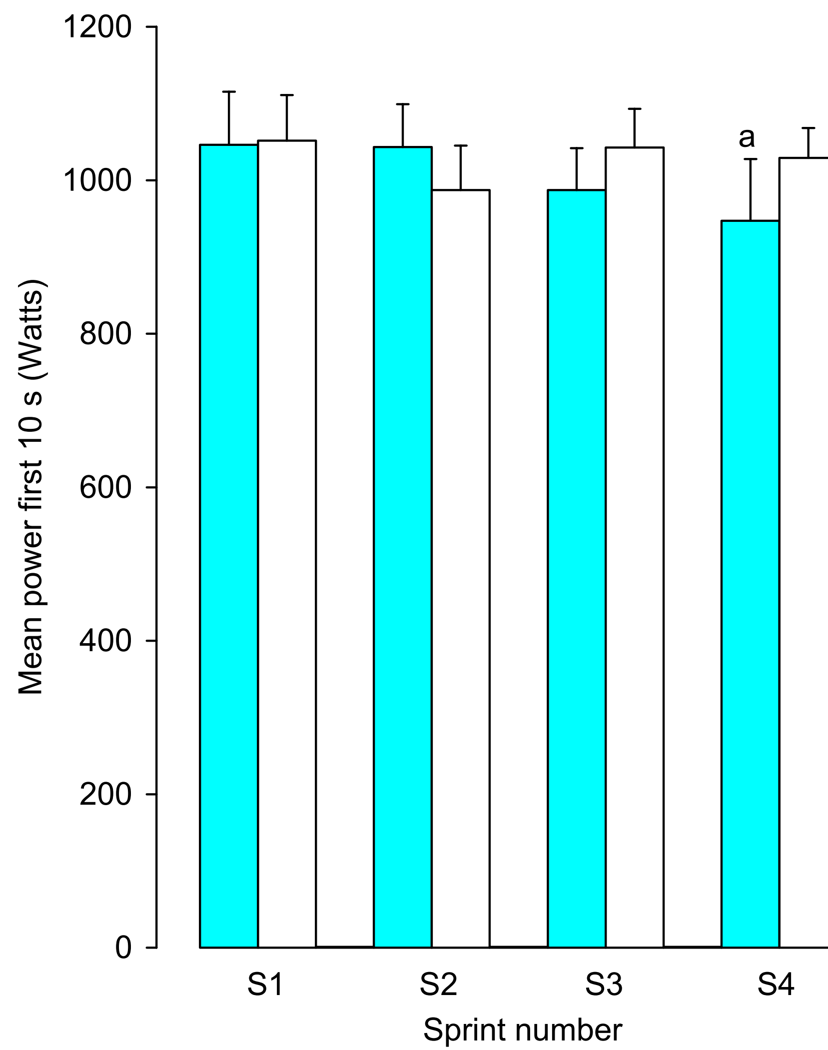

Figure 4.3: Effect of passive ( ) and active ( ) recovery between repeated 30-s sprint on mean power over first 10 s (MP_{10}). a, significantly different from initial sprint ($p<0.05$).

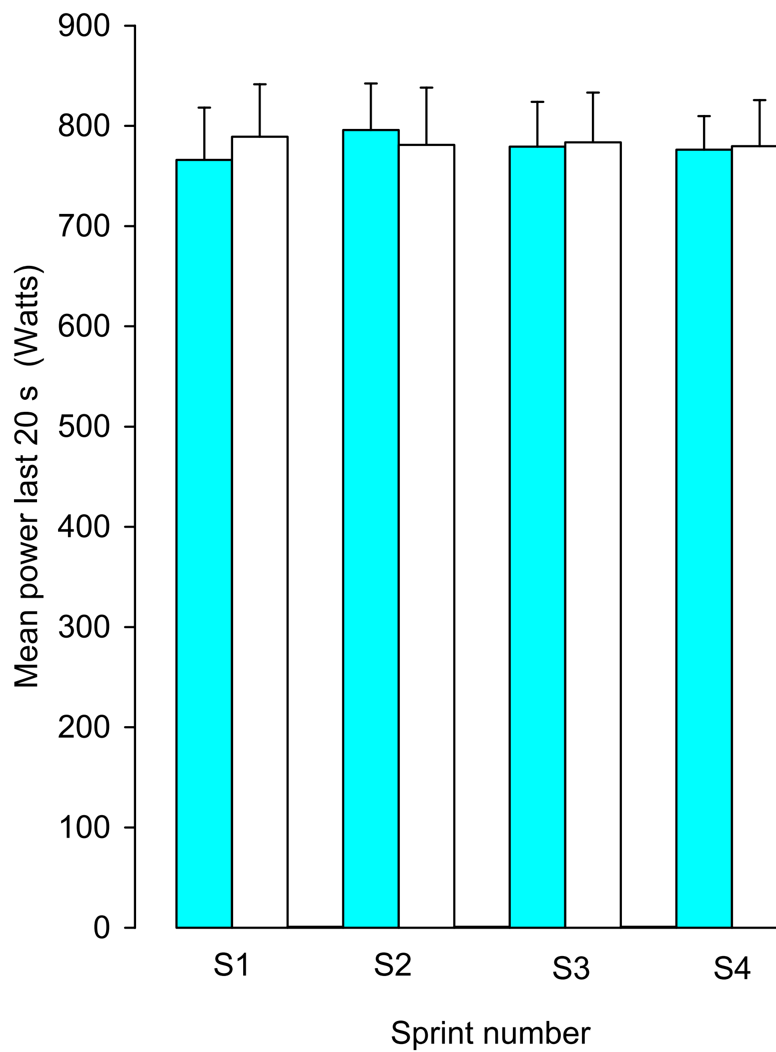

Figure 4.4: Effect of passive ( ) and active ( ) recovery between repeated 30-s sprint on mean power over last 20 s (MP_{-20}). No significant differences between treatments ($p>0.05$).

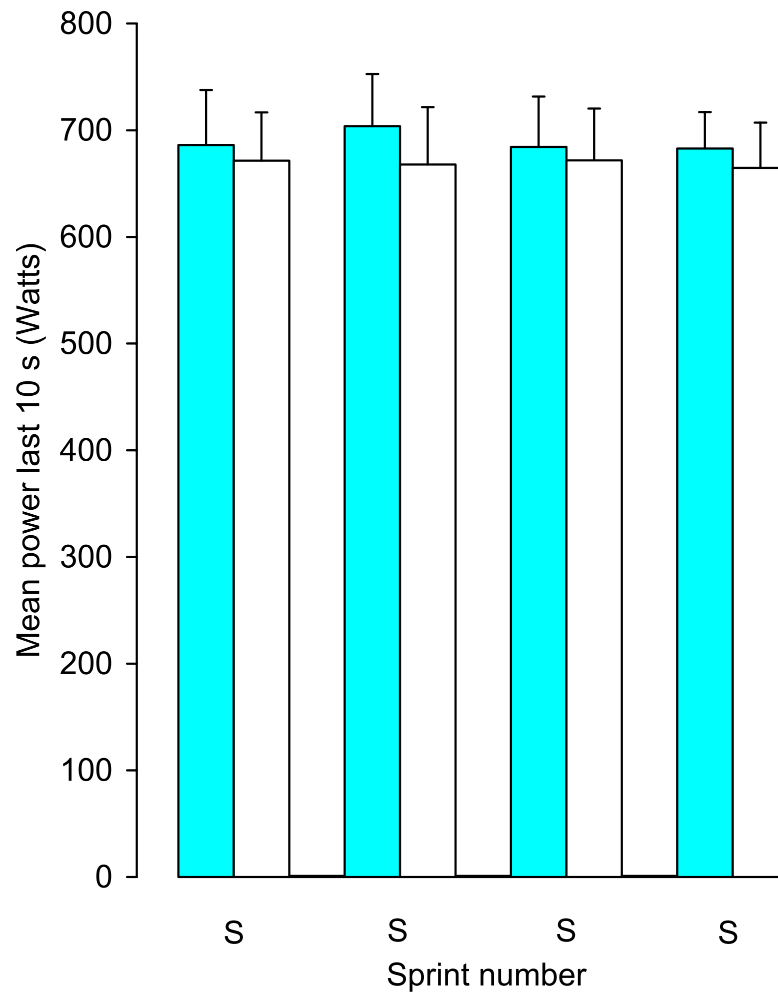

Figure 4.5: Effect of passive (■) and active (□) recovery between repeated 30-s sprint on mean power over last 10 s (MP-$_{10}$). No significant differences between treatments (p>0.05).

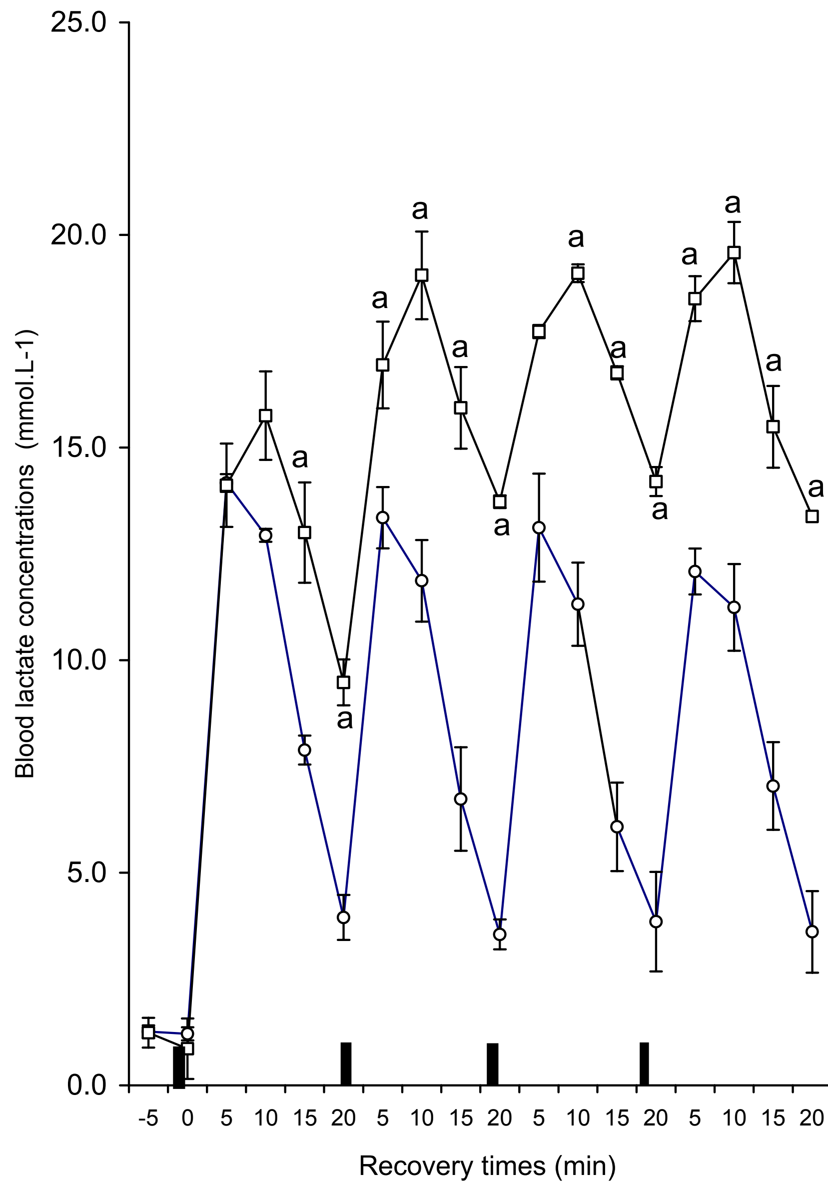

Figure 4.6: Effect of 20 min passive ( ) and active ( ) recovery after a repeated 30-s sprint on blood lactate concentrations a, significantly different between treatments (p<0.05).

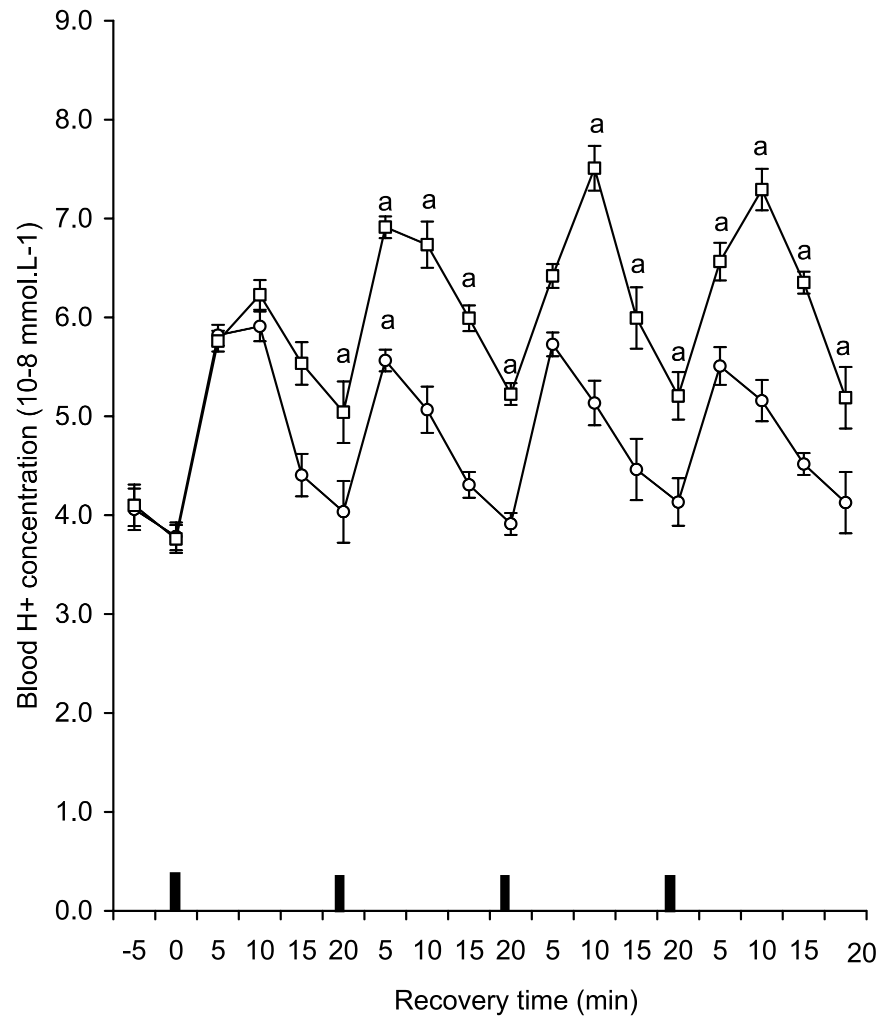

Figure 4.7: Effect of 20 min passive ( ) and active ( ) recovery after a repeated 30-s sprint on blood H^+ concentrations a, significantly different between treatments ($p<0.05$).

subsequent sprints when active recovery was performed between sprints were not different from those attained after the first sprint ($p<0.05$), but the rises in lactate levels were smaller ($P<0.05$). The differences in lactate levels between recovery protocols at the onset and end of active and passive recovery were highest immediately after the second and subsequent sprints than immediately after the first sprint ($p<0.05$; Fig. 4.6).

Immediately after 30 s of maximal cycling, blood H^+ levels increased to a similar extent in both experimental groups and reached maximal levels after 5 min of passive and active recovery (Fig. 4.7). During the next 15 min, the fall in blood H^+ levels was more pronounced during active than passive recovery ($P<0.05$), with the differences in H^+ levels being maximal after 20 min of recovery, and with H^+ levels returning to pre-exercise levels only during active recovery (Fig. 4.7). In response to each subsequent sprint, H^+ during passive recovery reached levels higher than after the first sprint ($p<0.05$), with these levels not being different between the second and subsequent sprints. After each 20-min period of passive recovery, H^+ fell to a similar extent but remained above basal pre-exercise levels (Fig. 4.7). In contrast, H^+ levels attained after the second and subsequent sprints in the active recovery group were lower than after the first sprint ($P<0.05$), and H^+ levels returned to basal pre-exercise levels after the second, third and last sprints (Fig. 4.7). The differences in H^+ levels between active and passive recovery treatments were higher immediately after the second and subsequent sprints than after the first sprint, but the differences in H^+ levels between recovery protocols after 20 min of recovery were similar across all sprints (Fig. 4.7).

4.4 Discussion

An earlier study has reported that mean power falls progressively in response to several consecutive 30-s sprints separated by 20 min of active or passive recovery, but to a lesser extent with active recovery (Thiriet et al., 1993). Given that early and late mean power during a 30-s sprint recover at different rates (Bogdanis et al., 1994) and respond differently to active recovery from a single sprint (Bogdanis et al., 1996), this raises the issue of whether this might also be the case in response to repeated sprints. For this reason, we undertook to compare the effect of 20 min of active and passive recovery on repeated sprint power output in order to evaluate whether active recovery improves sprint performance. Our results show that in response to four consecutive 30-s maximal cycling sprints, each separated by 20 min of either active or passive recovery administered in such a way as to minimise the possibility of a placebo effect, PP, MP_{-20} and MP_{-10} do not fall progressively between the first and last sprints and are not affected by active recovery. However, MP_{10} and MP_{30} decrease significantly between the first and last sprint of the passive recovery trial, but not when active recovery is performed between consecutive sprints.

Our findings thus support, in part, those of the only other study that compared the effect of 20 min of passive and active recovery on repeated sprint performance (Thiriet et al., 1993). In agreement with our findings, Thiriet and colleagues (1993) reported that the fall in mean power was more pronounced when passive rather than active recovery was performed between repeated sprints. However, our findings differ in that all indicators of sprint power examined here were similar across consecutive sprints in the active recovery trial, but not in the study of Thiriet and colleagues (1993) where mean power fell progressively. Despite their reported progressive fall in mean power, the benefit of active recovery on mean power

calculated from their results (~7%) appears to have been higher than that measured here (~3%). Arguably, meaningful comparisons between studies is complicated by the fact that the exercise protocol used by Thiriet and colleagues (1993) differed from that used here in that each exercise bout consisted of 91-105 s of exhaustive cycling at 130% $\dot{V}O_{2\ peak}$ as opposed to the 30-s maximal sprint effort adopted here. However, since the peak plasma lactate levels reported in both studies and their patterns of change were not markedly different, with approximately 17 mM in their studies as opposed to 20 mM here, this might be taken as evidence that both studies were addressing a comparable pattern of physical activity. Finally, although there is evidence that the benefit of active recovery on repeated sprint performance is much lower in trained athletes as opposed to untrained individuals (Spierer et al., 2003), this is unlikely to explain the differences between our findings and those of Thiriet and colleagues (1993) because trained athletes were involved in both studies.

Although Thiriet and colleagues (1993) were among the first to show that active recovery is beneficial to repeated sprint performance, it is important to note that their findings were not conclusive because of some major limitations with their experimental design. In particular, each sprint performed after active recovery in their study (1996) was not initiated from a stand-still position, as was the case with passive recovery. This is an important issue as this might have helped to attain peak power output in excess of what would have been possible from a stand-still position, and thus contributed to the difference in power output between active and passive recovery. It is noteworthy that this is a limitation shared by most (Signorile et al., 1993; Thiriet et al., 1993; Dorado et al., 2004), but not all, studies (Ahmaidi et al., 1996) concerned with the effect of active recovery on repeated sprint performance. More importantly, no attempt was made to minimise the possibility that a placebo

effect might account for the alleged benefit of active recovery (Thiriet et al., 1993). Indeed, most studies concerned with the effect of active recovery on single or repeated sprint performance suffer from the important limitation that the marginal improvement in sprint performance capacity following active recovery is likely to have resulted, at least in part, from a placebo effect, since they did not attempt to deceive their participants (Weltman et al., 1977, 1979; Bond et al., 1991; Signorile et al., 1993; Thiriet et al., 1993; Bogdanis et al., 1996, Felix et al., 1996; Ahmaidi et al., 1996; Donne, 2000; Connolly et al., 2003; Dupont et al., 2003; Spierer et al., 2004). As mentioned in Chapter 5, this is an important limitation considering that the subjects recruited in the study of Thiriet and colleagues (1996) and in many other studies were trained athletes, and thus highly likely to have been informed about the alleged benefits of active recovery, and thus to exhibit a placebo response. Since special care was taken to control for the aforementioned limitations, this study is the first to show that active recovery performed between repeated sprints, rather than a placebo effect, improves sprint performance. What remains to be explained, however, is the inhibitory effect of repeated sprints on MP_{10} and MP_{30} as opposed to PP, MP_{-10} and MP_{-20}.

The fall in MP_{30} between the first and fourth sprints in the passive recovery trial is unlikely to be the result of a decrease in PP or late mean power output (MP_{-10}, MP_{-20}), but is best explained the fall in early mean power. Indeed, PP, MP_{-10} and MP_{-20} in the passive recovery trial were not different between the first and fourth sprints, whereas the fall in MP_{10} (~5%) was not only more pronounced than in MP_{30}, but also of a magnitude such that it could account for most of the fall in MP_{30}. This raises the intriguing question of the mechanism whereby repeated sprints separated by passive recovery cause a fall in MP_{10} between the first and the fourth sprints, but not PP, MP_{-10} or MP_{-20}.

We tentatively propose that a faster protonation rate of Pi during the early stage of the fourth as opposed to the first sprint, followed by a more rapid fall in Pi protonation rate as the fourth sprint progresses due to increased reliance on oxidative metabolism might explain why only MP_{10} is affected by repeated sprints. This interpretation is based, in part, on the observation that blood H+ levels are higher prior to the fourth than the first sprint and on the assumption that the pattern of change in blood pH reflects that of intramuscular pH, with intramuscular pH thus expected to be lower prior to the fourth sprint in the passive recovery trial. Bearing in mind that there is growing evidence that inhibition of power output by low pH occurs indirectly via the protonation of Pi (Chapter 1), we propose that a higher proportion of the Pi released from phosphocreatine breakdown in the early stages of the fourth sprint would be expected to be protonated in comparison to first one, due to the increased H+ levels at the onset of the fourth sprint. This earlier rise in protonated Pi levels would, in turn, be predicted to reach more rapidly levels inhibiting power output (this interpretation, of course, is based on the assumption that the initial rates of phosphocreatine mobilisation are not different between the first and fourth sprints, as supported indirectly by the absence of differences in PP). Arguably, the absence of differences in power output between the later stages of the first and fourth sprints as more protonated Pi are expected to accumulate with a further fall in intramuscular pH requires some explanation.

Assuming that the lesser increase in blood lactate levels after the fourth sprint in comparison to the first one is indicative of an increased contribution of oxidative metabolism rather than a diminished capacity for lactate efflux, such an increased reliance on aerobic metabolism would be expected to be accompanied by a fall in the rate of H+ production and associated Pi protonation as sprint progresses. This would result in the eventual matching up of the rate of fatigue development during the later stages of the first and last sprint. It is important to stress that this

interpretation is further corroborated by our findings with the active recovery trial. Indeed, the similar pre sprint blood H+ levels together with the comparable magnitude of the exercise-mediated increase in blood lactate and H+ levels between the first and fourth sprints of the active recovery trial are associated with no significant differences in any of the indicators of power output between the first and fourth sprints. Arguably, the interpretation of our findings is highly conjectural and requires further experimental corroboration involving the measurements of a number of mediators of fatigue, especially given the many mediators that our explanation overlooks.

In conclusion, although many other studies have reported that active recovery improves repeated sprint performance they did not exclude the possibility of a placebo effect. This study shows for the first time that some aspects of sprint performance (MP_{10} and MP_{30}) improve even under conditions where the possibility of a placebo effect is minimised. This study also shows for the first time that the fall in mean power associated with repeated 30-s sprints results primarily from a fall in early as opposed to either late power output or peak power. This highlights the merits of breaking down power output into its early and late components rather than focusing solely on peak power or total mean power as is the case in many studies concerned with sprint performance. Arguably, more research is required to elucidate the molecular mechanism underlying the fatigue of early power output in repeated sprints.

CHAPTER 5

General discussion

5.1 General discussion

Many sporting events require athletes to engage in all-out sprint effort. In the most extreme cases, such as in short-distance running, swimming, track cycling or skating, success is determined ultimately by one's ability to perform well against time. Arguably, any preparatory strategies conducive to optimising all-out sprint performance in those events are of paramount importance to sprint athletes. For this reason, a sizable amount of research work has been devoted towards identifying the best "warm-up" practice to not only minimise the possibility of injuries, but also to enhance performance. In particular, the effectiveness of prior exercise (PE) as a means to warm-up before sprinting has received its share of research effort.

Despite the aforementioned interest in PE, the bulk of the research in this area has focused mainly on determining across a broad range of exercise tasks whether PE is beneficial to sprint performance (Bishop 2003a,b). Only a few investigators have attempted to determine how long and intense should PE be, and how much rest should be given between PE and a subsequent sprint to optimise performance. Also, only a limited number of studies have attempted to elucidate the mechanisms whereby PE improves sprint performance. Although these studies have provided evidence that both increasing muscle temperature and mobilising the cardiorespiratory system prior to exercise contribute largely to the benefit of PE on sprint performance, a number of questions remain without answers. In particular, as mentioned above, the response of sprint performance to PE duration and to the time elapsed between PE and sprinting have received little attention. Moreover, the relative importance of pre-sprint muscle temperature and cardiorespiratory mobilisation to sprint performance after PE is unknown. It also remains to be determined if the relative contribution of these mechanisms is stable or changes throughout a sprint. This latter question is an important one, since most research

has focused on time trials, peak power, total work or time to fatigue as indicators of sprint performance rather than breaking a sprint into its early and later power components. Another important question relates to situations where an athlete has to engage in a sprint shortly after one or several earlier sprints. Under these conditions, what is the most effective preparatory strategy to adopt prior to sprinting when performed after a previous sprint? Is it better to rest and face the risk of "cooling down" or to exercise lightly throughout recovery to optimise subsequent sprint performance when recovery is of a short duration? It was the primary aim of this thesis to address the aforementioned questions.

As mentioned above, little is known about the effect of both PE duration and of the inclusion of a rest period between PE and sprinting on sprint performance. Also, although the benefits of PE on the power output during an all-out sprint have been attributed primarily to either temperature-related mechanisms or to a mobilisation of the cardiorespiratory system, it is unclear which of these mechanisms contributes the most to the effect of PE on sprint performance. Given the evidence that muscle temperature is likely to preferentially affect early power output (Chapter 1), we hypothesised that, in response to PE, the increase in muscle and core temperatures will contribute the most to improving early power output during an all-out 30-s sprint, whereas the mobilisation of the cardiorespiratory system will play a more important role later in the sprint. In order to test this hypothesis, we compared the responses of a 30-s sprint to different PE protocols designed to control for either muscle temperature or pre-exercise $\dot{V}O_2$. A group of cyclists and triathletes performed either a 4-min PE and 10-min rest (PE_{4R}), 4-min PE (PE_4) or 20-min PE (PE_{20}) immediately before performing a 30-s sprint, with sprint performance evaluated by measuring PP, MP_{30}, MP_6, $MP_{10,}$ $MP_{-20,}$ and $MP_{-10.}$ We found that core and muscle temperatures increased significantly in response to PE_{20}, but were not significantly

affected by PE_4 and PE_{4R}, thus allowing us to isolate the effect of pre-sprint $\dot{V}O_2$. On the other hand, $\dot{V}O_2$ prior to sprinting increased to a similar extent in response to PE_{20} and PE_4, but not to PE_{4R}. Our findings in Chapter 2 show that MP_{30} was similar for PE_{20} and PE_4, but lowest after PE_{4R}. On the other hand, PP, MP_6 and MP_{10} were significantly greater after PE_{20} than after PE_{4R} or PE_4, but PP was not different between the latter two PE protocols. In contrast, MP_{-10} and MP_{-20} were higher after PE_4 than after $PE_{20,}$ and lowest after PE_{4R}.

The higher PP and MP_6 and MP_{10} when only pre-sprint muscle temperature is increased, and the absence of improvement in PP together with the small rise in early mean power output (MP_6 and MP_{10}) when pre-sprint $\dot{V}O_2$ is increased without changing muscle temperature suggest that: (a) increasing muscle temperature offers a better means than mobilising the cardiorespiratory system to improve peak power, and (b) both pre-sprint $\dot{V}O_2$ and muscle temperature enhance early mean power (MP_6 and MP_{10}). However, since increasing muscle temperature alone is detrimental to mean power later during a sprint (MP_{-20} and MP_{-10}), whereas increasing $\dot{V}O_2$ independently of muscle temperature enhances power output to the greatest extent during that time, this suggests that the benefit of mobilising the cardiorespiratory system is greatest in the later stage (>10 s) of a 30-s maximal sprint effort.

Overall, these findings show for the first time that there is a temporal transition in the mechanisms responsible for the increase in power output in response to PE performed immediately before a 30-s maximal sprint effort, with increased muscle temperature playing the most important role at the onset of exercise and the mobilisation of the cardiovascular system later (>10 s). On practical grounds, our findings suggest that prolonged PE (20 min) performed immediately before sprinting is better than short PE (4 min), at enhancing peak power and mean power output

during a short (< 10 s) maximal sprint effort such as a 100 m sprint or events requiring short bursts of maximal power. This is probably because prolonged PE increases muscle temperature and mobilises the cardiovascular system as suggested by our findings in Chapter 2. On the other hand PE durations of 4-20 min have similar overall effects on all-out sprint events lasting 30 s performed immediately after PE.. It is important to stress, however, that the length of time elapsed between PE and subsequent sprint performance should be short enough to prevent a significant fall in pre-sprint $\dot{V}O_2$ in order to achieve optimal mean power. Unfortunately, increasing the mobilisation of the cardiorespiratory system prior to sprinting might be difficult if not impossible to achieve in comparison to increasing muscle temperature in most sporting events, since several minutes might elapse between the end of PE and the beginning of a competitive all-out sprint. Also, it remains to be established the extent to which our findings on all-out sprint cycling performance extend to other activities such as running or swimming , to events where pacing is required or for intense physical activities lasting longer than 30 s. It must be stressed that the primary purpose of this study was to elucidate the physiological mechanisms underlying the effect of PE on sprint performance rather than to developing practical PE practices for sprint performance across sporting events.

Given that 20 min of PE performed immediately before a sprint improves sprint power, this might be taken as evidence that this type of exercise rather than a rest should be advocated if one is required to perform a sprint shortly after a previous one. In other words, our results raise the question of whether mild physical activity (< 40% $\dot{V}O_{2peak}$), or active recovery, performed between two consecutive sprints is preferable to a rest for the optimisation of sprint performance. Traditionally, active recovery as means to improve subsequent sprint performance has been advocated

on the grounds that this recovery mode accelerates the return of blood pH and lactate to basal pre-exercise levels, with low pH alleged to be a mediator of muscular fatigue. Our findings in Chapter 2 raise the possibility that being active between two consecutive sprints might have the additional advantage of maintaining elevated muscle and core temperature as well as mobilising the cardiorespiratory system prior to the second sprint, thus acting as a “warm-up”. Despite these indirect evidence suggesting that the best preparatory strategy to adopt prior to engaging in the second of two consecutive sprints is to actively recover as opposed to rest between sprints, it is unclear whether active recovery can improve recovery of sprint performance capacity. Indeed, as discussed in Chapter 1, most studies have reported that the impact of active recovery is either small (< 3%) or undetectable. We hypothesised that these conflicting results might be due in part (a) to a failure to control for placebo effect and (b) to the efficacy of this recovery protocol being compared at times close to full recovery (14-20 min).

In order to address these hypotheses and compare active and passive recovery when recovery is incomplete, we had to establish the time dependency of the recovery of several indicators of sprint performance including, PP, MP_{30}, MP_{+10}, MP_{-20} and MP_{-10}. To this end, a group of exercise-trained male athletes performed two 30-s sprints on a cycle ergometer, each separated by a recovery period of either 0, 1, 2, 5, 10, 15, 20 or 40 min. Based on the results of this experiment, passive and active recovery performed at an intensity of 40% $\dot{V}O_{2peak}$ were then compared when recovery was either 45-75% complete (4 min) or near complete (20 min), with great care taken to minimise the possibility of a placebo effect. We found that, although active recovery resulted in a faster return of blood lactate and pH to pre-exercise levels, it had no effect on the recovery of any of the indicators of sprint performance examined here, irrespective of recovery time. It is important to note that this absence of improvement, particularly after 20 min of recovery, also occurred despite

the possibility of higher cardiorespiratory mobilisation and increased muscle and core temperatures after active compared to passive recovery. On this basis, we concluded that although active recovery between two sprints speeds up the rate of H^+ disposal, it has no effect on early or late sprint power in exercise-trained individuals. Unfortunately, $\dot{V}O_2$ and temperature were not measured in this study, but if it were to be the case that, after 20 min of active recovery, both cardiorespiratory mobilisation and muscle/core temperature are elevated in comparison to passive recovery, the absence of difference in sprint performance between those recovery modes would suggest that sprint performance after active recovery is impaired to some extent.

Our findings also raise the possibility that a placebo effect might explain the results of those studies that have reported a marginal improvement in sprint performance capacity in response to active recovery (<3%). This is an important limitation shared by most studies on active recovery, especially considering that most have been performed on recreational athletes who might have been aware of the alleged benefits of this mode of recovery. In order to minimise the possibility of a placebo effect in the study described in Chapter 3, our participants were deceived by being led to believe that we were only concerned with the effect of active recovery on blood variables. There is a need in future studies to evaluate the magnitude of such a placebo effect on sprint performance in order to determine whether it has the potential to be large enough to explain the levels of alleged benefit of active recovery reported by others.

Although we have shown in Chapter 3 that in response to passive and active recovery, sprint power capacity recovers completely within 20 min despite marked differences in plasma H^+ levels between recovery protocols, there is evidence that mean power falls in response to several consecutive 30-second sprints each

separated by 20 min of active or passive recovery, but to a lesser extent with active recovery (Thiriet et al., 1993). Given the evidence from both Chapter 3 and the work of others (Bogdanis et al., 1996) that early and late mean power during a 30-s sprint recover at different rates (Bogdanis et al., 1994) and respond differently to active recovery from a single sprint (Bogdanis et al., 1996), this raises the question of whether this might also be the case in response to repeated sprints, an issue that has never been examined before.

In order to test the hypothesis that the early and late mean power output of each of repeated 30-s sprints respond differently to active and passive recovery, a group of trained athletes was subjected to four consecutive bouts of 30-second sprint, each separated by 20 min of either active recovery at 40% $\dot{V}O_2$ peak or passive recovery. Our results show that PP, MP_{-20} and MP_{-10} did not fall between the first and last sprints, and were not affected by active recovery. In contrast, we found that MP_{10} and MP_{30} decrease significantly between the first and last sprint of the passive recovery trial, but not when active recovery is performed between consecutive sprints. Finally, this study also showed that the fall in mean power associated with repeated 30-s sprints in the passive recovery trial results primarily from a fall in early, but not late power output. As discussed in Chapter 4, we propose that a faster protonation rate of Pi during the early stage of the fourth as opposed to the first sprint, followed by a more rapid fall in Pi protonation rate as the fourth sprint progresses due to increased reliance on oxidative metabolism might explain why only early mean power output is affected by repeated sprints.

5.2 Conclusions and direction for future research

In conclusion, this thesis shows that:

There is a temporal shift in the mechanisms responsible for the effect of PE on power output during a maximal sprint effort, with temperature-dependent mechanisms playing a more important role at the onset of the sprint and mobilisation of the cardiorespiratory system playing a more important role later.

On practical grounds, our findings suggest that in order to improve sprint performance, PE must be long enough to increase muscle temperature, and the recovery period between PE and the sprint must be brief in order to maintain $\dot{V}O_2$ above basal levels. Also, a prolonged PE (20 min) aimed at increasing both muscle temperature and $\dot{V}O_2$ better enhances best peak power and mean power output during a short (< 10 s) maximal sprint effort, whereas PE duration of 4-20 min have similar overall effects on a 30–s sprint.

Active recovery after a 30-s sprint does not improve recovery of sprint performance capacity compared to passive recovery even when both protocols are compared at a time when recovery is partial.

Our results support the view that a placebo effect might explain the findings of previous studies that have reported that active recovery improves recovery of exercise performance capacity after a sprint.

Although several studies have reported that active recovery improves repeated sprint performance, this is the first one to show that this is the case even under conditions where the possibility of a placebo effect is minimised.

The fall in mean power associated with repeated 30-s sprints in interspersed by 20-min recovery periods results primarily from a fall in early as opposed to either late power output or peak power.

The findings of Chapters 2, 3 and 4 highlight the merits of breaking down power output into its early and late components rather than focusing solely on peak power or total mean power as is the case in many studies concerned with sprint performance.

Several novel questions have arisen from our work. Our findings in Chapter 2 highlight the importance of carrying invasive work in the future to (1) corroborate our interpretation that the temperature effects of PE are mediated primarily via activation of non-oxidative metabolism, and (b) elucidate how the mobilisation of the cardiorespiratory system improves mainly late power output. Also, there is a need to determine the relative extent to which core and muscle temperatures determine the benefit of prolonged PE on peak power. Our work in Chapters 3 and 4 also raises a few important issues. There is a need to examine the extent to which a placebo effect can affect sprint performance. Also, our hyphothesis in Chapter 3 and 4 that initiating a maximal sprint effort from a stand-still position will result in a lower power output than if initiated while already cycling requires corroboration. Finally, more research is needed to determine the molecular mechanisms underlying our findings in Chapter 4. In particular, the mechanisms and mediators of fatigue alleged in Chapter 4 to be at the origin of the fatigue that takes place in response to repeated sprints remain to be elucidated.

References

REFERENCES:

Adams, G. R., Foley, J. M. and Meyer, R. A., (1990). Muscle buffer capacity estimated from pH changes during rest-to-work transitions. *Journal of Applied Physiology. 69(3): 968-972.*

Ahmaidi, S., Granier, P., Taoutaou, Z., Mercier, J., Dubouchaud, H. and Prefaut, C. (1996). Effects of active recovery on plasma lactate and anaerobic power following repeated intensive exercise. *Medicine and Science in Sports and Exercise.* 28 (4): 450-456.

Ainsworth, B. E., Serfas, R. C. & Leon, A. S. (1993). Effects of recovery duration and blood lactate level on power output during cycling. *Canadian Journal of Applied Physiology,* 18(1): 19-30.

Allen, D. G., Westerblad, H., Lee, J. A. and Lannergren, J. (1992). Role of excitation-contraction coupling in muscle fatigue. *Sports Medicine. 13: 116-126.*

Andzel, W. D., (1982). One mile run performance as a function of prior exercise. *Journal of Sports Medicine. 22 : 80-83.*

Armstrong, A. (1997). The effects of active recovery on the resynthesis of muscle glycogen following short-term continuous high intensity exercise. *Honours Thesis, Department of Human Movement, University of Western Australia.*

Asmussen, E. and Boje, O. (1945). Body temperature and capacity for work. *Acta Physiologica Scandinavia. 10: 1-22.*

Asmussen, E. and Mazin, B. (1978). A central nervous component in local muscle fatigue. *European Journal of Applied Physiology. 38: 9-15.*

Astrand, P. O., Hultman, E., Juhlin-Danfelt, A. and Reynold, G. (1986). Disposal of lactate during and after strenous exercise in humans. *Journal of Applied Physiology 61(1): 338-343.*

Atkinson, G., Todd, C., Reilly, T. P. and Waterhouse, J. M. (1999). Effect of time of day and warm-up on a cycling time trial. *4th annual Congress of European College of Sport Science, 14-17 July.*

Ball, K. L., Johnson, M. D., Solaro, R. J. (1994). Isoform specific interactions of troponin I and troponin C determine pH sensitivity of myofibrillar Ca^{2+} activation. *Biochemistry 33: 8464-8471.*

Balsom, P. D., Gaitanos, G. C., Ekblom, B. and Sjodin, B. (1994). Reduced oxygen availability during high intensity intermittent exercise impairs performance. *Acta Physiologica Scandinavica 1994, 152: 279-285.*

Balsom, P. D., Seger, J. Y., Sjodin, B., Ekblom, B. (1992). Maximal-intensity intermittent exercise: Effect of recovery duration. *International Journal of Sports Medicine, 13(7): 528-533, 1992.*

Bangsbo, J., Krustrup, P., Gonzalez-Alonso, J. and Saltin, B. (2000). Muscle oxygen kinetics at onset of intense dynamic exercise in humans. *American Journal of Physiology. 279: R899-R906.*

Barcroft, H. and Edholm, O.G. (1943). The effect of temperature on blood flow and deep temperature in the human forearm. *Journal of Physiology. 102: 5-12.*

Barcroft J., King W. R.(1909). The effect of temperature on the dissociation curve of blood. *Journal of Physiology. 39: 374-384.*

Barstow, T. J., Lamarra, N. and Whipp, B. J. (1990). Modulation of muscle and pulmonary O_2 uptakes by circulatory dynamics during exercise. *Journal of Applied Physiology. 68(3): 979-989.*

Bearden, S. E. and Moffat, R. J. (2001). VO_2 and heart rate kinetics in cycling: transitions from an elevated baseline. *Journal of Applied Physiology. 90: 2081-2087.*

Behm D. G.,and St-Pierre D. M. M. (1997). Effect of fatigue duration and muscle type on voluntary and evoked contractile properties. *Journal of Applied Physiology. 82(5): 1654-1661.*

Behnke, B. J., Kindig, C. A., Musch, T. I., Sexton, W. L. and Poole, D. C. (2002): Effect of prior contraction on muscle microvascular oxygen pressure at onset of subsequent contraction. *Journal of Physiology. 539: 927-934.*

Belcastro, A. N. and Bonen, A. (1975). Lactic acid removal rates during controlled and uncontrolled recovery exercise. *Journal of Applied Physiology. 39* (6): 932-936.

Bergh, U. and Ekblom, B. (1979). Influence of muscle temperature on maximal muscle strength and power output in Human Skeletal muscles. *Acta Physiologica Scandinavia. 107: 33-37.*

Bergstrom, M. and Hultman, E. (1991). Relation and force during fatigue and recovery of human quadriceps muscles: relations to metabolite changes. *Pfluegers Archives. 418: 153-160.*

Bevan, L., Laouris, Y., Reinking, R. M. and Stuart, D. G. (1992). The effects of stimulation pattern on the fatigue of single motor units in adult cats. *Journal of Physiology, London. 449: 85-108.*

Bigland-Ritchie, B. (1984). Muscle fatigue and the influence of changing neural drive. *Clinics in Chest Medicine. 5: 21-34.*

Bigland-Ritchie, B., Furbush, F. and Woods, J. J. (1986). Fatigue of intermittent submaximal voluntary contractions: central and peripheral factors. *Journal of Applied Physiology. 61: 421-429.*

Bigland-Ritchie, B. Jones, D. A., Hoskings, J. P. and Edwards, R. H.(1978). Central and peripheral fatigue in sustained maximum voluntary contractions on human quadriceps muscle. *Clinical Science and Molecular Medicine. 64(2): 401-413.*

Bigland-Ritchie, B., (1981). EMG and fatigue of human voluntary and stimulated contraction. In: *Human Muscle Fatigue: Physiological Mechanism*, edited by Portel, R. and Whelan, J., *London: Pitman Medical: 130-156.*

Bigland-Ritchie, B., Bellemare, F. and Woods, J.J. (1986). Excitation frequencies and site of fatigue. In: *Human Muscle Power*, edited by N. L. Jones, N. MacCartney and A.J. McComas. Champaign, Ill: Human Kinetics*: 197-214*.

Bigland-Ritchie, B., Dawson, N.J., Johansson, R.S. and Lipold, O.C.J. (1986). Reflex origin for the slowing of motoneurons firing rates in fatigue of human voluntary contractions. *Journal of Physiology, London. 379: 451-459.*

Bigland-Ritchie, B., Johansson, R. and Woods, J. J. (1983). Does a reduction in motor drive is necessarily result in force loss during fatigue? In: *International series of Sports Sciences. Biochemistry of exercise,* Edited by Knuttgen, H.G., Vogel, J.A. and Poortmans, J. Champaign, IL: Human Kinetics, *13: 864-870.*

Bigland-Ritchie, B., Johansson, R., Lippold, O. C., Smith, S. and Woods, J. J. (1983). Changes in motoneuron firing rates during sustained maximal voluntary contractions. *Journal of Physiology. 340 : 335-346.*

Bigland-Ritchie, B., Kukulka, C. G., Lipold, O. C. and Woods, J J. (1982). The absence of neuromuscular transmission failure in sustained maximal voluntary contractions. *Journal of Physiology. 330: 265-278.*

Bishop, D. Bonetti, D. and Spencer, M. (2003). The effect of an intermittent, high-intensity warm-up on supramaximal kayak ergometer performance. *Journal of Sports Sciences. 21: 13-20.*

Bishop, D., Spencer, M., Duffield, R. and Lawrence, S. (2001). The validity of a repeated sprint ability test. *Journal of Science and Medicine in Sport. 4 (1): 19-29.*

Bishop, D. (2003). Warm-Up I: Potential mechanism and the effects of passive warm-up on exercise performance. *Sports Medicine. 33(6): 439-454.*

Bishop, D. (2003). Warm-up II: Performance changes following active warm up and how to structure the warm up. *Sport Medicine. 33 (7): 483-98.*

Blanchard, E. M., Pan, B. S. and Solaro, R. J. (1984). The effect of acidic pH on the ATPase activity and troponin Ca^{2+} binding of rabbit skeletal myofilaments. *Journal of Biology and Chemistry. 259: 3181-3186.*

Blazev, R. and Lamb, G. D. (1999). Low [ATP] and elevated [Mg^{2+}] reduce depolarization-induced Ca^{2+} release in rat skinned skeletal muscle fibres. *Journal of Physiology. 520: 203-215.*

Blinks, J. R., Rudel, R. and Taylor, S. R. (1978). Calcium transient in isolated amphibians skeletal muscle fibres: detection with aequorin. *Journal of Physiology (London). 277: 291-323.*

Bogdanis, G. C., Nevill, M. E., Boobis, L. H. and Lakomy, H. K. A. (1996). Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *Journal of Physiology. 80(3): 876-884.*

Bogdanis, G. C., Nevill, M. E., Lakomy, H. K. A. (1994). Effect of previous dynamic arm exercise on power output during repeated maximal sprint cycling. *Journal of Sport Science. 12: 363-370.*

Bogdanis, G. C., Nevill, M. E., Lakomy, H. K. A., Graham, C. M. and Louis G. (1996). Effect of active recovery on power output during repeated maximal sprint cycling. *Journal Applied Physiology. 74:461-469.*

Bogdanis, G. C., Nevill, M. E., Lakomy, H. K. and Boobis, L. H. (1998). Power output and muscle metabolism during and following recovery from 10-20 s of maximal sprints in humans. *Acta Physiologica Scandinavica. 163: 261-272.*

Bogdanis, G. C., Neville, M. E., Boobis, L. H. and Lakomy, H. K. (1995). Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *Journal of Physiology. 482(2): 467- 480.*

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

Bonen, A. and Belcastro, A. N. (1976). Comparison of self selected recovery Methods: Lactic acid removal rates. *Medicine and Science in Sports. 8(3): 176-178.*

Bonen, A., Ness, G. W., Belcastro, A. N. and Kirby, R. L. (1985). Mild exercise impedes glycogen repletion in muscle. *Journal of Applied Physiology. 58(5):1622-1629.*

Bonhert, B., Ward, S. A. and Whipp, B. J. (1998). Effect of prior arm exercise on pulmonary gas exchange kinetics during high-intensity leg exercise in humans. *Experimental Physiology. 83: 557-570.*

Boobis, A. R., Murray, S., Seddon, C. E. and Davies, D. S. (1987). In vitro studies of induction and inhibition of drug oxidation in man. *Pharmacology and Therapeutics. 33(1): 101-108.*

Bouissou, P., Defer, G., Guezennec, C. Y., Estrade, P. Y. and Serrurier, B. (1988). Metabolic and blood catecholamine responses to exercise during alkalosis. *Medicine and Science in Sports and Exercise. 20(3): 228-232.*

Brooks, G. A. (1986). The lactate shuttle during exercise and recovery. *Medicine and Science in Sports and Exercise. 18(3): 360-368*

Brooks, G. A., Hittelman, K. J., Faulkner, J. A. and Beyer, R. F. (1971). Temperature, skeletal muscle mitochondrial functions, and oxygen debt. *American Journal of Physiology. 220(4): 1053-1059.*

Brum, G. and Rios, E. (1987). Intramembrane charge movement in frog skeletal muscle fibres. Properties of charge 2. *Journal of Physiology, London. 387: 489-517.*

Brum, G., Fitts, R., Pizarro, G. and Rios, E. (1988). Voltage sensors of the frog skeletal muscle membrane require calcium to function in excitation-contraction coupling. *Journal of Physiology, London. 398: 475-505.*

Bucthal, F., Kaiser, E. and Knappeis, G.G. (1944). Elasticity, viscosity and plasticity in the cross striated muscle fibre. *Acta Physiologica Scandinavica*. 8: 16-37.

Burnley, M., Doust, J. H., Ball, D. and Jones, A. M. (2002). Effect of prior heavy exercise on VO_2 kinetics during heavy exercise are related to changes in muscle activity. *Journal of Applied Physiology. 93: 167-174.*

Burnley, M., Doust, J. H., Carter, H. and Jones, A. M. (2001). Effect of prior exercise and recovery duration on oxygen uptake kinetics during heavy exercise in humans. *Experimental Physiology. 86 (3): 417-425.*

Burnley, M., Doust, J. H., Jones, A. M. (2002). Effects of prior heavy exercise, prior sprint exercise and passive warming on oxygen uptake kinetics during heavy exercise in humans. *European Journal of Applied Physiology. 87: 424-32.*

Burnley, M., Doust, J. H. and Jones, A. M. (2005). Effect of prior warm-up regime on severe cycling performance. *Medicine and Science in Sports and Exercise. 37(5): 838-845.*

Burnley, M., Doust, J. H. and Jones, A. M. (2002). Effects of prior warming and sprint exercise on oxygen uptake kinetics during heavy exercise in humans. *European Journal of Applied Physiology. 87: 424- 432.*

Burnley, M., Jones, A. M., Carter, H., Doust, J. H. (2000). Effects of prior exercise on phase II pulmonary oxygen uptake kinetics during exercise. *Journal of Applied Physiology. 89: 1387-1396.*

Byrd, S. K., McCutcheon, L. J., Hodgson, D. R. and Gollnick, P.D. (1989). Altered Sarcoplasmic reticulum function after high intensity exercise. *Journal of Applied Physiology 67: 2072-2077.*

Campbell-O'Sullivan, S. P., Constantin-Teodosiu, D, Pierce, N. and Greenhaff, P. L. (2002). Low intensity exercise in humans accelerates mitochondrial ATP production and pulmonary oxygen kinetics during subsequent more intense exercise. *Journal of Physiology. 583: 931-939.*

Carlile, F. (1956). Effect of preliminary passive warming on swimming performance. *Research Quarterly for Exercise and Sport.* 27(2): 143-151.

Casey, A., Short, A. H., Curtis, S. and Greenhaff, P. L. (1996). The effect of glycogen availability on power output and metabolic response to repeated bouts of maximal isokinetic exercise in man. *European Journal of Applied Physiology 72: 248-255.*

Chase, P. B. and Kushmerick, M. J. (1995). Effects of physiological ADP concentrations on contraction of single skinned fibres from rabbit fast and slow muscles. *American Journal of Physiology. 268: C480-C490.*

Chase, P. B. and Kushmerick, M. J. (1988). Effects of pH on contraction of rabbit fast and slow skeletal muscle fibres. *Biophysiology Journal. 53: 935-946.*

Chasiotis, D., Sahlin, K. and Hultman, E. (1982). Regulation of glycogenolysis in human muscle at rest and during exercise. *Journal of Applied Physiology. 53: 708-715.*

Choi, D., Cole, K. J., Goodfaster, B. H., Fink, W. and Costill, D. L. (1994). Effects of passive and active recovery on the resynthesis of muscle glycogen. *Medicine in Science and Sports exercise 26(8): 992-996.*

Chooke, R., Franks, K., Luciani, G. B. and Pate, E. (1998). The inhibition of rabbit skeletal muscle contraction by hydrogen ions and phosphate. *Journal of Physiology (London). 395: 77-97.*

Clamann, H. P. and Broecker, K. T. (1979). Relation between force and fatiguability of red and pale skeletal muscles in man. *American Journal of Physical Medicine. 58: 70-85.*

Connoly, D. A. L., Brennan, K. M. and Lauzon, C. D. (2003). Effects of active versus passive recovery on power output during repeated bouts of short term, high intensity exercise. *Journal of Sports science and Medicine. 2: 47-51.*

Conley, K. E., Blei, M. L., Richards, T. L., Kushmerick, M. J. and Jubrias, S. A. (1999). Activation of glycolysis in human muscle in vivo. *American Journal of Physiology. 273: C306-C315.*

Cooke, R. and Pate, E. (1985). The effects of ADP and phosphate on the contraction of muscle fiber. *Biophysiology Journal. 48: 789-798.*

Cooke, R. and Pate, E. (1988). Addition of phosphate to active muscle fibres probes actomyosin states with the powerstroke. *Pfluegers Archives. 414: 73-81.*

Cooke, R. and Pate, E. (1989). Addition of phosphate to active muscle fibers probes actomyosin states within the power-stroke. *Pflugers Archives. 414: 73-81.*

Costill, D.L., Barnett, A., Sharp, R., Fink, W. J. and Katz, A. (1983). Leg muscle pH following sprint running. *Medicine and Science in Sports and Exercise. 15: 325-329.*

Coupland, M. E., Puchert, E. and Ranatunga, K. W. (2001). Temperature dependence of active tension in mammalian (rabbit psoas) muscle fibres: Effect of inorganic phosphate. *Journal of Physiology (London). 536: 879-891.*

Crow, M. T. and Kushmerick, M. J. (1982). Chemical energetics of slow and fast twitch muscle of mouse. *Journal of General Physiology. 79: 147-166.*

Dantzig, J. A., Goldman, Y. E., Millar, N. C., Lacktis, J. and Homsher, E. (1992). Reversal of the cross-bridge force-generating transition by photogeneration of phosphate in rabbit psoas muscle fibres. *Journal of Physiology (London). 451: 247-278.*

Dantzig, J. A., Hibberd, M. G., Trentham, D. R. and Goldman, Y. E. (1991). Cross-bridge kinetics in the presence of MgADP investigated by photolysis of caged ATP in rabbit psoas muscle fibres. *Journal of Physiology 432: 639-680.*

Dawson, M. J. (1988). The relation between muscle contraction and metabolism: studies by 31P nuclear magnetic resonance spectroscopy. *Advances in Experimental Medical Biology. 226: 433-448.*

Dawson, M. J., Gadian, D. G. and Wilkie, D. R. (1977). Contraction and recovery of living muscles studied by 31P nuclear magnetic resonance. *Journal of Physiology. 267: 703-735.*

Dawson, M. J., Gadian, D. G. and Wilkie, D. R. (1978). Muscular fatigue investigated by phosphorus nuclear magnetic resonance. *Nature. 274: 861-866.*

Dawson, M. J., Gadian, D. G. and Wilkie, D. R. (1980). Mechanical relaxation rate and metabolism studied in fatiguing muscle by phosphorus nuclear magnetic resonance. *Journal of Physiology. 299: 465-484.*

Dawson, M. J., Gadian, D. G. and Wilkie, D. R. (1986). The [H_2PO_4] may determine cross-bridge cycling rate and force production in living fatiguing muscle (abstract). *Biophysiology Journal. 49: 268a.*

De Bruyn-Prevost, P. and Lefebvre, F. (1980). The effect of various warming up intensities and durations during a short maximal anaerobic exercise. *European Journal of Applied Physiology. 43: 101-107.*

De Vries, H.A. (1959). Effects of various warm-up procedures on 100-yard times of competitive swimmers. *Research Quarterly for Exercise and Sport*. 30: 11-22.

Di Prampero, (1970). An analysis of oxygen debt contracted in submaximal exercise. *Journal of Applied Physiology. 29(5): 547-571.*

Dodd, S., Powers S. C., Callender, T. & Brooks, E. (1984). Blood lactate disappearance at various intensities of recovery exercise. *Journal of Applied Physiology: Respiratory Environment Exercise Physiology. 57(5):1462-1465.*

Donaldson, S. K. and Hermansen, L. (1978). Differential, direct effects of H^+ on Ca^{++}-activated force of skinned fibres from the soleus, cardiac and adductor magnus muscle of rabbits. *Pfluegers Archives. 376: 55-65.*

Donaldson, S. K. B. (1983). Effect of acidosis on maximum force generation of peeled mammalian skeletal muscle fibres. In: *International Series on Sport Sciences. Biochemistry of Exercise,* edited by Knuttgen, H. G., Vogel, J. A. and Poortsmans, J. Champaign, Ill: Human kinetics, vol. 13, p. 126-133.

Dorado, C., Sanchis-Moysi, J. and Calbet, J. A. (2004). Effect of recovery mode on performance, O_2 uptake, and O_2 deficit during high-intensity intermittent exercise. *Canadian Journal of Applied Physiology. 29(3): 227-244.*

Drust, B., Rasmussen, P., Mohr, M., Nielsen, B. and Nybo, L. (2005). Elevation in core and muscle temperature impairs repeated sprint performance. Acta *Physiologica Scandinavica. 183: 181-190.*

Duke, A. M. and Steele, D. S. (2000). Characteristics of phosphate-induced Ca^{2+} efflux from the SR in mechanically skinned rat skeletal muscle fibres. *American Journal of Physiolog. 278: C126-C135.*

Duke, A. M. and Steele, D. S. (2001a). Interdependent effects of inorganic phosphate and creatine phosphate on sarcoplasmic reticulum Ca^{2+} regulation in mechanically skinned rat skeletal muscle. *Journal of Physiology. 531: 729-742.*

Duke, A. M. and Steele, D. S. (1998). Effects of cyclopiazonic acid on Ca^{2+} regulation by the sarcoplasmic reticulum in saponin-permeabilized skeletal muscle fibres. *Pflugers Archives 436: 104-111.*

Duke, A. M. and Steele, D. S. (2001b). Mechanisms of reduced SR Ca^{2+} release induced by morganic phosphate in rat skeletal muscle fibers. *American Journal of Physiology 281: C418-C429.*

Dupont, G., Blondel N. and Berthoin, S. (2003). Performance for short intermittent runs: active recovery vs. passive recovery. *Journal of Applied Physiology 89: 548-554.*

Dutka, T. L., Lamb, G. D. (2000). Effect of lactate on depolarization-induced Ca^{2+} release in mechanically skinned skeletal muscle fibers. *American Journal of Physiology. 278: C517-C525.*

Edman, K. A. P. and Mattiazzi, A. R. (1981). Effects of fatigue and altered pH on isometric force and velocity of shortening at zero load in frog muscle fibres. *Journal of Muscle Research Cell Motility. 2: 321-334*.

Edward, R. G. and Lippold, O. C. (1956). The relation between force and integrated electrical activity in fatigue muscle. *Journal of Physiology. 132: 677-681.*

Edwards, R. H., Harris, R. C., Hultman, E., Kaijser, L., Hoh, D. and Nordesjo, L. O. (1972). Effects of temperature on muscle energy metabolism and endurance during successive isometric contraction sustained to fatigue, of quadriceps muscle in man. *Journal of Physiology. 220: 335-352.*

Febbraio, M. A., Carey, M. F., Snow, R. J., Stathis, C. G. and Hargeaves, M. (1996). Influence of elevated muscle temperature on metabolism during intense, dynamic exercise. *American Journal of Physiology. 271: R1251-R1255.*

Febbraio, M.A. (2000). Does muscle function and metabolism affect exercise performance in the heat? *Exercise and Sports Sciences Reviews*. 28(4): 171-176.

Fabiato, A. and Fabiato, F. (1978). Effects of pH on the myofilament and the Sarcoplasmic recticulum of skinned cell from cardiac and skeletal muscle. *Journal of Physiology*. 276: 233-255.

Felix, S.D., Manos, T.M., Jarvis, A. T., Jenssen, B. E., Headly, S. A. (1997). Swimming performance following different recovery protocol in female collegiate swimmer. *Journal of Swimming Research. 12: 1-6.*

Fink, W. J., Costil, D L. and Van Handel P. J. (1975). Leg muscle metabolism during exercise in the heat and cold. *European Journal of Applied Physiology Occupational Physiology. 34(3): 183-190.*

Fitts, R. H. (1992). Substrate supply and energy metabolism during brief high intensity exercise: importance of limiting performance. In: *Perspective in Exercise Science and Sport Medicine. Energy Metabolism in exercise and Sports,* edited by Lamb, D.R. and Gisolfi, C.V. Dubuque, IA: Brown & Benchmark, 5: 53-99.

Fitts, R. H. (1994). Cellular mechanisms of muscle fatigue. *Physiology Reviews. 74(1): 49-94.*

Fitts, R.H., and Holloszy, J. O. (1977). Contractile properties of rat soleus muscle: effects of training and fatigue. *American Journal of Physiology. 233: C86-C91.*

Fitts, R.H., and Holloszy, J. O. (1978). Effects of fatigue and recovery on contractile properties of frog muscle. *Journal of Applied Physiology. 45: 899-902.*

Fletcher, W. M. (1902). The relation of oxygen to the survival metabolism of muscle. *Journal of Physiology. 28: 474-498.*

Fletcher, W. M. and Hopkins, G. (1907). Lactic acid in amphibian muscle. *Journal of Physiology. 35: 247-309.*

Freygang, W. H., Goldstein, D. A. & Hellam, D. C. (1964). The after-potential that follows trains of impulses in frog muscle fibres. *Journal of General Physiology. 47: 929-952.*

Fryer, M. W., Owen, V. J., Lamb, G. D. and Stephenson, D. G. (1995). Effects of creatine phosphate and P_i on Ca^{2+} movements and tension development in rat skinned skeletal muscle fibres. *Journal of Physiology (London) 482: 123-140.*

Fukuba, Y., Hayashi, N., Koga, S. and Yoshida, T. (2002). VO_2 kinetics in heavy exercise is not altered by prior exercise with a different muscle group. *Journal of Applied Physiology. 92: 2467-2474.*

Fukuba, Y., Tanoue, A., Tanaka, M. and Miura, A. (1998). Effect of prior'acid-up' exercise on pulmonary O2 kinetics during heavy exercise. In: Nose, H., Nadel, E. R., Moromoto, T. *The 1997 Nagano Symposium on sports Sciences*. Carmel (IN): Cooper, 1998: 132-138.

Gaitanos, G. C., Williams, C., Boobis, L. H., and Brooks, S. (1993). Human muscle metabolism during intermittent maximal exercise. *Journal of Applied Physiology. 75(2): 712-719.*

Garland, S. J. (1991). Role of small diameter afferents in reflex inhibition during human muscle fatigue. *Journal of Physiology. 435 : 547-558.*

Garland, S. J. and Kaufman, M. P. (1995). Role of muscle afferents in the inhibition of motoneurons during fatigue. *Advanced Experimental Medical Biology. 384: 271-278.*

Gastin, P. B. (2001). Energy system interaction and relative contribution during maximal exercise. *Sports Medicine, 31 (10): 725-741.*

Genovely, H. and Stamford, B. A. (1982). Effect of prolonged warm-up exercise above and below anaerobic threshold on maximal performance. European *Journal of Applied physiology, Occupational Physiology. 48(3): 323-330.*

Gerbino, A., Ward, S. and Whipp, B. (1996). Effects of prior exercise on pulmonary gas-exchange kinetics during high-intensity exercise in humans. *Journal of Applied Physiology. 80(1): 99-107.*

Gibala, M. J. and Saltin, B. (1999). PDH activation by dichloroacetate reduces TCA cycle intermediates at rest but not during exercise in Humans. *American Journal of Physiology. 277; E33-E38.*

Gisolfi, C., Robinson, S. and Turrel, E. S. (1966). Effects of aerobic work performed during recovery from exhausting work. *Journal of Applied Physiology. 21(6): 1767-1772.*

Gleeson, T. T. (1996). Post exercise lactate: a comparative review of sites, pathway and regulation. *Annual Review. 58: 565-581.*

Godt, R. E., and Nosek, T. M. (1989). Changes in intracellular milieu with fatigue or hypoxia depress contraction of skinned rabbit skeletal and cardiac muscle. *Journal of Physiology. (London). 412: 155-180.*

Gollnick, P. D. and Hermansen, L. (1973). Biochemical adaptation to exercise: anaerobic metabolism. In: *Exercise and Sport Science Reviews, Ed : Willmore, J. H. New York: Academic. 1-43*.

Granier, P., Mercier, B., Mercier, J., Anselme, F. and Prefaut, C. (1995). Aerobic and anaerobic contribution to Wingate test performance in sprint and middle-distance runners. *Journal of Applied Physiology and Occupational Physiology. 70(1): 58-65.*

Grassi, B., Poole, D. C., Richardson, R. S., Knight, D. R., Errickson, B. K. and Wagner, P. T. (1996). Muscle O_2 uptake kinetics in humans: implications for metabolic control. *Journal of Applied Physiology. 80(3): 988-998.*

Grassi, B., Gladden, L. B., Samaja, M. et al. (1998). Faster adjustment of O_2 delivery does not effect VO_2 - kinetics in isolated in situ canine muscle. *Journal of Applied Physiology. 85 (4): 1394-1403.*

Grassi, B., Hogan M. C., Kelley, K. M., Aschenbach, W. G., Hamman, J. J., Evans, R. K., Patillo, R. E. and Gladden, L. B. (2000). Role of convective O_2 delivery in determing VO_2 on kinetics in canine muscle contracting at peak VO_2. *Journal of Applied Physiology. 89(4): 1293-1303.*

Gray, S. and Nimmo, M. (2001). Effect of active, passive or no warm-up on metabolism and performance during high-intensity exercise. *Journal of Sport Science. 19(9): 693-700.*

Greenhaff, P. L. and Timmonss, J. A. (1998). Interaction between aerobic an anaerobic metabolism during intense muscle contraction. *Exercise and Sports Science Reviews. 26: 1-30.*

Growther, J., Carey, M. F., Kemper, W. F. and Conly, K. E. (2002). Control of glycolysis. *American Journal of Physiology. 282: E67-E73.*

Gutin B., Sterwart K., Lewis S.,and Kruper J. (1976). Oxygen consumption in the first stages of strenuous work as a function of prior exercise. *Journal Sports Medicine. 16: 60-64.*

Gyork, S. (1993). Effect of repeated tetanic stimulation on excitation-contraction coupling in cut muscle fibres of frog. *Journal of Physiology. 464: 699-710.*

Hajoglou, A., Foster C, De Koning, J. J., Lucia, A., Kernozek, T. W. and Pocari, J. P. (2005). Effect of warm-up on cycle time trial performance. *Medicine and Science in Sports and Exercise. 37(9): 1608-1614.*

Hamada T., Sale D. G., MacDougall J. D., and Tarnopolsky M. A. (2003). Interaction of fibre type, potentiation and fatigue in human knee extensor muscles. *Acta Physiologica Scandinavica. 178: 165-173.*

Hargreaves, M. (1995). Skeletal muscle carbohydrate metabolism during exercise. In: Exercise Metabolism. Ed. M. Hargreaves, Champaign, IL: *Human Kinetics, 41-72*.

Hawley, J. A., Williams, M. M., Hamling, G. C. and Walsh, R. M. (1989). Effect of task-specific warm-up on anaerobic power. *British Journal of Sports Medicine. 23(4): 233-236*

Hepple, R. T. (2002). The role of O_2 supply in muscle fatigue. *Canadian Journal Applied Physiology 27(1): 56-69.*

Hermansen, L. (1971). Lactate production during exercise. In: *Advance in Experimental Medicine and Biology. Muscle metabolism during exercise, Edited by Pernow, B. and Saltin B. New York: Plenum. 11, 401-407*.

Hermansen, L. (1979). Effect of acidosis on skeletal muscle performance during maximal exercise in man. *European Physiopathology and Respiration (Bulletin). 15: 229-239.*

Hermansen, L. (1981). Effect of metabolic changes on force generation in skeletal muscle during maximal exercise. *Ciba Foundation Symposium Journal. 82 : 75-88.*

Hermansen, L. and Osnes, J. B. (1972). Blood and muscle pH after maximal exercise in man. *Journal of Applied Physiology 32: 304-398.*

Hermansen, L. and Stensvold, I. (1972). Production and removal of lactate during exercise in man. *Acta Physiologica and Scandinavica. 86: 191-201.*

Hermansen, L. and Vaage, O. (1977). Lactate disappearance and glycogen synthesis in human muscle after maximal exercise. *American Journal of Physiology. 233(5): E422-E429.*

Herzig, J. W., Peterson, J. C., Ruegg, J. C. and Solaro, R. J. (1981). Vanadate and phosphate ions reduced tension and increase cross-bridge kinetics in chemically skinned heart muscle. *Biochemistry-Biophysiology Acta. 672: 191-196.*

Hitchcock, H. C. (1989). Recovery of short-term power after dynamic exercise. *Journal of Applied Physiology. 67(2): 677-681*.

Hogan, M. C. (2001). Fall in intracellular Po2 at the onset of contractions in xenopus single skeletal muscle fibers. *Journal of Applied Physiology. 90: 1871-1876.*

Horswill, C. A., Costill, D.L., Fink, W. J., Flynn, M. G., Kirwan, J. P., Mitchell, J. B. and Hournard, J. A. (1988). Influence of sodium bicarbonate on sprint performance: Relationship to dosage. *Medicine and Science in Sports and Exercise. 20: 566-569.*

Howell, J. N. and Oetliker, H. (1987). Effects of repetitive activity, ruthenium red, and elevated extracellular calcium on frog skeletal muscle: Implication for t-tubule conduction. *Canadian Journal of Physiology and Pharmacology. 65: 691-696.*

Howell, J. N. and Snowdowne, K. W. (1981). Inhibition of tetanus tension by elevated extracellular calcium concentration. *American Journal of Physiology. 240: C193-C200.*

Hultman, E. and Sjohlm, H. (1983). Electromyogram, force and relaxation time during and after continuous electrical stimulation of human skeletal muscle in situ. *Journal of Physiology. 339: 33-40.*

Hultman, E., Bergstrom, M., Spriet, L.L. and Sonderlund, K. (1990). Energy metabolism and fatigue, in Taylor, A. W., Galnick, P. D., Green, H. J., Ianuzzo, C. D., Nable, E. G., Metivier, G and Sutton, J. R. (eds.), *International Series on Sport and Exercise: Biochemistry of exercise VII, (Vol. 21), Human Kinetics Publishers Inc., USA.*

Ikai., M. and Steinhans, A. H. (1961). Some factors modifying the expression of human strength. *Journal of Applied Physiology. 16: 157-163.*

Ikai, M. and Yabe, k. (1969). Training effect of muscular endurance by means ofvoluntary and electrical stimulation. *European Journal of Applied Physiology. 28: 55-60.*

Inbar, O. and Bar-Or, O. (1975). The effect of intermittent warm-up on 7-9 year old boys. *European Journal of Applied Physiology. 34: 81-89.*

Ingjer F. and Stromme S. B. (1979). Effect of active, passive or no warm-up on the physiological response to heavy exercise. *European Journal of Applied Physiology. 40: 273-282.*

Jacobs, I. (1981). Lactate concentrations after short, maximal exercise at various glycogen levels. *Acta Physiologica Scandinavica. 111: 465-469.*

Karlsson, J. and Saltin, B. (1970). Lactate, ATP and CP in working muscle during exhaustive exercise in man. *Journal of Applied Physiology 29: 598-602.*

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

Jeukendrup, A. E., Saris, W. H. and Wagenmakers, A. J. (1998). Fat metabolism during exercise: a review. Part 1: Fatty acid mobilization and muscle metabolism. (1998). *International Journal Sport Medicine. 19(4): 231-244.*

Jones, A. M., Koppo, K. and Burnley, M. (2003). Effects of prior exercise on metabolic and gas exchange responses to exercise. *Sports Medicine. 33(13): 949-971.*

Kabbara, A. A. and Allen, D. G. (1999). The role of calcium stores in fatigue of isolated single muscle fibres from the cane toad. *Journal of Physiology. 519: 169-176.*

Karatzaferi, C., De Haan, A., Ferguson, R. A., Van Mechelen, W. and Sargeant, A. J. (2001). Phosphocreatine and ATP content in human single muscle fibres before and after maximal dynamic exercise. *Pflugers Archives. 442: 467-474.*

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

Karvonen, J. (1992). Importance of warm-up and cool down on exercise performance. *Medicine in Sport and Science. 35: 189-214.*

Kavanagh, M. F. and Jacobs, I. (1988). Breath-by-breath oxygen consumption during performance of the Wingate Test. *Canadian Journal of Sports Science. 13(1): 91-93.*

Kawai, M. (1986). The role of orthophosphate in crossbridge kinetics in chemically skinned rabbit psoas fibres as detected with sinusoidal and step length alterations. *Journal of Muscle Research and Cell Motility. 7: 421-434.*

Kartz, A., Barnett, A., Costill, D. L., Fink, W. J. and Sharp, R. L. (1983) Leg muscle pH following sprint running. *In Biochemistry of Exercise International Series on Sport Science, Vol. 13.*

Kartz, A., Costill, D. L., King, D. S. Hargreaves, M. and Fink, W. J. (1984). Maximal exercise tolerance after induced metabolic alkalosis. *International Journal of Sport Medicine 5: 107-110.*

Kernell, D. (1969). Synaptic conductance changes and the repetitive impulse discharge of spinal motoneurones. *Brain Research. 15: 291-314.*

Kiens B., Saltin B., Wallye L., et al. (1989). Temporal relationship between blood flow changes and release of ions metabolites from muscle upon single work contractions. *Acta Physiologica Scandinavica. 213: 235-244.*

Klausen, K., Knuttgen, H. G. and Foster., H. V. (1972). Effect of pre-existing high blood lactate concentration on maximal exercise performance. *Scandinavian Journal of Clinical and Laboratory Investigation. 30(4): 425 – 419.*

Koga, S., Shiojiri T., S., Kondo N. and Barstow, T. J. (1997). Effect of increased muscle temperature on oxygen uptake kinetics during exercise. *Journal of Applied Physiology 83(4): 1333-1338.*

Koppo, K., Jones, A. M. and Bouckaert, J. (2003). Effect of prior heavy arm and leg exercise on VO2 kinetics during heavy leg exercise. *European Journal of Applied Physiology. 88: 593-600.*

Koppo, K., Bouckaert, J. (2001). The effect of prior of high-intensity cycling exercise on the VO2 kinetics during high intensity-cycling exercise is situated at the additional slow component *International Journal of Sports Medicine. 22: 21-26.*

Koppo, K., Jones, A. M., Vanden Bossche, L. and Bouckaert, J. (2002). Effect of prior exercise on Vo2 slow component is not related to muscle temperature. *Medicine and Science in Sports Exercise. 34: 1600-1604.*

Korge, P. (1995). Factors limiting adenosine triphosphatase function during high intensity exercise. *Sports medicine. 20: 215-225.*

Krebs, E. G., Graves, D. J. and Fischer, E. H. (1959). Factors affecting the activity of Ammonia metabolism in exercise and fatigue: A review. *Medicine and Science in Sports and Exercise. 15: 41-50.*

Laveder, G. and Bird, S.R. (1989). Effects of sodium bicarbonate ingestion upon repeated sprints. *British Journal of Sports Medicine. 23: 41-45.*

Krustup, P., Gonzales-Alonso, J. Quistorff, B. and Bangsbo, J. (2001). Muscle heat production and anaerobic energy turnover during repeated intense dynamic exercise in humans. *Journal of Physiology. 536: 947-956.*

Lakomy H. K. (1986). Measurement of work and power output using friction-loaded cycle ergometer. *Ergonomics. 29(4): 509-517.*

Lannergren, J. and Westerblad, H. (1989). Maximum tension and force velocity properties of fatigue, single Xenopous muscle fibres studied by caffeine and high K^+. *Journal of Physiology. 409: 473-490.*

Laver, D. R., Lenz, G. K. E. and Dulhunty, A. F. (2001). Phosphate ions channels in sarcoplasmic reticulum of rabbit skeletal muscle. *Journal of Physiology. 535: 715-728.*

LeRumeur, E., LeMoyec, L., Toulouse, P., LeBars, R. and deCertaines, J. (1990). Muscle fatigue unrelated to phosphocreatine and pH: An "in vivo" 31-P NMR spectroscopy study. *Muscle and Nerve 13: 438-444.*

Lindinger, M. I. and Sjogaard, G. (1991). Potassium regulation during exercise recovery. *Sport Medicine. 11: 382-401.*

Lüttgau, H. C., Gottshalk, G. and Berwe, D. (1987). The effect of calcium and Ca antagonists upon excitation-contraction coupling. *Canadian Journal of Physiology and Pharmacology 65: 717-723.*

MacDonald, M. J., Naylor, H. L., Tschakovsky, M. E. and Hughson, R. L. (2001). Peripheral circulatory factors limit rate of increase in muscle O2 uptake at onset of heavy exercise. *Journal of Applied Physiology. 90: 83-89.*

MacDonald, M. J., Pedersen, P. K. and Hughson, R. L. (1997). Acceleration of VO2 kinetics in heavy submaximal exercise by hyperoxia and prior high-intensity exercise. *Journal of Applied Physiology. 83(4): 1318-1325.*

MacDonald, W.A. and Stephenson, D. G. (2001). Effects of ADP on sarcoplasmic recticulum function in mechanically skin skeletal muscle fibres of the rat. *Journal of Physiology. 532: 499-508.*

MacIntosh, B. R., Rassier, D. E. (2002). What is fatigue? *Journal of Applied Physiology 27(1): 42-55.*

Mainwood, G. W. and Lucier, G. E. (1972). Fatigue and recovery in isolated frog sartorius muscles: The effects of bicarbonate concentration and associated potassium loss. *Canadian Journal of Physiology and Pharmacology. 50: 132-142.*

Ma, J. and Zhao, J. (1994). Highly cooperative and hysteretic response of the skeletal muscle ryanodine receptor to changes in proton concentrations. *Biophysical Journal. 67: 626-633.*

Malarecki, I. (1954). Studies on physiological justification of application of so-called warming up. *Acta Physiological Polonica. 5(4): 543-546.*

Margaria, R. Olivia, R. D., Di Prampero, P.E. & Cerretelli, P. (1969). Energy utilization in intermittent exercise of supramaximal intensity. *Journal of Applied Physiology. 26(6): 752-756.*

Martyn, D. A. and Gordon, A. M. (1992). Force and stiffness in glycerinated rabbit psoas fibers. *Pfluegers Archives. 99: 795-816.*

Massey, B. H., Johnson, W. R. and Kramer, G. F. (1961). Effect of warm-up exercise upon muscular performance using hypnosis to control the physiological variable. *The Research Quarterly. 32(1): 63-69.*

McArdle, W. D., Katch, F.I. and Katch. V. L. (1991). *Exercise Physiology: Energy,Nutrition and Human Performance (3rd ed.), Lea & Feibiger, Philadelphia.*

McCartney, N., Spriet, L. L., Heigenhauser, G. J. F., Kowalchuk, J. M. and Jones, N. L. (1986). Muscle power and metabolism in maximal intermittent exercise. *Journal of Applied Physiology. 60: 1164-1169.*

McCully, K. K., Boden, B., Tucheler, M., Fountain, M. R. and Chance, B. (1989). Wrist flexor muscles of elite rowers measured with magnetic resonance spectroscopy. *Journal of Applied Physiology 67: 926-932.*

McCully, K. K., Clark, B. J., Kent, A., Wilson, J. and Chance B. (1991). Biochemical adaptations to training: implications for resisting muscle fatigue. *Canadian Journal of Physiology and Parmacology. 69: 274-278.*

McCutcheon, L. J., Geor R. J. and Hindcheliff, K. W. (1999). Effects of prior exercise on muscle metabolism during sprint exercise in humans. *Journal of Applied Physiology. 87(5): 1914-1922.*

McDermot, J.C. and Bonen, A. (1992). Glyconeogenic and oxidative lactate utilization in skeletal muscle. *Canadian Journal of Physiology and Pharmacology 70: 142-149.*

McKenna, M. J., Green, R. A., Shaw, P. F. and Meyer, A. D. M. (1987). Test of anaerobic power and capacity. *The Australian Journal of Science and Medicine in Sport. 19(2): 13-17.*

McMaster W. C., Stoddard T. & Duncan W. (1989). Enhancement of blood lactate clearance following maximal swimming: Effect of velocity of recovery swimming. *The American Journal of Sport Medicine. 17(4): 472-476.*

McNaughton, L., Curtin, R., Goodman, G., Perry, D., Turner, B. and Showell, C. (1991). Anaerobic work and power output during cycle ergometer exercise: Effects of bicarbonate loading. *Journal of Sports Sciences 9: 151-160.*

Medbo, J. I. snd Tabata, I. (1989). Relative importance of aerobic and anaerobic energy release during short-lasting exhausting bicycle exercise. *Journal of Applied Physiology. 67(5): 1886-1996.*

Grojinovsky, A and Magel, R. (1982). Effect of warm-up on running performance. *The Research Quarterly. 41(1): 116-120.*

Merton, P. A. (1954) Voluntary strength and fatigue. *Journal of Physiology. 123: 553-564.*

Metzger, J. M. (1996). Effects of phosphate and ADP on shortening velocity during maximal and submaximal calcium activation of the thin filament in skeletal muscle fibres. *Biophysiology Journal. 70: 409-417.*

Metzger,J. M. and FittsR. H. (1987). Role of intracellular pH in muscle fatigue. *Journal of Applied Physiology. 62: 1392-1397*.

Metzger, J. M., Greaser, M. L. and Moss, R. L. (1989). Variations in cross-bridge attachment rate and tension with phosphorylation of myosin in mammalian skinned skeletal muscle fibres. *Journal of General Physiology. 93: 855-883.*

Metzger, J. M. Mechanism of chemomechanical coupling in skeletal muscle during work. In: *Perspectives in Exercise Science and Sport Medicine*. Vol 5: Energy Metabolism in Exercise and Sport, edited by D.. Lamb and C. V. Gisolfi. Dubuque, I. A.: Brown and Benchmark, 1992, p 1-44.

Metzger, J. M. and Moss, R. L. (1990). pH modulation of the kinetics of $Ca2^{+}$ sensitive cross-bridge state transition in mammalian single skeletal muscle fibres. *Journal of Physiology 42: 751-764*.

Merton, P. A. (1954). Voluntary strength and fatigue. *Journal of Physiology. 123: 553-564.*

Millar, N. C. and Homsher, E. (1990). The effect of phosphate and calcium of force generation in glycerinated rabbit skeletal muscle fibers. *Journal of Biology and Chemistry. 265: 20234-20240.*

Miller, R. G., Boska, M. D., Moussavi, R. S., Carson, P. J. and Weiner, M. W. (1988). ^{31}P nuclear magnetic resonance studies of high energy phosphates and pH in human muscle fatigue. *Journal of Clinical Investigation. 81: 1190-1196*.

Miller, R. G., Giannini, D., Milner-Brown, H. S., Layzer, R. B., Koretsky, A. P., Hooper, D. and Weiner, M. W. (1987). Effects of fatiguing exercise on high energy phosphates, force, and EMG: evidence for three phases of recovery. *Muscle Nerve. 10: 810-821.*

Monedero J., and Donne B. (2000). Effect of recovery interventions on lactate removal and subsequent performance. *International Journal of Sports Medicine. 21: 593-597.*

Mutch, B. J. and Banister E. W. (1983). Ammonia metabolism in exercise and fatigue: A review. *Medicine and Science in Sports and Exercise. 15: 41-50.*

Myburgh, K. and Cooke, R. (1997). Response of compressed skinned skeletal muscle fibers to conditions that simulate fatigue. *Journal of Applied Physiology. 82: 1297-1304.*

Nakamaru, Y., and Schwartz, A. (1972). The influence of hydrogen ion concentration on calcium binding and release by skeletal muscle sarcoplasmic reticulum. *Journal of General Physiology. 59: 22-32.*

Neville, M. E., Bogdanis, G. C., Boobis, L. H., Lakomy, H. K. A. and Williams, C. (1996). Muscle metabolism and performance during sprinting. In: *R. J. Maughan and S. M. Shireffs (Eds) Biochemistry of Exercise IX*. 243-260 Human Kinetics, Champaign.

Nordlund, M. M., Thorstensson, A. and Cresswell, A. G. (2004). Central and peripheral contributions to fatigue in relation to level of activation during repeated maximal voluntary isometric plantar flexions. *Journal of Applied Physiology. 96: 218-225.*

Nordsborg, N., Mohr, M., Pedersen, L. D., Nielsen, J. J., Langberg, H. and Bangsbo, J. (2003). Muscle interstitial potassium kinetics during intense exhaustive exercise: effect of previous arm exercise. *American Journal of Physiology. 285: R143-R148.*

Nosek, T. M., Fender, K. Y. and Godt, R. E. (1987). It is the dipronated inorganic phosphate that depresses the skinned skeletal muscle fibres. *Science. 236: 191-193.*

Nosek, T. M., Leal-Cardoso, J. H., McLaughlin, M. and Godt, R. E. (1990). Inhibitory influence of phosphate and arsenate on contraction of skinned skeletal and cardiac muscle. *American Journal of Physiology. 259: C933-939.*

O'Brian, B., Payne, W., Gastin, P. and Burge, C. (1997). A comparison of active and passive warm ups on energy system contribution and performance in moderate heat. The *Australian Journal of Science and Medicine in Sport. 29(4): 106-109.*

Osness, J. B. and Hermansen, L. (1972). Acid-base balance after maximal exercise of short duration. *Journal of Applied Physiology 32: 59-63.*

Ozyener F. Rossiter H. B., Ward S.A. et. al. (2001). Influence of exercise intensity on the on and off-transient kinetics of pulmonary oxygen uptake in humans. *Journal of Physiology. 533(3): 891-902.*

Paroline, M. L., Spriet, L. L., Hultman, E., Matsos, M. P., Hollidge-Horvat, M. G., Jones, N. L. and Heigenhauser, G, J. F. (2000). Effects of PDH activation by dichloroacetate in human skeletal muscle during exercise in hypoxia. *American Journal of Physiology. 279: E752-E761.*

Pate, E., Bhimani, M., Frank-Skiba, K. and Cooke R. (1995). Reduced effect of pH on skinned rabbit psoas muscle mechanics at high temperatures: Implications for fatigue. *Journal of Physiology (London). 486: 689-694.*

Pate, E. and Cooke, R. (1989). A model of crossbridge action: the effects of ATP, ADP and P_i. *Journal of Muscle Research and Cell Motility. 10: 181-196.*

Pate, E. and Cooke, R. (1989). Addition of phosphate to active muscle fibres probes actomyosin states within the powerstroke. Pflugers Archives. 414: 73-81.

Pedersen, T. H. (2004). Intracellular acidosis enhances the excitability of working muscle. Science. 305: 1144-1147.

Perrey, S., Scott, J., Mourot, L. and Rouillon, J. D. (2003). Cardiovascular and oxygen uptake kinetics during sequential heavy cycling exercise. *Canadian Journal of Applied Physiology. 28(2): 283-298.*

Peters-Futre, E. M. P., Noakes, T. D., Raine, R. I. And Terblanche, S. E. (1987). Muscle glycogen repletion during active post-exercise recovery. *American Journal of Physiology. 16: E305-E311.*

Pizarro, G., Fitts, R., Uribe, I. and Rios, E. (1989). The voltage sensor of excitation contraction coupling in skeletal muscle. Ion dependence and selectivity. *Journal of General Physiology. 94: 405-428.*

Posterino, G. S., Dutka, T. L. and Lamb G. D. (2001). L(+)-lactate does not affect twitch and titanic responses in mechanically skinned mammalian muscle fibres. *Pflugers Archives. 442: 197-203.*

Posterino, G. S. and Fryer, M. W. (1998). Mechanisms underlying phosphate-induced failure of Ca^{2+} release in single skinned skeletal muscle fibres of the rat. *Journal of Physiology 512: 97-108.*

Potma, E. J. and Stienen, G. J. M. (1996). Increase in ATP consumption during shortening in skinned fibres from rabbit psoas muscle: effects of inorganic phosphate. *Journal of Physiology 496: 1-12.*

Poulus, A. J., Doctor, H. J. and Westra, H. G. (1974). Acid-base balance and subjective feelings and fatigue during physical exercise. *European Journal of Applied Physiology and Occupational Physiology 33: 207-213.*

Ranatunga, K. W. Effect of acidosis on tension development in mammalian skeletal muscle. *Muscle Nerve 10: 439-445.*

Racinais, S., Blonc, S. and Hue, O. (2005). Effects of active warm-up and diurnal increase in temperature on muscular power. *Medicine and Science in Sports and Exercise. 37(12): 2134-2139.*

Renaud, J. M. (1986). Is the change in intracellular pH during fatigue large enough to be the main cause of fatigue? *Canadian Journal of Physiology and Pharmacology 64: 764-767.*

Ren, J. M. and Hultman, E. (1989). Regulation of phosphorylase-a activity in human skeletal muscle. *Journal of Applied physiology. 69: 919-923.*

Rios, E., and Pizarro, G. (1991). The voltage sensor of excitation-contraction coupling in skeletal muscle. *Physiological Reviews. 71: 849-908.*

Rios, E., Ma, J. and Gonzlez, A. (1991). The mechanical hypothesis of excitation-contraction (EC) coupling in skeletal muscle. *Journal of Muscle Research and Cell Motility. 12: 127-135.*

Ross, A. and Leveritt, M. (2001). Long-term metabolic and skeletal muscle adaptation to short sprint training: implication for sprint training and tapering. *Sport Medicine. 31(15): 1063-1082.*

Rossiter, H. B., Ward, S. A., Doyle, V. L., Howe, F. A., Griffiths, J. R. and Whipp, B. J. (1999). Inferences from pulmonary O_2 uptake with respect to intramuscular [phosphocreatine] kinetics during moderate exercise in humans. *Journal of Physiology. 518: 921-932.*

Rossiter, H. B., Ward, S. A., Kowalchuk, J. M., Howe, F. A., Griffiths, J. R. and Whipp, B. J. (2001). Effect of prior exercise on oxygen uptake and phosphocreatine kinetics during high-intensity knee-extension exercise in humans. *Journal of Physiology. 537: 291-303.*

Rousseau, E. and Pinkos, J. (1990). pH modulates conducting and gating behavior of single calcium release channels. *Pflugers Archives 415: 645-647.*

Sale, D.G. (2002). Postactivation potentiation: role in human performance. *Exercise and Sports Sciences Reviews*. 30(3): 138-143.

Sahlin, K., Erdstrom, L., Sjoholm, H. and Hultman, E. (1981). Effect of lactic acid accumulation and ATP decrease on muscle tension and relaxation. *The American Journal of Physiology. 20(3): C121-C126.*

Sahlin, K., Erdstrom, L. and Sjoholm, H. (1983).Fatigue and creatine phosphate depletion during carbon dioxide-induced acidosis in rat muscles. *American Journal of Physiology. 245: C15-C20.*

Sahlin, K. (1978). Intracellular pH and energy metabolism in skeletal muscle of man. *Acta Physiologica Scandinavica. 455(supp): 1-56.*

Sahlin, K., Harris, R. C. and Hultman, E. (1979). Resynthesis of creatine phosphate in human muscle after exercise in relation to intramuscular pH and availability of oxygen. *Scandinavian Journal of Clinical and Labaratory Investigation. 39: 551-558.*

Sahlin, K., Harris, R. C., Nylind, B. and Hultman, E. (1976). Lactate content and pH in muscle samples obtained after dynamic exercise. *Pflugers Archives. 367: 143-149.*

Sahlin, K. (1986). Muscle fatigue and lactic acids accumulation. *Acta Physiological Scandinavica. 556: 83-91.*

Sahlin K. (1992). Metabolic factors in fatigue. *Sports medicine 13(2): 99-107*

Sahlin, K. and Broberg S. (1990). Adenine nucleotide depletion in human muscle during exercise: causality and significance of AMP deamination. *International Journal of Sport Medicine. 11(2): S62-S67.*

Sahlin, K. And Ren, J. M. (1989). Relationship of contraction capacity to metabolic changes during recovery from fatiguing contraction. *Journal of Applied Physiology. 67(2): 648 -654.*

Sahlin, K., Tongkonogi, M., and Sőderlund K. (1998). Energy supply and muscle fatigue in humans. *Acta Physiological Scandinavica. 162: 261-266.*

Saltin, B. Gage, A. P., Stolwijk, J. A. J. (1968). Muscle temperature during sub maximal exercise in man. *Journal of Applied Physiology 25(6): 679-688*

Saltin, B., Nazar, K., Costill, D. L., Stein, E., Jansson, E., Essen, B. and Gollnick, D. (1976). The nature of training response; peripheral and central adaptations of one-legged exercise. Acta *Physiologica Scandinavica. 96(3): 289-305.*

Sargeant, A. J. (1983). Effect of muscle temperature on maximal short term power output in man. *Journal of Physiology. 341: 35p.*

Sargeant, A. J. (1987). Effect of muscle temperature on leg extension force and short-term power output in humans. *European Journal of Applied Physiology. 56: 693-698.*

Sargeant, A. J. and Dolan P. (1987). Effect of prior exercise on maximal short-term power output in humans. *Journal of Applied Physiology 63(4): 1475-1480.*

Saugen, E. Vollestad, N. K., Gibson, H., et al. (1997). Dissociation between metabolic and contractile responses during intermittent isometric exercise in man. *Experimental Physiology. 82: 213-226.*

Scheuermann, B. W., Bell C, Peterson D. H. et al. (2002). Determinants of oxygen kinetics for moderate exercise are speeded in older humans by prior heavy exercise. *Journal of Applied Physiology. 92: 6009-6015*.

Scheuermann, B. W., Hoelting, B. D., Noble, M. L. and Barstow, T. J. (2001). The slow component of O2 uptake is not accompanied by changes in muscle EMG during repeated bouts of heavy exercise in humans. *Journal of Physiology. 531: 245-256*.

Sejersted, O. M. Electrolyte imbalance in body fluids as a mechanism of fatigue during exercise. In: *Perspectives in Exercise Science and Sport Medicine, Energy Metabolism in Exercise and Sport,* edited by D. R.Lamb and C. V. Gisolfi. Dubuque, I. A.: Brown & Benchmark, 1992, p. 149-200.

Signorille J. F., Ingalls C. & Tremblay L. M. (1993). The effect of active and passive recovery on short-term, high intensity power output. *Canadian Journal of Applied Physiology. 18: 1 31-42.*

Sjogarard, G., Adams, R. P. and Saltin, B. (1985). Water and ion shifts in skeletal muscle of humans with intense dynamic knee extension. *American Journal of Physiology. 248: R190-196.*

Sjogarard, G. (1991). Role of exercise induced potassium fluxes underlying muscle fatigue: a brief review. *Canadian Journal of Physiology and Pharmacology. 69(2): 238-245.*

Spencer, M., Bishop, D., Dawson, B. and Goodman, C. (2005). Physiological and metabolic responses of repeated-sprint activities: specific to field-based team sports. *Sports Medicine. 35(12): 1025-1044.*

Spierer, D. K., Goldsmith R., Baran, D. A., Hryniewicz K., and Katz S. D. (2003). Effect of active vs passive recovery on work performed during serial supramaximal exercise tests. *International Journal of Sports Medicine 25: 109-144.*

Stamford, B. A., Moffatt, R. J. Weltman, A., Maldonado, C. & Curtis, M. (1978b). Blood lactate disappearance after supramaximal one-legged exercise. *Journal of Applied Physiology. 45(2): 244-248.*

Stamford, B. A., Rowlland, R. & Moffatt R. J. (1978a). Effects of prior severe exercise on assessment maximal oxygen uptake. *Respiratory Environmental and Exercise Physiology. 44(4): 559-563.*

Stamford, B. A., Weltman, A., Moffatt, R. J. & Fulco, C. (1978c). Effects of severe prior exercise on assessment of maximal oxygen uptake during one-versus two-legged cycling. The *Research Quarterly 49(3): 363-371.*

Stamford, B. A., Weltman, A., Moffat, R. & Sady, S. (1981). Exercise recovery above and below anaerobic threshold following maximal work. *Journal of Applied Physiology. 51(4): 840-844.*

Steele, D. S. and Duke, M. D. (2003). Metabolic factors contributing to altered Ca^{++} regulation in skeletal muscle fatigue. *Acta Physiologica Scandinavica. 179: 39-48.*

Stephens, J. A. and Taylor, A. A. 91972). Fatigue of maintained voluntary muscle contraction in man. *Journal of Physiology. 220: 1-18.*

Stephenson, D. G., Lamb, G. D., Stephenson, G. M (1998). Events of the excitation-contraction-relaxation (ECR) cycle in fast- and slow-twitch mammalian muscle fibres relevant to muscle fatigue. *Acta Physiology Scandinavia. 162: 229-245.*

Stewart, I. B. and Sleivert, G. G. (1998). The effect of warm-up intensity on range of motion and anaerobic performance. *Journal of Orthophaedic and Sports Physical Therapy. 27(2): 154-161.*

Stewart, K., Gutin, B. and Lewis, S. (1973). Prior exercise and circulorespiratory endurance. *The Research Quarterly, 44 (2): 169-177.*

Stienen, G. J. M., Versteeg, P. G. A., Papp, Z and Elizinga, G. (1992). Mechanical properties of skinned rabbit psoas and soleus muscle fibres during lengthening: effects of phosphates and Ca^{2+}. *Journal of Physiology. 451: 503-523.*

Spencer, M., Bishop, D., Dawson, B. and Goodman C. (2005). Physiological and metabolic responses of repeated-sprint activities. *Sports Medicine. 35(12): 1-21.*

Spierer, D. K., Goldsmith R., Baran, D. A., Hryniewicz K., and Katz S. D. (2003). Effect of active vs passive recovery on work performed during serial supramaximal exercise tests. *International Journal of Sports Medicine 25: 109-144.*

Spriet, L. L., Lindinger, M. I., McKelvie, R. S., Heigenhauser, G. J. F. and Jones, N. L. (1989). Muscle glycogenolysis and H+ concentration during maximal intermittent cycling. *Journal of Applied Physiology. 66(1): 8-13.*

Spriet, L. L. (1995). Caffeine and performance. *International Journal of Sport Nutrition. 5: S84-S99.*

Taylor, E. W. (1977). Transient phase of adenosine triphosphate hydrolysis by myosin, heavy meromyosin and subfragment I. *Biochemistry. 16: 732-739.*

Tesch, P. A. and Wright, J. E. (1983). Recovery from short term intense exercise: its relation to capillary supply and blood lactate concentration. *European Journal of Applied Physiology. 52: 98-103.*

Tesch, P. A., Sjodan, B., Thorstensson, A. & and Karlsson J. (1978). Muscle fatigue and its relation to lactate accumulation and LDH activity in man. *Acta Physiologica Scandinavica. 103: 413-420.*

Theorell H. (1934). The effect of temperature on myoglobin. *Biochemistry. 73: 268.*

Thiriet P., Gonaz D., Woiassi D., Oumarou T., Gelas H. & Lacour J. R. (1993). The effects of various recovery modalities on subsequent performance, in consecutive supramaximal exercise. *The Journal of Sports Medicine and Physical Fitness. 33(2): 118-129.*

Thomas, M. M., Cheung, S. S. Elder, G. C. and Sleivert, G. (2006). Voluntary muscle activation is impaired by core temperature rather than local muscle temperature. *Journal of Applied Physiology. 100: 1361-1369.*

Thompson, L. V. and Fitts R. H. (1992). Muscle fatigue in the frog semitendinosus: role of high-energy phosphate and P_i. *American Journal of Physiology*. 263: C803-809.

Thompson, H. (1958). Effect of warm-up upon physical performance in selected activities. *Research Quarterly Exercise and Sports. 29(2): 55-63.*

Tokiwa, T. and Tonomara, Y. (1965). The pre-steady stsate of the myosine-adenosine triphosphate system. *Journal of Biochemistry. 57: 616-626.*

Tordi, N., Perrey, S., Harvey, A. and Hughson, R. L. (2002). Oxygen uptake kinetics during two bouts of heavy cycling separated by fatiguing sprint exercise in humans. *Journal of Applied Physiology. 94: 533-541.*

Vandervoort, A.A., Quinlan, J. and McComas, A.J. (1983). Twitch potentiation after voluntary contraction. *Experimental Neurology*. 81: 141-152.

Vollestad, N. K., Sejersted, O. M., Bahr, R. et al. (1988). Motor drive and metabolic responses during repeated submaximal contractions in humans. *Journal of Applied Physiology. 64: 1421-1427.*

Vyskocil, F., Hnik, P., Rehfeldt, H., Vejsada, R. and Ujec, E. (1983). The measurement of K+e concentration changes in human muscles during volitional contractions. *Pflugers Archives. 399: 235-237.*

Weiner, W. M., Moussavia, R. S. Baker A. J., Boska, M. D. and Miller R. G. (1990). Constant relationships between force, phosphate concentration, and pH in muscles with differential fatigability. *Neurology. 40: 1888-1893.*

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

Weltman, A., Stamford, B. A., Moffatt, R. J. & Leslie, K. (1977). Exercise recovery, lactate removal, and subsequent high intensity exercise performance. *Research Quarterly, 48: 786-796.*

Westerblad, H. and Allen, D. G. (1991). Changes in myoplasmic calcium concentration during fatigue in single mouse muscle fibers. *Journal of General Physiology. 98: 615-635.*

Westerblad, H. and Allen D. G. (1992). Myoplasmic free Mg^{2+} concentration during repetitive stimulation of single fibres from mouse skeletal muscle. *Journal of Physiology. 453: 413-414.*

Westerblad, H. and Allen D. G. (2002). Recent advances in the understanding of skeletal muscle fatigue. *Current Opinion of Rheumatology. 14: 648-652.*

Westerblad, H., Bruton, J. D. and Lannergren, J. (1997). The effect of intracellular pH on contractile function of intact, single fibres of mouse muscle decline s with increasing temperature. *Journal of Physiology (London). 500: 193-204*.

Westerblad, H. and Lannergren, J.(1988). The relation between force and intracellular pH in fatigue, single xenapus muscle fibers. *Acta Physiologica Scandinavica. 13: 83-89.*

Westerblad, H. and Lannergren J. (1991). Slowing of relaxation during fatigue in single mouse muscle fibre. *Journal of Physiology*. 434: 323-336.

Weiner, M. W., Moussavi, R. S., Baker, A. J., Boska, M. D. and Miller, R. G. (1990). Constant relationships between force, phosphate concentration, and pH in muscles with differential fatigability. *Neurology. 40: 1888-1893.*

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

Weltman, A., Stamford, B. A., Moffatt, R. J. & Leslie, K. (1977). Exercise recovery, lactate removal, and subsequent high intensity exercise performance. *The Research Quarterly. 48: 786-796.*

Westerblad, H. and Allen, D. G. (1996). The effects of intracellular injections of phosphate on intracellular calcium and force in single fibres of mouse skeletal muscle. *Pflugers Archives 431: 964-970.*

Westerblad, H., Dahlstedt, A. J. and Lannergren, J. (1998). Mechanisms underlying reduced maximum shortening velocity during fatigue of intact, single fibres of mouse muscle. *Journal of Physiology (London). 510: 269-277.*

West, T. G., Curtin, N. A., Ferenczi, M. A., He, Z. H., Sun, Y. B., Irving, M. and Woledge, R. C. (2003). Actomyosin energy turnover declines while force remains constant during isometric muscle contraction. *Journal of Physiology (London). 555: 27-43.*

Wilke, D. R. (1986). Muscle fatigue: effect of hydrogen ions and inorganic phosphate. *Federation Proceedings. 45: 2921-2923*.

Wilkes, D., Gledhill, N. and Smyth, R. (1983). Effect of acute induced metabolic alkalolis on 800m racing time. *Medicine and Science in Sports and Exercise, 15: 277-280.*

Willis, W. T., and Jackman, M. R. (1994). Mitochondrial function during heavy exercise. *Medicine and Science in Sport and Exercise. 26: 1347-1353*.

Wilson, J. R., McCully, K. K., Mancini, D. M., Boden B. and Chance, B. (1988). Relationship of muscular fatigue to pH and diprotonated P*i* in humans: a ^{31}P-NMR study. *Journal of Applied Physiology. 64: 2333-2339*.

Wiseman, R. W., Beck, T. W. and Chase, P. B. (1996). Effect of intracellular pH on force development depends on temperature in intact skeletal muscle from mouse. *American Journal of Physiology. 271: C878-886.*

Wooton, S. A., and Williams, C. (1983). The influence of recovery duration on repeated maximal sprints. In Biochemistry of exercise. International Series of Sport Sciences. (edited by H. G. Knuttgen, J. A., Vogel and J. Portmans), pp. 579-583. *Champaign, III. : Human kinetics.*

Wright, V. and Johns, R.J. (1961). Quantitative and qualitative analysis of joint stiffness in normal subjects and in patients with connective tissue disease. *Annals of the Rheumatic Diseases*. 20: 34-36.

Young M., Fricker P., Maughan R., and MacAuley D. (1998). The traveling athlete: issues relating to the commonwealth games, Malaysia 1998. *Journal of Sports Medicine. 32: 77-81.*