# THE EFFECTS OF DIFFERENT INTERMITTENT PRIMING STRATEGIES ON 3KM CYCLING PERFORMANCE

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## Attestation of authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, if contains no material previously published or written by another person, nor material to which the substantial extent has been accepted for the qualification of any degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made.

Signed.....

Date.....

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## Abstract

Priming exercise, or the 'warm-up', is an accepted practice prior to exercise participation, physical training or sporting competition. Traditionally, low intensity exercise has been used prior to both short- and long-duration events in an effort to prepare the athlete, but not fatigue them. Recently, however, a more scientific approach to priming exercise has been considered important, with some research suggesting that a high intensity intermittent priming strategy may be optimal. However, given the paucity of performance focussed 'warm-up' studies, and that existing data regarding high-intensity priming strategies is inconclusive, the aim of this thesis was to determine the effects of three high-intensity intermittent priming strategies on physiological responses and subsequent 3km laboratory time-trial (TT) performance.

Ten well-conditioned endurance-trained male cyclists (mean  $\pm$  SD: age, 28.3  $\pm$  8.4 yr, body mass, 81.8  $\pm$  11.6 kg, stature, 1.8  $\pm$  0.1 m,  $\dot{V}O_{2peak}$ , 4.6  $\pm$  0.5 L·min<sup>-1</sup>) were recruited for this study. After an initial incremental exercise test to exhaustion, participants completed four 3km time trials (TT) on four separate occasions, each preceded by a different priming strategy. These included a 'self-selected' (control) condition, and three high-intensity intermittent priming strategies of varying intensity (100% and 150% of the power at  $\dot{V}O_{2peak}$ , and all-out) and fixed duration (15 minutes), each in predetermined random order. Five minutes passive rest separated each priming exercise condition from the experimental 3km-TT. Oxygen uptake ( $\dot{V}O_2$ ) and heart rate (HR) were measured continuously, while blood lactate concentration ([BLa]) and core temperature (T<sub>C</sub>) were recorded at rest, postpriming exercise, and immediately prior to and following the 3km-TT. In an attempt to provide a mechanistic explanation for changes in performance,  $\dot{V}O_2$  kinetic variables were determined from the  $\dot{V}O_2$  data. Performance was quantified as a mean power (W<sub>mean</sub>) and total time taken to complete the 3km-TT. Mean power output and time taken for each 500m segment of the 3km-TT were also calculated.

Results demonstrated that the athletes self-chosen priming condition (378.6  $\pm$  44.0 W) resulted in W<sub>mean</sub> that was slightly greater than both the lowest (376.3  $\pm$  44.9 W; 0.7%; p = 0.57) and moderate (373.9  $\pm$  47.8 W; 1.5%, p = 0.30) intensity intermittent priming

condition, but significantly greater than the 'all-out' intermittent sprint priming condition (357.4  $\pm$  44.5 W; 5.8%, p = 0.0033). Similar differences were observed for time. While differences existed in the O<sub>2</sub> deficit (however, mainly non-significant), these differences did not provide clear explanations for the differences in performance, with the moderate priming condition displaying a significantly reduced O<sub>2</sub> deficit (59.4  $\pm$  15.6 L, p < 0.05), despite the non-significant change in W<sub>mean</sub>, compared to the self-chosen priming condition (73.3  $\pm$  18.6 L). Additionally no significant differences were observed in either the time constant or the mean response time of  $\dot{V}O_2$ . Significant findings with regard to HR, [BLa] and T<sub>C</sub> were observed, but consistent with  $\dot{V}O_2$  kinetic variables, they were not related to, nor explain performance changes.

In conclusion, regardless of intensity, different high-intensity intermittent priming exercise did not improve 3km-TT performance more than the control condition (self-chosen). A priming strategy that is overly intense was detrimental to subsequent cycling performance. The observed finding that a self-chosen priming strategy resulted in a comparable performance suggests that athletes are able to self-select (consciously or sub-consciously) a 'warm-up' that is of appropriate intensity/duration. Further work utilising the priming strategies from the current study with events of shorter duration is required to further clarify how priming strategies of this nature may affect track cycling performance.

### **Chapter One: Introduction**

Priming exercise, or the 'warm-up', is an accepted practice in the preparation for any sporting or exercise participation or competition. However, many coaches, conditioners and athletes often have applied no scientific criteria on the intensity chosen to perform priming exercise, commonly just performing that which they have always done, or those before them have done. It is now being recognised by coaches and sports scientists that the priming exercise for supramaximal performance may be more important than previously considered. This has generated scientific interest on how best to utilise the time prior to performance, as demonstrated by increased research publications regarding the physiology of prior exercise.

The proposed benefits of priming exercise are to prepare the energy systems involved in oxygen ( $O_2$ ) transport and utilization, thereby allowing the athlete to reach a higher level of aerobic metabolism more rapidly (Bishop, Bonetti, & Dawson, 2001; Bishop, Bonetti, & Spencer, 2003). There are, however, many other physiological (metabolic, biochemical and neuromuscular) benefits to priming exercise that may also improve performance. More specifically, these may include moderately elevated lactate concentration within the blood and muscles (Cairns, 2006), increased muscle and core temperature ( $T_M$  and  $T_C$ , respectively; Mohr, Krustrup, Nybo, Nielsen, & Bangsbo, 2004), elevated heart rate (HR; Stewart & Sleivert, 1998) and greater muscle fibre activation (Krustrup, Soderland, Mohr, & Bangsbo, 2004). These factors all have beneficial or adverse influences on performance in the subsequent activity, depending on the magnitude of the response. The magnitude of the response can depend on how intense or long the priming exercise is, as well as whether it is a continuous or discontinuous (i.e. intermittent) bout of exercise.

Traditionally, lower intensity exercise has been used in preparation for both short- and long-duration events (sub-maximal and supramaximal intensities) in an effort to prepare the athlete, but not fatigue them. Recently, however, a more scientific approach to priming exercise has been considered important (Bishop, 2003a, 2003b). There are few studies that have investigated different priming strategies and their effect on a variety of subsequent performance tests (e.g. Bishop et al., 2001; Bishop et al., 2003; Hajoglou, Foster, de Koning et al., 2005). Unfortunately, however, the results of these studies have not provided

coaches and athletes with consistent findings. Furthermore, inconsistent priming conditions (duration and intensity) and mode of exercise [e.g. kayaking (Bishop et al., 2001; Bishop et al., 2003), cycling (Hajoglou et al., 2005; Jones, Wilkerson, Burnley, & Koppo, 2003; Wilkerson, Koppo, Barstow, & Jones, 2004) and treadmill exercise (Billat, Bocquet, Slawinski et al., 2000)] make comparisons between studies (and recommendations for coaches) difficult to ascertain.

Whilst there are many studies that have investigated the effects of different 'warm up' approaches, or 'prior exercise' conditions on subsequent physiological responses (e.g. Billat, Bocquet et al., 2000; Burnley, Jones, Carter, & Doust, 2000; Koga, Shiojiri, Kondo, & Barstow, 1997; Stewart & Sleivert, 1998), few have considered actual sport-specific performance measures (Bishop et al., 2001; Bishop et al., 2003; Hajoglou et al., 2005; Mohr et al., 2004). Of particular interest to this proposed thesis are the two performancefocussed studies by Bishop and colleagues (Bishop et al., 2001; Bishop et al., 2003) and more recently the study by Hajoglou et al (2005). The results from the study of Bishop et al. (2001) demonstrated significantly improved average power in the first half of the twominute kayak ergometer trial in the moderate intensity priming exercise when compared to the highest intensity (5.4%), and a tendency for low intensity priming exercise to be better than high (4.2%). Despite, the greater mean and peak powers in the moderate and low priming intensities during the first half of the test, performances were very similar in the second half of the test. These results suggest that performance is improved with a moderately intense priming exercise, resulting in moderately elevated lactate acidosis, rather than a higher intensity priming strategy, which resulted in a greater metabolic cost and significant lactate acidosis. The authors conclude that a certain degree of metabolic acidemia may be necessary to sufficiently accelerate  $\dot{V}O_2$  kinetics and thus increase the aerobic contribution to the performance event, whilst saving anaerobic energy contributions for later in the trial. Conversely, too much acidosis (as per a very high-intensity priming strategy) may interfere with muscle contractile processes and reduce subsequent performance.

Following on from these findings, Bishop et al (2003) endeavoured to further refine the 'warm up' for supra-maximal exercise performance. Subsequently, they sought to determine the difference between the previous successful moderate intensity continuous

priming exercise with that of a high-intensity intermittent (rather than continuous; Bishop et al., 2001) priming strategy. Interestingly, the high intensity *intermittent* priming strategy that resulted in a moderate elevation of blood lactate concentration ([BLa]) was found to enhance performance more than a moderate intensity *continuous* priming strategy. This evidence provided further support for an increased [BLa] being a factor that contributes to the increased power output and improved overall performance. In addition, the authors suggested that the significantly greater peak power after the high-intensity intermittent priming strategy could be attributed to the stimulation of fast twitch motor units before the performance trial. To extend this work, it would be useful to further investigate the optimal intensity of the intermittent bouts as part of the overall priming strategy. Bishop et al. (2003) only used 200%  $\dot{VO}_{2peak}$ . It is not yet known whether 100% or 150%  $\dot{VO}_{2peak}$  would have a similar, or more substantial effect.

More recently, the influences of different priming strategies were investigated with regards to three-kilometre cycle ergometer time trial (3km-TT) performance (Hajoglou et al., 2005). Priming strategies consisted of a no prior exercise control, an easy 'warm-up' (EWU) and a hard 'warm-up' (HWU) followed by six minutes of rest. Following the 3km-TT's, and in agreement with Bishop et al. (2001), the  $\dot{V}O_{2peak}$  attained was not significantly different amongst conditions. As expected, post-priming [BLa] was greater with increasing priming intensity. Overall, the EWU and HWU had very similar 3km-TT times of 266.8 ± 12.0 s and 267.3 ± 3 s respectively, and both were significantly faster than the control condition of no prior exercise. While the observed data provides evidence of the benefits of prior exercise before high intensity maximal exercise, it did not provide evidence of greater benefits of high intensity continuous priming exercise over moderate intensity priming exercise.

Based on the aforementioned studies, it would appear beneficial to further investigate the use of high intensity intermittent priming strategies. Track cycling includes events varying in duration from 'sprint' events lasting around ten seconds, to 'endurance' events lasting up to one hour. The track event of interest for the current study is the pursuit. The distances for individual and team pursuits are 4km (men) and 3km (women and juniors). Pursuits could be considered 'sprint-endurance' events, where high speeds are required over

extended periods (~4min 15s world class men four kilometres, ~3min 25s world-class women three kilometres), similar to middle-distance running. Clearly, the results in an event of this nature would surely be influenced, at least in part, by the pre-event priming strategy. Thus, this thesis aimed to 1) determine the effects of three high-intensity intermittent priming strategies on the physiological responses (emphasis will be placed on the kinetic response of  $\dot{V}O_2$ , [BLa], HR and core temperature (T<sub>C</sub>)) in comparison to the participants self-chosen (i.e. current practice) priming exercise and 2) determine the effect of different intensity intermittent priming strategies on 3km-TT laboratory cycle performance. It is envisaged that the findings of this study will be of interest to track cycling coaches, and potentially a range of coaches in other sports where the intensity/duration of events are similar.

### **Chapter Two: Literature Review**

#### 1. Introduction

'Warming-up' is accepted practice in the preparation for any sporting or exercise participation or competition. However, many coaches, conditioners and athletes themselves often applied no scientific criteria on the intensity chosen to perform that 'warm-up', commonly just performing that which they have always done, or those before them have done. Previous research into fundamental physiology associated with prior exercise has generated further scientific interest on how to utilise the time prior to *performance*. Research into this area has resulted in greater recognition by coaches and conditioners that the 'warm-up' in supramaximal performance may be more important than it was once considered.

The proposed benefits of the 'warm-up' are to prepare the energy systems involved in oxygen  $(O_2)$  transport and utilization, thereby allowing the athlete to reach a higher level of aerobic metabolism more rapidly (Bishop et al., 2001; Bishop et al., 2003). There are however other physiological and metabolic benefits to priming exercise that may contribute to improved performance. More specifically, these may include moderately elevated lactate concentration within the blood and muscle, increased muscle and core temperature (Q<sub>10</sub> effect), elevated heart rate and greater muscle fibre activation (Burnley, Doust, & Jones, 2002; Gray, Devito, & Nimmo, 2002). Additionally, as displayed in Figure 1-1, it has also been proposed that physiological responses to 'warm-up' may be categorised into temperature related and non-temperature related factors (Bishop, 2003a). These factors can all have beneficial or adverse influences on performance in the subsequent activity depending on the magnitude of the evoked response, and whether the event is a continuous (e.g. running) or discontinuous (intermittent, e.g. soccer) endurance activity (Mohr et al., 2004), strength or power exercises such as weight-lifting or throwing events (Leveritt & Abernethy, 1999), flexibility (Stewart & Sleivert, 1998) or agility (Young, James, & Montgomery, 2002).

Maximal and supramaximal exercise is that which is performed at an intensity equal to, or greater than that which the body can provide energy aerobically, i.e. above the peak rate of oxygen uptake ( $\dot{VO}_{2peak}$ ) (Medbo & Tabata, 1993). Because of this oxygen deficit, the

active muscles must obtain additional energy from other sources, i.e. via anaerobic glycolysis. Therefore, emphasis may be shifted to other physiological factors to prime the body's energy systems for performance than compared with submaximal tasks.

Temperature related Decreased resistance of muscles and joints Greater release of oxygen from haemoglobin and myoglobin Speeding of metabolic reactions Increased nerve conduction rate
Greater release of oxygen from haemoglobin and myoglobin Speeding of metabolic reactions
Speeding of metabolic reactions
Increased nerve conduction rate
Increased thermoregulatory strain
Non-temperature related
Increased blood flow to muscles
Elevation of baseline oxygen consumption
Postactivation potentiation
Psychological effects and increased preparedness

Table 2-1: Possible effects of warm-up from Bishop (2003a)

Traditionally, lower intensity exercise has been used in preparation for both short- and long-duration events (sub-maximal and supramaximal intensities) in an effort to prepare the athlete, but not fatigue them. However, some recent evidence suggests that a low intensity 'warm-up' may not be as beneficial as one of high intensity. However, the results of these studies have not all provided coaches and athletes with consistent results.. Furthermore, inconsistent priming conditions (duration and intensity) and varying mode of exercise, e.g. kayaking (Bishop et al., 2001; Bishop et al., 2003), cycling (Hajoglou et al., 2005; Jones et al., 2003; Wilkerson et al., 2004) and treadmill exercise (Billat, Bocquet et al., 2000), make comparisons between studies, and recommendations for coaches, difficult. It has also been proposed that priming exercise utilising a non-specific exercise with a different muscle group, or using passive methods of 'warm-up' (e.g. heat packs, hot water), may be just as beneficial at eliciting the same physiological response but improving subsequent performance (Burnley, Doust, Ball, & Jones, 2002; Burnley, Doust, & Jones, 2002; Koppo & Bouckaert, 2000; Koppo, Jones, & Bouckaert, 2003; Koppo, Jones, Vanden Bossche, & Bouckaert, 2002). This may be achieved through preparing the major energy systems without inducing fatigue of the active muscles.

Based on the above, it would appear beneficial to further investigate which priming strategies are the most beneficial and most appropriate for the demands of the specific competition. In two parts, this review will explore the different priming strategies investigated by various researchers in relation to 1) physiological responses and 2) their effect on performance. The variables are represented strongly in the literature investigating prior exercise (both in mechanistic and performance-focussed studies) are the kinetic response of  $\dot{V}O_2$ , blood lactate accumulation and core temperature, and therefore, particular emphasis is placed on these.

#### 2. 'Warm-up'/priming strategies/prior exercise

'Warm-up' is performed before most types of exercise, and mode, duration and intensity differ depending on the subsequent performance task and facilities available. Differences also exist with regards to whether it is an active or passive 'warm-up', and in the level of specificity towards the competition event (Gray & Nimmo, 2001). It has been proposed that the physiological responses to 'warm-up' may be categorised into both temperature related and non-temperature related factors (Bishop, 2003a), and existing evidence suggests that temperature alone is not deterministic of the  $\dot{V}O_2$  response or performance (Burnley, Doust, & Jones, 2002; Gray et al., 2002; Koga et al., 1997). Therefore, it seems the terms 'priming' or 'prior exercise' may be more appropriate to use than 'warm-up'. There does not however appear to be any consistent priming strategy for each specific event, rather they follow a somewhat similar protocol that is intended to achieve the appropriate physiological responses. For example, priming of the muscular-tendinous unit (Avela, Kyrolainen, & Komi, 1999; Young & Behm, 2003; Young & Elliott, 2001), greater anaerobic priming for short-duration high-intensity performance (Heubert, Billat, Chassaing et al., 2005; Stewart & Sleivert, 1998), and increased aerobic priming for events of greater duration (Jones et al., 2003).

With respect to track cycling events, there is little peer-reviewed, documented evidence of current practice in priming for competitive performance. There are however some articles that have described the physiological requirements and power outputs in pursuit riding (Broker, Kyle, & Burke, 1999) as well as the processes involved in the attainment of world and Olympic pursuit titles (Schumacher & Mueller, 2002). The study of Schumacher and

Mueller (2002) would also appear to be of more relevance and to possess greater validity given that it is reported the practice of a world-record German pursuit team, and in contrast to many other peer reviewed academic journal articles, it provided not only the theoretical and physiological requirements of the event, but also the practical aspects of testing, training and priming for the event. The paper described different training 'zones', as determined in lactate-profiling assessments, and these zones were used not only for the training in preparation for the 2000 Olympic games, but also in the priming for the competition event. These zones were labelled according to their intensity and how they fit into the overall periodised plan, and included "Evolution" training (anaerobic threshold, high cadence, track-specific motor skill training), "Peak" training (low intensity, higher volume road training, ~50%  $\dot{VO}_{2peak}$ ).

Schumacher and Mueller (2002) reported that the team performed the same standardised 'warm-up' before each heat of the team-pursuit, consisting of 20 minutes riding "Basic" intensity, and two repetitions of five minutes (20 minutes recovery between) at "Evolution" training intensity, followed by another ten minutes at "Basic" intensity, finishing 20 minutes before competition. This priming strategy is of long duration (60 minutes), relatively low intensity (most around "Basic" intensity), and somewhat continuous in nature, and while it resulted in a world record performance, priming exercise of much higher intensity, shorter duration and of a more intermittent nature has been investigated (Billat, Bocquet et al., 2000) including some demonstrating improved performance than that of a lower intensity continuous priming exercise (Bishop et al., 2003). These investigations have provided some support of priming exercise which is quite in contrast to the current practice and the long held belief that the 'warm-up' must be of a low intensity and long duration in order to be effective. Additionally, there are also those that have investigated alternative 'warm-up' strategies, including priming a different muscle group (Fukuba, Hayashi, Koga, & Yoshida, 2002) and passive warming methods (Burnley, Doust, & Jones, 2002; Gray & Nimmo, 2001). This new evidence is intriguing, and has stimulated interest in the scientific approach to different priming strategies for high-intensity competition.

#### 3. Effects on physiological responses

Exercise results in many physiological responses which are dependant on the magnitude of that exercise. The emphasis placed on each of the different systems and mechanisms is also influenced by the nature of the exercise. There is a myriad of both peripheral and central acute responses to exercise, and the magnitude to which the various mechanisms respond, to a large extent, is outside the athlete's control during maximal effort exercise (such as during a race). However it is not uncontrollable during the 'warm-up' or priming phase of exercise. The different physiological mechanisms, and how they may be manipulated during the priming exercise to influence subsequent performance, will be discussed in the following sections. The physiological responses that are most widely investigated with regards to priming exercise and the ensuing exercise include oxygen uptake ( $\dot{VO}_2$ ), blood lactate concentration ([BLa]) and core temperature ( $T_c$ ). These are the focus of the first section of the review, with some review of other factors where appropriate (i.e. where they have been investigated in individual studies).

#### 3.1 Effects on Oxygen Uptake (VO<sub>2</sub>) and the VO<sub>2</sub> Kinetic Response

The study of oxygen uptake ( $\dot{V}O_2$ ) kinetics concerns the investigation and description of physiological mechanisms responsible for the dynamic  $\dot{V}O_2$  response to exercise and its subsequent recovery (Jones & Poole, 2005, p. 13). The magnitude of this  $\dot{VO}_2$  response and the magnitude to which the parameters describing this response are represented are dependant on both the mode and intensity of exercise. To date, several mechanisms have been proposed to determine the time-course of  $\dot{V}O_2$  at exercise onset. These include blood lactate concentration ([BLa]; Jones et al., 2003) and core temperature ( $T_C$ ; Bishop, 2003a), but perhaps most widely considered mechanisms with regards to the kinetic response would be O<sub>2</sub> transport (Hughson, Tschakovsky, & Houston, 2001) and utilisation (Walsh, Howlett, Stary, Kindig, & Hogan, 2005). However, the debate surrounding these variables in particular, and which is a greater determinant of the observable O2 response and ensuing performance, is unresolved (Tschakovsky & Hughson, 1999). Many researchers are in the process of providing evidence either in favour of one variable, or against the other (Bangsbo, Krustrup, Gonzalez-Alonso, Boushel, & Saltin, 2000; Bearden & Moffatt, 2001; Gerbino, Ward, & Whipp, 1996; Grassi, 2001, 2005; Grassi, Gladden, Samaja, Stary, & Hogan, 1998; Hughson et al., 2001; MacDonald, Naylor, Tschakovsky, & Hughson, 2001;

MacDonald, Pedersen, & Hughson, 1997; Tordi, Perrey, Harvey, & Hughson, 2003; Walsh et al., 2005). In terms of priming, identification of the physiological mechanism(s) that greatest influences the  $\dot{V}O_2$  response would be useful and could provide sports scientists with valuable information in their attempt to improve athletes' performance. In this regard, several experimental investigations have been undertaken in an effort to identify key mechanisms and determinants of the  $\dot{V}O_2$  response, especially those related to prior-exercise/priming and its subsequent physiological effects (e.g. Bangsbo et al., 2000; Bearden & Moffatt, 2001; Jones et al., 2003).

Proposed benefits of a priming intervention include increasing O<sub>2</sub> delivery, which in turn would accelerate  $\dot{VO}_2$  kinetics during high intensity exercise in comparison to the control condition (Billat, Bocquet et al., 2000; Burnley, Koppo, & Jones, 2005; Hajoglou et al., 2005). However, whilst some investigators refute this proposed accelerated  $\dot{VO}_2$  kinetic response (Jones et al., 2003), others suggest it is dependant on the intensity of the ensuing exercise (Burnley et al., 2000). If indeed there is an accelerated kinetic response, the ensuing effect may be a higher  $\dot{VO}_2$  earlier on in the exercise or a higher attainable peak  $\dot{VO}_2$  ( $\dot{VO}_{2peak}$ ), which in turn should result in an improved exercise performance. There are many different types of interventions and priming ('warm-up') strategies to enhance  $\dot{VO}_2$ kinetics, but for the purpose of this review, emphasis will be placed on determining the effects of different intensities of prior exercise and non-exercise 'passive warm-up' methods on exercise performance and its associated physiological response.

#### 3.1.1 Kinetic parameters of interest

There are several kinetic parameters that can be determined from the  $\dot{V}O_2$  response at exercise onset. As aforementioned, it is important to recognise that the occurrence, rate and/or magnitude of each parameter can be largely influenced by the intensity of the exercise.

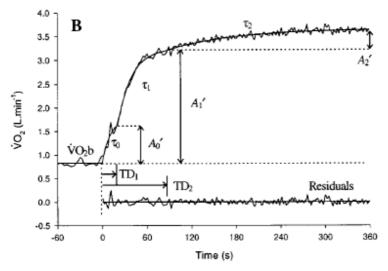
#### *Primary time constant (tau, \tau)*

In studies to date, regardless of the research focus, the major kinetic parameter of interest has been the phase II on transient time constant (tau,  $\tau$ ). Essentially,  $\tau$  indirectly reflects the

rate of change in  $\dot{V}O_2$  in muscle (measured at the mouth) at exercise onset (following the end of phase I, the cardiodynamic phase; Whipp, Ward, Lamarra, Davis, & Wasserman, 1982) and is evident regardless of exercise intensity. The phase II  $\tau$  also largely determines the magnitude of the O<sub>2</sub> deficit (Jones & Poole, 2005, p. 20). A decreased phase II  $\tau$  would appear to be beneficial to performance, given its potential to place less dependence on the anaerobic system, thus resulting in a decreased O<sub>2</sub> deficit. To reflect the overall  $\dot{V}O_2$ kinetic response, some studies have used the mean response time (MRT), a composite measure of the phase I and phase II  $\tau$ . The MRT can be calculated either as MRT = TD +  $\tau$ , or by a multiple-term exponential function of the weighted sum of these two variables (Jones & Poole, 2005; MacDonald et al., 1997). A shorter (i.e. faster)  $\tau$  (or MRT) following an intervention indicates an accelerated  $\dot{V}O_2$  kinetic response, reflecting a greater oxidative capacity in the active muscle, which causes a decrease in the O<sub>2</sub> deficit. An illustrative example of each of these kinetic parameters can be observed in *Figure 2-2*.

#### *VO*<sub>2</sub> *slow component*

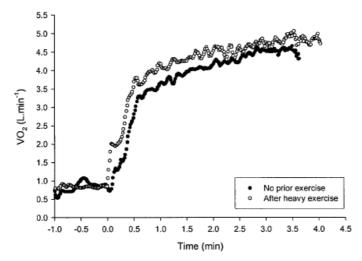
In recent years, the  $\dot{VO}_2$  slow component, in particular the mechanism determining it, has gained considerable interest (Barstow, 1994; Barstow, Buchthal, Zanconato, Cooper, & Cooper, 1994; Whipp, 1994). The slow component can be defined as an additional, slowly developing component rise in  $\dot{VO}_2$  (i.e. absence of, or delayed, steady-state  $\dot{VO}_2$ ) occurring between 800-100 seconds of heavy (above LT) exercise (Barstow, 1994; Whipp, 1994) to an extent which is above that expected from the  $\dot{VO}_2$ -work rate relationship (Marles, Mucci, Legrand, Betbeder, & Prieur, 2006; Paterson & Whipp, 1991 as cited in Burnley, Koppo & Jones, 2005). In an effort to determine its cause, it has been investigated with some of the potential determinants controlled, including increased muscle temperature (Koga et al., 1997), increased muscle acidosis (Burnley, Doust, & Jones, 2002), or increased type II muscle fibre recruitment (Krustrup et al., 2004). In *Figure 2-2*, the  $\dot{VO}_2$  slow component is displayed as  $A_2'$  - the amplitude of difference in  $\dot{VO}_2$  for that corresponding timeframe. In terms of prior exercise, any beneficial effects result in a reduction in  $\tau$  (or MRT) and/or the amplitude of the slow component.



*Figure 2-1:* Example of VO<sub>2</sub> response to heavy-intensity exercise fitted with triple exponential model.  $\tau$ VO<sub>2</sub>, effective VO<sub>2</sub> time constant;  $\tau_0$ ,  $\tau_1$ , and  $\tau_2$ , time constants; VO<sub>2</sub>(b), baseline VO<sub>2</sub> measured in the 3 min before onset of exercise;  $A_0'$ ,  $A_1'$ , and  $A_2'$ , amplitudes for exponential curves; TD<sub>1</sub> and TD<sub>2</sub>, time delays. (Burnley et al., 2000)

#### 3.1.2 Accelerated VO<sub>2</sub> kinetic response

Prior heavy (Burnley et al., 2000; Fukuba et al., 2002; Hajoglou et al., 2005; MacDonald et al., 1997) and low-intensity (Campbell-O'Sullivan, Constantin-Teodosiu, Peirce, & Greenhaff, 2002) exercise has been previously reported to decrease the MRT compared to trials with prior exercise of a lower intensity or no prior exercise, respectively (Table 2-1). Campbell-O'Sullivan et al. (2002) employed a protocol in which subjects cycled at a moderate intensity (75% VO<sub>2peak</sub>) for ten minutes preceded by either a low intensity (55%  $\dot{VO}_{2peak}$ ) ten minute warm-up (trial A), no warm-up (trial B), or at the moderate intensity for one minute preceded by either a low intensity ten minute warm-up (trial C) or no warmup (trial D). The purpose of the one minute trials was to allow for muscle biopsies to be taken for the spectrophotometric determination of adenosine triphosphate (ATP), phosphocreatine (PCr), creatine (Cr), glucose-6-phosphate (G-6-P), lactate acetylcarnitine and free carnitine. These variables will be discussed in section 3.3. It was found that trial A accelerated the MRT (31.3  $\pm$  2.4s; p < 0.05) when compared to trial B (45.1  $\pm$  2.8s) and resulted in a 35% reduction in O<sub>2</sub> deficit (1.5  $\pm$  0.2 vs. 2.3  $\pm$  0.3 L trials A and B, respectively; p < 0.05). Interestingly, it was also noted that there was a non-significant difference in the amplitude of the  $\dot{V}O_2$  from baseline ( $\dot{V}O_{2-B}$ ) to the new steady state ( $\dot{V}O_2$ ss) between the experimental trials. This was despite the reduction in MRT and O<sub>2</sub> deficit, as well as the adjusted  $\dot{V}O_{2-B}$  following the moderate exercise that was preceded by lowintensity exercise (trial A, 5.0 ± 0.7 L·min<sup>-1</sup>) in relation to moderate exercise with no preceding exercise (trial C, 4.2 ± 0.5 L·min<sup>-1</sup>). This suggests that, irrespective of what percentage of  $\dot{V}O_{2peak}$  was attained following the priming exercise and the MRT to achieve that change in  $\dot{V}O_2$ , there is a peak value of oxygen uptake that can be achieved in the subsequent exercise regardless of priming conditions. However, this was not consistent amongst all studies, with some evidence of an elevated  $\dot{V}O_2$  response throughout the trial following prior heavy exercise in contrast to a 'no-prior exercise' control condition, and similar baseline values, as displayed in *Figure 2-3* (Jones et al., 2003).



*Figure 2-2:* Breath-by-breath VO2 response. No prior exercise (control; *closed circles*) and prior heavy-intensity exercise (*open circles*). Note the elevated VO<sub>2</sub> throughout exercise after prior heavy-intensity exercise (Jones et al., 2003)

In a more recent study, the effects of different priming intensities on three kilometre cycling time trial (3km-TT) performance in well-trained cyclists were investigated by Hajoglou et al. (2005). As well as focussing on the performance gains associated with prior exercise, this study incorporated measures of  $\dot{V}O_2$  kinetics, with the primary focus on the MRT and some examination of  $\tau$  and TD. Three 'warm-up' conditions were investigated: 1) easy warm-up (EWU) consisting of five minute segments at 70, 80 and 90% of ventilatory threshold (VT); 2) hard warm-up (HWU) of five minute segments at 70, 80 and 90% of VT followed by three minutes at the respiratory compensation threshold (RCT), and 3) a control condition with no prior warm-up (CON). Hajoglou et al. (2005) reported that indeed there were differences in the  $\dot{V}O_2$  kinetic response depending whether priming exercise was performed prior to the 3km-TT or not, however, they failed to demonstrate

differences between EWU and HWU. The differing  $\dot{VO}_2$  kinetic responses were also accompanied by changes in contributions of aerobic and anaerobic energy sources. The study of Hajoglou et al. (2005) was similar in design (and outcomes) to another priming study investigating the  $\dot{VO}_2$  kinetics in cycling (Burnley et al., 2000) in aspects of both the methodologies and results.

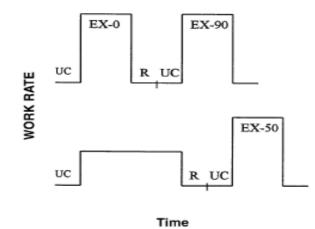
Burnley et al. (2000) also implemented heavy cycling in their investigation of the physiological effects of different priming intensities on subsequent cycling performance. However, in contrast to Hajoglou et al. (2005), Burnley et al. (2000) assessed the effects of priming exercise in a fixed time (and therefore total work) trial, rather than a 'performance-test' such as a 3km-TT. Additionally, moderate (80% of LT) and heavy (50% $\Delta$ ) prior exercise were the only two intensities investigated. The different conditions of the 'square-wave' design (e.g. *Figure 2-4*) included a) moderate exercise preceded by moderate exercise, b) heavy exercise preceded by moderate exercise, c) moderate exercise preceded by six minutes at 20W. The same  $\dot{VO}_2$  kinetics parameters of interest as Hajoglou et al. (2005) were examined, as well as the  $\dot{VO}_2$  slow component. The similarities and differences between the parameters of interest and methodologies of these two studies made for interesting comparisons of the physiological responses to the prior exercise.

Despite some minor differences in protocol/methods, both Hajoglou et al. (2005) and Burnley et al. (2000) reported a decreased MRT in the subsequent heavy exercise bout. Noteworthy also in the results of Hajoglou et al. (2005) was that the MRT was significantly shorter after both 'warm-up' conditions ( $45 \pm 10$ s and  $41 \pm 12$ s, EWU and HWU, respectively) than CON ( $52 \pm 13$ s), but that the MRT of EWU and HWU were not significantly different from each other. Such difference in MRT is likely to have contributed to the finding that power output attributable to aerobic sources was significantly less for CON at every 500 metre segment of the time trial in comparison to HWU, and at all but two 500 metre segments in comparison to EWU. This decreased contribution of aerobic energy sources would have increased the demand on the limited anaerobic sources (Nummela & Rusko, 1995). Regardless of the observed differences in  $\dot{VO}_2$  following each priming condition, the highest  $\dot{V}O_2$  reached at any 500 metre segment was not significantly different amongst conditions.

There was a non-uniform  $\tau$  response, and therefore MRT, displayed between the two studies, with regards to the comparisons between the greater and less intense exercise preparations. While both reported no difference in MRT between the moderate and heavy priming conditions when they were followed by moderate (Burnley et al., 2000) or maximal (Hajoglou et al., 2005) performance, Burnley et al. (2000) reported that the kinetic response in the heavy trial responded differently depending on whether or not it was preceded by moderate or heavy cycling. However, regardless of whether prior exercise was of moderate or heavy intensity, it had no significant effect on any  $\dot{V}O_2$  kinetic variables during subsequent moderate intensity exercise. This also included the no prior exercise condition, i.e. first bout of moderate exercise in conditions a) and c). However, although MRT in all prior exercise conditions were considerably longer for heavy than moderate trials, the MRT for heavy exercise preceded by heavy exercise (47.0  $\pm$  3.1s) was significantly less than the MRT for heavy exercise with moderate or no prior exercise (61.8)  $\pm$  4.5 and 65.2  $\pm$  4.1s respectively). This suggests that there was a greater amount of O<sub>2</sub> available earlier on in the exercise, and the participants were able to achieve the required  $\dot{V}O_2$  sooner due to the 'priming' effect of the prior heavy exercise. This would be expected to result in a decreased O<sub>2</sub> deficit, and therefore decreased dependence on anaerobic energy sources.

A reduction in the  $\dot{V}O_2$  slow component after prior heavy exercise has been described in a number of studies (Burnley, Doust, Ball et al., 2002; Burnley, Doust, & Jones, 2002; Koppo & Bouckaert, 2000; Koppo et al., 2003; Koppo et al., 2002; Marles et al., 2006; Sahlin, Sørensen, Gladden, Rossiter, & Pedersen, 2005). Burnley et al. (2000) suggested that the most important effect of prior heavy exercise was the consistently and significantly reduced amplitude of the  $\dot{V}O_2$  slow component (*Table 2-1*) which, along with non-significant changes in the phase II response profile, led to a significantly lower net end-exercise  $\dot{V}O_2$  in comparison to the initial bout of heavy exercise (*Table 2-2*). The possible reason for the inconsistency between the studies of Hajoglou et al. (2005) and Burnley et al. (2000) may be explained by the small difference in absolute intensities between the two

active 'warm-up' conditions in the Hajoglou et al. (2005) study, which caused no difference in MRT for EWU and HWU; whereas in the study of Burnley et al. (2000), the duration was constant and the intensity between conditions differed greatly.



*Figure 2-3:* Example of a square-wave transition protocol. UC unloaded cycling; R rest, EX-0, EX-50, EX-90 exercise (Koppo & Bouckaert, 2000)

Burnley et al.'s (2000) findings of a reduced MRT and slow component are in agreement with other studies involving cycling (MacDonald et al., 1997) and running (Billat, Bocquet et al., 2000), which suggests that the priming effect is similar for these modes of exercise involving large muscle groups of the lower body, even with the added contributions of the upper body and stretch shortening cycle component in running. MacDonald et al. (1997) compared cycling exercise at VT with prior exercise at half-way between VT and  $\dot{V}O_{2peak}$  $(50\%\Delta)$  and with no prior exercise, as well as investigating the effect of exercising in a hyperoxic state (i.e. with greater  $O_2$  availability) on the  $\dot{V}O_2$  kinetic response. Billat et al. (2000) compared priming exercise of a moderate and continuous nature to one of a highintensity intermittent nature, both of equal duration (20 minutes), on a continuous severe intensity (intermediate velocity between velocity at LT (4 mmol·L<sup>-1</sup>) and  $\dot{V}O_{2max}$ , v $\Delta 50$ ) run to exhaustion, which was designed to induce a  $\dot{V}O_2$  slow component. The moderate priming exercise was 'sub-LT' ( $W_{SUB}$ ) and was run at 50% v  $\dot{V}O_{2max}$  and followed by five minutes rest, and the high-intensity intermittent priming exercise was 'supra-LT' (W<sub>SUPRA</sub>) and consisted of alternating 30s at 100%  $v\dot{V}O_{2max}$  and 30s at 50%  $v\dot{V}O_{2max}$  and a longer rest of 15 minutes to allow for the recovery of  $\dot{VO}_2$  to resting levels.

Despite the differences in the design of their studies, both MacDonald et al. (1997) and Billat et al. (2000) demonstrated that prior exercise speeds the  $\dot{V}O_2$  response and significantly alters the  $\dot{V}O_2$  slow component. MacDonald et al (1997) stated that the faster on-transient kinetics with hyperoxia and prior exercise, as evidenced by a reduced MRT  $(53.4 \pm 2.6 \text{ versus } 37.8 \pm 3.3 \text{ and } 46.2 \pm 3.0 \text{s}, \text{ respectively}), \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ smaller O}_2 \text{ deficit } (1.55 \pm 0.20 \text{ smaller O}_2 \text{ sma$ versus  $1.15 \pm 0.13$  and  $1.46 \pm 0.09$  ml, respectively) and reduced slow component (93 ± 13 versus  $30 \pm 7$  and  $46 \pm 8$  ml, respectively), provided evidence that supply of O<sub>2</sub> contributes to control of tissue  $\dot{V}O_2$  for high-intensity exercise. Billat et al. (2000) also reported that both the  $\tau$  and the  $\dot{V}O_2$  slow component decreased significantly (40% and 45%, respectively) in the hard intermittent priming condition, compared with a continuous submaximal strategy. While VO<sub>2peak</sub> and HR<sub>peak</sub> were not significantly different following exercise from that of an initial incremental test to exhaustion, Billat et al. (2000) reported that seven out of eight runners developed a  $\dot{V}O_2$  slow component (291 ± 153 mL·min<sup>-1</sup>) after the light priming condition, versus only two out of eight after the hard intermittent priming condition (-143  $\pm$  271 mL·min<sup>-1</sup>) which suggests that an intensity- specific warm up is necessary to minimise physiological stress.

It is interesting to note that similar  $\dot{V}O_2$  kinetic responses (reduced  $\tau$ , MRT and slow component) occur in the Billat et al. (2000) study and other studies investigating  $\dot{V}O_2$ kinetics (Marles et al., 2006), which like MacDonald et al. (1997), were quite different in design and mode of exercise and utilised continuous cycling exercise. This provides some evidence that the mode (i.e. cycle versus run) and nature of the exercise (i.e. continuous versus high-intensity intermittent) and, in some cases, body position (i.e. seated when cycling versus upright when running) do not have a substantial influence on the  $\dot{V}O_2$ response to exercise, providing the relative workload is similar. However, when more extreme changes in body position are evident, i.e. from an upright to a prone (Rossiter, Ward, Kowalchuk et al., 2001) or supine position (Robergs, Costill, Fink et al., 1990), it is clear that the kinetic variables are affected. Indeed, there are studies that have demonstrated that heavy exercise in the supine position results in significantly slower  $\dot{V}O_2$ kinetics in comparison to upright cycling (Koga, Shiojiri, Shibasaki et al., 1999) and leg extension exercise (MacDonald, Shoemaker, Tschakovsky, & Hughson, 1998) of identical load. Additionally, MacDonald et al. (1998) also reported that the kinetics of leg blood flow were slower during supine than that of upright leg extension exercise, demonstrating association between alveolar  $\dot{V}O_2$  and blood flow in the active muscle(s). The collective results from these studies demonstrates that irrespective of the mode of exercise, the  $\dot{V}O_2$ kinetic response can significantly benefit from the priming exercise. This was demonstrated above by the consistently reduced  $\tau/MRT$  and  $\dot{V}O_2$  slow component. The greater availability of  $O_2$  and/or faster rate of  $O_2$  utilisation would surely benefit the ensuing heavy exercise through greater utilisation of oxidative pathways.

A recent study that was similar in design to that of Billat et al. (2000) was conducted by Judelson, Rundell, Beck, King and Laclair (2004) using moderately to well-trained  $(\dot{V}O_{2peak}: 62.7 \pm 6.7 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}; n = 10)$  cross country skiers. The subjects in that study, who were familiar with treadmill running, were required to perform an incremental running exercise to exhaustion preceded by either no prior exercise (MAX), by high-intensity submaximal run and a 20 minute rest period (discontinuous protocol; DC) or by high-intensity sub-maximal run with no rest period (continuous protocol; CON). The intensity of the submaximal runs in DC and CON were set at one stage higher than each individual's LT. Although there was no significant difference in VO<sub>2peak</sub> obtained between the two active priming conditions, the difference between MAX and DC approached significance (p =0.059; Table 2-2). This was shown by the trend of the decreased DC  $\dot{V}O_{2peak}$ , with half showing a significant decrease and half showing no difference in  $\dot{V}O_{2peak}$ . It is unclear whether an increase in subject numbers may have made this difference significant or removed any trend at all. However, due to the non-significant difference in VO<sub>2peak</sub> for DC, the authors concluded that the subjects exhibited greater aerobic capacity during the incremental (MAX) trial. The apparent reason for the dissimilarity between this study and Billat et al. (2000) in terms of the magnitude of effect of prior exercise, was the relative intensity of the prior exercise and the long rest period following the DC prior exercise. Prior exercise was terminated when subjects [BLa] went one  $mM \cdot L^{-1}$  above the value at the end of the first stage. Peak  $\dot{V}O_2$  values reached during the trial ranged between ~70% and ~90% of previously attained peak values. The athletes that performed at ~70%  $\dot{V}O_{2peak}$ would no doubt have found the task less fatiguing than those who recorded VO<sub>2</sub> values that were ~90%  $\dot{V}O_{2peak}$ . Similarly, performing at a moderate to high intensity (i.e. 70 – 90%  $\dot{V}O_{2peak}$ ) for some participants would have been fatiguing, whereas others would not have to as great an extent. These factors would add to the variance in results, removing the ability to detect significant effects for the ensuing trial. Unfortunately, it was not reported if there was any difference in the stage reached with each trial, and it is therefore impossible to speculate what effect prior exercise had on performance. It can be concluded, however, that if it is timed properly and rest time is sufficient (i.e. around 20 minutes in this case), prior exercise will not affect physiological data in the subsequent trial.

Recent studies (Bishop et al., 2001; Bishop et al., 2003) report findings consistent to those of Judelson et al. (2004) with regards to the non-significant difference in  $\dot{V}O_{2peak}$  between different prior exercise conditions following the performance trial. In these particular studies, two minute kayak tests preceded by 15 minutes of priming exercise at aerobic threshold, 50% $\Delta$ , anaerobic threshold, or a combination of 50% $\Delta$  and supramaximal sprint efforts were used (Bishop et al., 2001; Bishop et al., 2003). Prior exercise conditions had no effect on  $\dot{V}O_{2peak}$ , total  $\dot{V}O_2$  (or  $\dot{V}CO_2$ ), aerobic energy contribution, or accumulated oxygen deficit (AOD) during the trials, regardless of the increased aerobic response and elevated baseline  $\dot{VO}_2$  following the more intense priming conditions (*Table 2-2*). These findings, in particular an unchanged AOD, imply that the  $\dot{V}O_2$  kinetic response in near supra-maximal kayak performance was not influenced differently between priming conditions of different intensity and nature (intermittent versus continuous). Although the  $\dot{V}O_{2peak}$  response was consistent among these three studies, there appears to be some discrepancies with regards to the actual  $\dot{V}O_2$  kinetic response. This may warrant further investigation into the effects of priming exercise on the  $\dot{V}O_2$  kinetic response in maximal and supramaximal exercise. It is possible that exercise that is 'heavy' but still submaximal (i.e. below  $\dot{V}O_{2peak}$ ) may exhibit  $\dot{V}O_2$  kinetic responses which may in fact be influenced by priming exercise, or 'warm-ups', of differing intensities, differently, or to a different magnitude, than that of supramaximal exercise. This in-turn may affect performance differently than that which has been shown by Bishop et al. (2001; 2003) in supramaximal two minute exercise.

Judelson et al.'s (2004) more recent study coheres well with the work of Bishop et al. (2001; 2003) despite differing in terms of rest duration (e.g. 20 minutes versus five minutes) between priming exercise and performance trial, and Bishop et al. (2001; 2003) using a much greater intensity during the performance trial (e.g. two minutes 'all-out' TT versus T<sub>lim</sub> at LT + one stage). Given the high demand on anaerobic energy systems during the two minute kayak TT's, and therefore potentially increased AOD in comparison to that which may be expected during exercise just above LT (Craig, Norton, Convers et al., 1995), it could be expected that a greater intensity and shorter duration performance trial would benefit much more from the early accelerated kinetic response. This would serve to increase the contribution of aerobic energy pathways early in exercise and preserve anaerobic energy stores for later in the trial (Hajoglou et al., 2005; Nummela & Rusko, 1995). There are however several limitations to the studies of both Judelson et al. (2004) and Bishop et al. (2001; 2003). First, although neither  $\tau$  nor the slow component were directly measured, the proposed increased aerobic contribution was not obvious in either kayak study, as evidenced by a non-significant difference in AOD between any conditions for either study. Secondly, although the AOD can provide some indication of whether there has been greater availability/utilisation of O<sub>2</sub> dependant energy pathways, the kinetic variables that best describe this process of an accelerated/changed  $\dot{VO}_2$  response ( $\tau/MRT$ and  $\dot{V}O_2$  slow component) were not calculated, or at least reported in these studies.

Accelerated  $\dot{V}O_2$  kinetics, as evidenced by reduced MRT or  $\tau$ , is not evident in all studies with respect to differing intensities of prior exercise (Wilkerson et al., 2004) even with the presence of a reduced slow component (Burnley, Doust, Ball et al., 2002) or significantly increased HR in the higher intensities (Jones et al., 2003; Koga et al., 1997). This lack of acceleration in  $\dot{V}O_2$  kinetics would seem to result in an increase in end-exercise attained  $\dot{V}O_{2peak}$  with heavy prior exercise (see *Figure 2-3*). However, such a conclusion will not be strong considering only two studies demonstrating no change in MRT but increased  $\dot{V}O_{2peak}$  have been reported (Jones et al., 2003; Wilkerson et al., 2004). Previously mentioned studies with reduced  $\dot{V}O_2$  slow component still demonstrated increased endexercise  $\dot{V}O_2$  (Burnley, Doust, & Jones, 2002). There is also data showing that priming exercise of greater intensity can speed the  $\dot{V}O_2$  kinetics, however not via a reduced primary  $\tau$ , but rather through a reduction in the MRT and  $\tau$  (Bearden & Moffatt, 2001). Bearden et al. (2001) also suggested that the most important response to the priming may be the decrease in the amplitude and increase in the TD of the slow component (i.e. there was a smaller slow component of which the onset was delayed) (Table 2-1; Bearden & Moffatt, 2001).

As mentioned above, the  $\dot{VO}_2$  slow component relates to a rise in  $\dot{VO}_2$  above what would be predicted from the VO<sub>2</sub>/work rate relationship (Marles et al., 2006; Paterson & Whipp, 1991 as cited in Burnley, Koppo & Jones, 2005). A useful study was performed by Marles et al. (2006) to investigate this association between the  $\dot{VO}_2$  slow component and VO<sub>2</sub>/work rate relationship, in which tests combining 'square-wave' protocols with incremental ramp assessments compared with traditional 'square-wave' protocols were used. While it was not a true 'performance' test, it did allow for investigation of the  $\dot{VO}_2$ slow component and VO<sub>2</sub>/work rate relationship during maximal exercise preceded by either six minutes of heavy (90% VO<sub>2max</sub>) or no prior exercise, and also its effects on peak power (W<sub>peak</sub>). Of interest from this study was that, while a decrease in slow component was again displayed during the second bout of heavy exercise along with a significantly different VO<sub>2</sub>/workrate relationship, that same VO<sub>2</sub>/workrate relationship was not affected by prior exercise in the incremental test to exhaustion, despite a decreased W<sub>peak</sub>. These results demonstrated that there was no relationship between the  $\dot{V}O_2$  slow component and the disproportionate increase in  $\dot{V}O_2$  in incremental exercise (generally at the higher stages, which the authors labelled the "extra  $\dot{V}O_2$ ") than would be expected from the  $\dot{V}O_2$ /workrate relationship. This implies that the physiological mechanisms that control the  $\dot{V}O_2$  slow component, whatever they may be, are not necessarily the same as those controlling the VO<sub>2</sub>/workrate relationship during incremental exercise, as may be the case with the steady-state exercise in square wave protocols. Additionally, Marles, Legrand, Blondel et al. (2007) demonstrated in a subsequent study that, while the  $\dot{V}O_2$  slow component and the "extra  $\dot{V}O_2$ " relationship were significantly related, these two factors responded differently following a high-intensity interval training intervention, with a significant decrease in the "extra  $\dot{V}O_2$ " but not the  $\dot{V}O_2$  slow component. This data again provides of at least some evidence of disassociation between these two factors, as did the

reversed training-induced adaptations to the "extra  $\dot{V}O_2$ ", but no increase in the  $\dot{V}O_2$  slow component following six weeks of detraining.

In summary, some studies show that an accelerated  $\dot{V}O_2$  kinetic response occurs as the result of the greater intensity priming condition, as evidenced by a reduced  $\tau/MRT$  and the reduction in the amplitude (e.g. Burnley et al., 2000; Hajoglou et al., 2005; MacDonald et al., 1997) (and sometimes complete deletion) of the slow component (Billat, Bocquet et al., 2000). Conversely, some studies were in disagreement with this finding however, with some showing no reduction in MRT or  $\tau$  (Burnley, Doust, Ball et al., 2002; Jones et al., 2003; Koga et al., 1997; Wilkerson et al., 2004). There are inconsistent findings with regard to whether there is any change in the end exercise  $\dot{V}O_2$  between conditions, with some reporting a significant difference following the performance trial between conditions (Burnley, Doust, & Jones, 2002; Koppo & Bouckaert, 2000; Koppo et al., 2003; Wilkerson et al., 2004) and many reporting no change (Bangsbo, Krustrup, Gonzalez-Alonso, & Saltin, 2001; Billat, Bocquet et al., 2000; Bishop et al., 2001; Bishop et al., 2003; Burnley, Doust, Ball et al., 2002; Burnley et al., 2000; Hajoglou et al., 2005; Judelson et al., 2004; Koppo et al., 2002). Performance trials of maximal and supramaximal intensities are less likely to have a change in peak attained  $\dot{V}O_2$  following the trial, as it is expected that  $\dot{V}O_{2peak}$  will be achieved, irrespective of how one prepares or 'primes' themselves for it. Whereas those studies that did report significant difference mostly consisted of submaximal 'square-wave' designs, with constant total work in the performance trial between conditions. This would in theory allow for a greater variation in the peak attained  $\dot{VO}_2$ values, as participants would still be exercising well within their aerobic capabilities, in contrast to an intensity close to  $\dot{V}O_{2peak}$ , which would be expected to result in decreased variation, in addition to quicker attainment, of peak values. One may speculate that an accelerated  $\dot{V}O_2$  kinetic response would appear beneficial as it could result in reduced dependency on the limited anaerobic energy pathways, as indicated by an observed reduction in AOD. Again however, AOD was not consistent in all studies, with evidence of a reduced AOD from heavy prior exercise (Gerbino et al., 1996), while others report no difference between conditions in studies involving heavy (Judelson et al., 2004) and supramaximal (Bishop et al., 2001; Bishop et al., 2003) performance trials. Together, these findings suggest that a priming strategy of greater intensity is physiologically beneficial and

results in greater  $O_2$  availability and/or utilisation earlier to the onset of exercise. However, due to some conflicting findings, it is unclear what is optimal to elicit the desired  $\dot{V}O_2$  kinetic response and further work to determine the optimal priming intensity is clearly required.

Reference	Participants	<b>Priming Strategy</b>	Effect on MRT/T	Effect on Slow Component
MacDonald et al. (1997)	M = 4, $FM = 3$ : $MT$	4min 25W, 10min 50%∆, 6min 25W no prior exercise	Reduced MRT (-13.5%, -7.2 s)	Significantly reduced (-50.5%, -47 ml·min <sup>-1</sup> )
Billat et al. (2000)	8 M ET: 60 ± 16km/week training, not familiar with severe interval training	20min AT 50% vVO <sub>2max</sub> , 5min rest, T <sub>lim</sub> at 50%Δ 20min alternating 30s at 100% & 30s at 50% vVO <sub>2max</sub> , 15min rest, T <sub>lim</sub> at 50%Δ	- Reduced τ (-45.1%, -23 s)	- Significantly reduced (-149.1%, -434 ml·min <sup>-1</sup> )
Jones et al. (2003)	7 M: MT	6min 50%∆ no prior exercise	NS	NS
Burnley, Doust, & Jones (2002)	9 M: MT	<ol> <li>heavy exercise, HE: 2 x 6min cycle, 50%∆</li> <li>2) sprint exercise: 30s at 17km/hr (120rpm) cycle preceding HE</li> <li>3) 40min passive warming lower limbs, precededing HE</li> </ol>	NS	Significantly reduced (-59.6%, -280 ml·min <sup>-1</sup> ) Significantly reduced (-42.6%, -200 ml·min <sup>-1</sup> ) NS
Koga et al. (1997)	7 M: MT	Normal muscle temperature Elevated muscle temperature	NS	NS
Koppo & Bouckaert (2000)	12 (9M, 3FM): MT	Ex-0: 6min at 90% VO <sub>2peak</sub> with no prior exercise (control) EX-90: 2nd bout of 6min at 90% VO <sub>2peak</sub> EX-50: 12.1 $\pm$ 0.8min at 50% VO <sub>2peak</sub> followed by 6min at 90% VO <sub>2peak</sub>		- Significantly reduced (-59.3%, -143 ml·min <sup>-1</sup> ) Significantly reduced (-28.2%, -68 ml·min <sup>-1</sup> )

Reference	Participants	Priming Strategy	Effect on MRT/T	Effect on Slow Component
		1) No warm-up, CON	Greater MRT	ı
Hajoglou et al. (2005)	8 M WT cyclists & triathletes,	2) Easy warm-up, EWU: 5min segments at 70, 80 & 90% VT	Reduced MRT from CON (-13.5%, -7 s)	ı
	local class	3) Hard warm-up, HWU: 5min segments at 70, 80 & 90% VT, + 3min at RCT, 6min rest	Reduced MRT from CON (-21.2%, -11 s)	·
		a) 6min 80% LT preceded by 6min 80% LT	NS	NS
-		b) 6min 50%∆ preceded by 6min 80% LT	NS	NS
Burnley et al. (2000)	8M, 2FM: MI	c) 6min 80% LT preceded by 6min 50%Δ	NS	NS
		d) 6min 50% $\Delta$ preceded by 6min 50% $\Delta$	Reduced MRT [τ] (-27.9 [-21.6]%, -18.2 [-9.7] s)	Significantly reduced (-63.0%, -170 ml·min <sup>-1</sup> )
Wilkerson et al. (2004)	7 M: MT, recreationally active	no prior exercise, T <sub>lim</sub> 105% VO <sub>2peak</sub> 3x30s all-out cycling & 15min rest, T <sub>lim</sub> 105% VO <sub>2peak</sub>	NS	1 1
		6min leg cycling (90% cycle VO <sub>2peak</sub> ), no prior exercise (LE-C)		- - - - - - - - - - - - - - - - - - -
Koppo et al. (2003)	10 M: MT - WT	2nd bout of heavy leg cycle exercise: LE-L	NS	E-C [& LE-A] LE-C [ $\& LE-A$ ] (-41.6 [30.3] %, -273 [167]
		heavy leg cycle preceded by heavy arm crank (90% arm VO <sub>2peak</sub> ): LE-A		Significantly less than LE-C (-16.1%, -106 ml·min <sup>-1</sup> )

Reference	Participants	Priming Strategy	Effect on MRT/t	Effect on Slow Component
Koppo et al. (2002)	6 PE students: MT	6min cycle at 90% VO <sub>2peak</sub> , no prior exercise 2nd bout of 6min cycle at 90% VO <sub>2peak</sub>	1 1	- Significantly reduced (-56.7%, -315 ml·min <sup>-1</sup> )
		passive warming, 6min cycle at 90% VO <sub>2peak</sub>		NS
Campbell-O'Sullivan et al. (2002)	7 M: MT, recreationally active	Trial A: 55% VO <sub>2max</sub> cycle 10min, 3min passive rest, 10min 75% VO <sub>2max</sub>	Reduced MRT (-30.6%, -13.8 s)	NS
		Trial B: cycle 10min 75% VO <sub>2max</sub>		
Burnley, Doust, Ball et al. (2002)	8 M: MT	Square-wave cycling exercise: 2 x 6min heavy exercise (70%Δ between VT & VO <sub>2peak</sub> )	NS	Significantly reduced (-49.4%, -390 ml·min <sup>-1</sup> )
		L1-ex: 6min supra-LT leg cycling (LT + $\Delta 50\%$ ), no prior exercise	,	
Fukuba et al. (2002)	6 M	L2-ex: 2nd bout supra-LT leg cycling	Reduced MRT [7] (-23.9 [-29.6]%, -12.3 [-15.5] s)	Significantly reduced (-47.5%, -198 ml·min <sup>-1</sup> )
		A1-ex to A2-ex: 6min heavy arm cranking (~1W/kg body weight), 6min supra-LT leg cycling	Increased MRT [r] from L2-ex only (20.9 [28.8]%, 10.3 [14.9] s)	NS
Rossiter et al. (2001)	M 7	Square-wave knee extensor exercise: 2 x 6min heavy exercise	Reduced $\tau$ (-%, - s)	Significantly reduced (-60.3%, -4.1 ml·min <sup>-1</sup> )

Reference	Participants	<b>Priming Strategy</b>	Effect on MRT/T	Effect on Slow Component
Marles et al. (2006)	10 M competitive sprinters	Square-wave cycling exercise: 2 x 6min heavy exercise (90% VO <sub>2max</sub> )	·	Significantly reduced (-79.9%, -269.8 ml·min <sup>-1</sup> )
Bearden & Moffatt	TW. W 9	Square-wave cycling exercise: 2 x 10min heavy exercise (30%Δ)	Reduced MRT in 2 <sup>nd</sup> bout (-20.2%, -11.2 s)	Reduced in 2nd bout (-44.8%, -130 ml·min <sup>-1</sup> )
(2001)	1 M 1 M	Square-wave cycling exercise: 2 x 10min moderate exercise (90% GET)	No significant difference between $1^{st} \& 2^{nd}$ bouts	No significant difference between 1 <sup>st</sup> & 2 <sup>nd</sup> bouts
Sahlin et al. (2005)	9 M: MT, recreationally active	heavy cycling: 10min at 75% VO <sub>2peak</sub> , no prior exercise 2nd bout heavy cycling: separated by 2min rest, 2 x 2min & 1 x T <sub>lim</sub> at 110% VO <sub>2peak</sub> & 3min rest	No significant difference	Significantly reduced (-104.4%, -263 ml·min <sup>-1</sup> )

GET: gas exchange threshold

RCT: respiratory compensation threshold VT/LT: ventilatory/lactate threshold

50% A: half-way between LT & VO<sub>2peak</sub> (Burnley et al., 2002, Koga et al., 1997), GET & VO<sub>2peak</sub> (Jones et al., 2003), or VT & VO<sub>2peak</sub> (MacDonald et al., 1997)

MT, ET, WT: moderately, endurance and well-trained, respectively

# 3.1.3 Passive and active prior warming, 'warming-up' with different muscle groups and end-exercise VO<sub>2</sub>

To determine whether the effects of priming exercise are related to central (i.e. heart rate, T<sub>C</sub>, VO<sub>2</sub>, [BLa]) or peripheral (i.e. muscle temperature (T<sub>M</sub>) and muscle lactate/acidity) responses, investigators have compared the effect of upper and lower body prior exercise on subsequent exercise in the lower body (Fukuba et al., 2002), while others have directly investigated of effect of increased muscle temperature on the kinetic response (Koga et al., 1997). The 'warming up' of the muscle in preparation for exercise is traditionally achieved through exercise. However, increasing the temperature of the local musculature can also be achieved through passive warming. This is achieved using hot water perfused pants (Koga et al., 1997), warm/hot water baths (Burnley, Doust, & Jones, 2002), or heat packs in combination with heat creams and rubs (Koppo et al., 2002). Indeed, several studies have been conducted which have investigated how passive warming affects physiological responses and performance in comparison to active (exercise) 'warm-ups' in an attempt to confirm or deny the proposed possibility of the benefits of 'warm-up' being related to increases in core  $(T_C)$ and skin (T<sub>s</sub>) temperature (Burnley, Doust, & Jones, 2002; Koga et al., 1997; Koppo et al., 2002).

To date, the majority of studies show that although an increase in  $T_M$  often accompanies the priming exercise (Mohr et al., 2004) and it's associated  $\dot{VO}_2$  kinetic response (Koga et al., 1997), it is only an *association*, and not the *cause* of the altered kinetics. For example, two of these studies showed that passive warming of the legs did not have the same magnitude of effect on  $\dot{VO}_2$  kinetic variables as the prior heavy exercise (Burnley, Doust, & Jones, 2002; Koppo et al., 2002). During these studies it was ensured that both passive and active warming resulted in equal increases in  $T_M$  prior to exercise. The findings demonstrated that the reduction in  $\tau$ , MRT or  $\dot{VO}_2$  slow component are not directly in response to the greater increase in  $T_M$  per se, as previously suggested (for examples see: Bishop, 2003a; Mohr et al., 2004), but as a result of more intense muscular contractions in the greater intensity prior exercise. A contribution/influence from the central circulation should not be ignored since it has also been shown that an active 'warm-up' produced the greatest heart rate (HR) response throughout exercise which is likely to have increased O<sub>2</sub> transport processes (Burnley, Doust, Ball et al., 2002; Koppo et al., 2002). Taking these findings into consideration, it may not be appropriate to use the term 'warm-up', as it is not just an increase of the  $T_C$  or  $T_M$  that is the goal (or outcome) of the 'warm-up'; rather a 'priming' of the whole physiological system, including  $\dot{V}O_2$ , [BLa] (as discussed in greater detail below) and temperature.

Interestingly, the pulmonary  $\dot{V}O_2$  kinetic response (measured at the mouth) to exercise is reflective of actual  $\dot{V}O_2$  within the active muscle ( $O_2$  utilised in the muscle). This fact has generated interest in the pulmonary  $\dot{V}O_2$  kinetic response to priming with a different muscle group. One such 'square-wave' protocol study, with this muscle  $\dot{V}O_2$ concept in mind, utilised both arm crank and cycle exercise prior to subsequent cycle exercise (Koppo et al., 2003). It was reported that, while HR was significantly higher following the second bout of exercise in both prior exercise conditions than following the control (i.e. first bout of cycling exercise), the reduction in the  $\dot{V}O_2$  slow component in the cycling trial was greater when preceded by heavy cycling than heavy arm crank exercise (Koppo et al., 2003). This could be interpreted that the effect of prior exercise on the speeding of  $\dot{V}O_2$  kinetics is greater when the prior exercise is performed with the same muscle group. Interestingly, prior heavy cycling resulted in significantly reduced  $\dot{V}O_{2peak}$  than no both prior exercise (see *Figure 2-3* for an example of the elevated  $\dot{V}O_2$ response) and prior arm crank exercise (as displayed in Table 2-2). The authors stated that the reduced end-exercise  $\dot{V}O_2$  in the prior cycling condition in comparison to the other conditions was likely a result of the reduced amplitude of the  $\dot{V}O_2$  slow component. This  $\dot{V}O_2$  slow component reduction may reflect that residual metabolism was present, as evidenced by higher baseline  $\dot{V}O_2$ , which may have caused the true magnitude of change in end  $\dot{V}O_2$  values to be obscured.

To investigate if the accelerated  $\dot{V}O_2$  kinetics were caused by an increase in [BLa], another study was carried out using different muscle groups in the priming and subsequent exercise, which elicited equal [BLa] (Fukuba et al., 2002). The  $\dot{V}O_2$  kinetic responses were compared during six minutes of supra-lactate threshold (LT) cycling following 1) no prior exercise (L1-ex); 2) a set of cycling at the same supra-LT intensity (LT +  $\Delta$ 50%; range: 190-240 watts) and six minutes active recovery of unloaded cycling (L2-ex); or 3) six minutes of heavy arm cranking (~1 watts·kg<sup>-1</sup> body weight; range: 60-80 watts) and six minutes active recovery of unloaded cycling (A2-ex; where A1-ex was the initial set of arm cranking and A2-ex was the subsequent heavy cycling). The researchers declared that the prior bout of high intensity cycling (L2-ex) caused a

significantly greater acceleration in  $\dot{V}O_2$  kinetics than the other three conditions, as evidenced by the effect of  $\tau$  causing a decreased MRT. When implementing a double exponential model in an attempt to describe the kinetics more precisely, it was found that the  $\dot{V}O_2$  slow component (calculated by the change in  $\dot{V}O_2$  from the third to sixth minute,  $\dot{V}O_2$  (6-3)) was attributed to causing the difference in the overall kinetics between L1-ex and L2-ex, due to its significant reduction in L2-ex. This is in contrast to the change in  $\tau$ , as it would appear when using the mono-exponential, which was accounted for in the primary component, where the primary component  $\tau$  ( $\tau_P$ ) was not significantly different between conditions. Fukuba et al. (2002) also suggested that in the second bout (i.e. L2-ex) the 'aided'  $\dot{V}O_2$  may be credited to improved  $O_2$  transport to the exercising musculature, greater  $O_2$  utilisation in exercising muscles, and/or their interaction.

The conclusion regarding  $O_2$  transport and utilisation in the active muscles is likely due to the observation that prior exercise of comparative intensity by a different muscle group did not aid in the acceleration of the phase II  $\tau$ . This is consistent with the conclusion given in the review by Whipp (1994) that the mechanisms affecting the  $\dot{V}O_2$ kinetics, the slow component in particular, are influenced by the exercising limbs due to the increase in muscle fibre recruitment. This hypothesis of recruitment of additional muscle fibres affecting the slow component during heavy exercise has been further demonstrated by the emergence of a slow component (which was reduced or nonexistent during moderate exercise) being accompanied by additional type II fibre recruitment (Krustrup et al., 2004). Additionally, it has been proposed that because vasodilation increases the  $O_2$  availability (and therefore oxidative metabolism) to both active and inactive muscle fibres are recruited, they will have sufficient  $O_2$ , potentially accelerating  $\tau$  (Bearden & Moffatt, 2001).

It is worthy of note however that it has also been reported that induced muscle alkalosis (i.e. metabolic not respiratory alkalosis) actually accelerates the  $\dot{V}O_2$  kinetic response (25% reduction in  $\tau$ ) also during heavy (87%  $\dot{V}O_{2peak}$ ) exercise (Zoladz, Szkutnik, Duda, Majerczak, & Korzeniewski, 2005). The authors stated that increased metabolic alkalosis, in contrast to respiratory alkalosis, increases the concentration of CO<sub>2</sub> and HCO<sub>3</sub>, but that it was unclear how this would influence the intracellular pH at the onset

of exercise. It is possible that the resulting increases in respiratory  $CO_2$  and  $HCO_3$  may be associated with the accelerated  $\dot{V}O_2$  kinetic response as caused by increased [BLa] which was proposed previously by other researchers (Bishop et al., 2001)

The study of Bearden & Moffatt (2001) was interesting in that it not only reported the change in HR and  $\dot{V}O_2$  over different conditions, but also applied similar kinetic models to HR that are employed when examining the  $\dot{V}O_2$  response (i.e.  $\tau$ , TD and slow component). This not only allowed a more in depth description of the HR response, but also displayed HR and  $\dot{V}O_2$  kinetics disassociation, providing evidence that the HR response (i.e.  $O_2$  delivery) does not always mirror that of the  $\dot{V}O_2$  response. For example, although both  $\dot{V}O_2$  and HR kinetics displayed a decreased amplitude and increased TD (which was not significantly different between HR and  $\dot{V}O_2$ ) of the slow component, the same could not be displayed with respect to  $\tau$ . While the off-transient  $\tau$  differed between exercise transitions for HR, there were no significant differences between conditions for the  $\dot{V}O_2$  off-transient  $\tau$ . For both the on- and off-transient kinetics, in nearly all instances significant differences were apparent between HR and the corresponding  $\dot{V}O_2 \tau$  values. The authors stated that this disassociation emphasises the importance of local blood flow control in matching  $O_2$  demand and delivery during non-steady-state exercise.

It is interesting to note that while a disassociation between the HR and  $\dot{V}O_2$  kinetics has been made apparent (Bearden & Moffatt, 2001), evidence also exists that the elevated cardiac output and HR following heavy prior exercise was related to a decreased  $\tau$ , and therefore providing evidence that accelerated kinetics may be related to  $O_2$  transport (Tordi et al., 2003). The researchers stated that this estimated increase in cardiac output, along with the more rapid adaptation in oxidative metabolism (i.e. decreased  $\tau$ ) indirectly indicated that the muscle blood flow was increased as a consequence of the greater metabolic demand of the greater intensity prior exercise. Therefore, prior heavy exercise increases oxygen delivery through an increase in cardiac output, which again provides evidence of at least some oxygen delivery limitation in  $\dot{V}O_2$  during heavy exercise.

In summary, most studies demonstrated that performing higher intensity in comparison to lower intensity or no prior exercise will result in an accelerated  $\dot{V}O_2$  kinetic response,

whereas  $\dot{V}O_{2peak}$  is often unaffected. Based on evidence provided by the majority of studies presented in this review, the two variables that appear to be affected the greatest are the MRT and amplitude of the  $\dot{V}O_2$  slow component, which are both significantly decreased with an increase in priming exercise intensity. This accelerated  $\dot{V}O_2$  kinetic response will mean that more oxygen is available sooner after the onset of exercise and accordingly the oxygen deficit is reduced, thereby increasing the contribution of the oxygen-dependant energy systems and decreasing contribution of the fatiguing anaerobic systems.

<i>Table 2-2</i> : Effects on end-exercise peak attained VO <sub>2</sub>	cise peak attained VO <sub>2</sub>			
Reference	Participants	Priming Strategy	Effect on VO <sub>2peak</sub> Between Intensities	Effect on VO <sub>2peak</sub> from Incremental/Control
Billat et al. (2000)	8 M, ET: 60 ± 16km/week training volume	20min AT 50% vVO <sub>2max</sub> , 5min rest, T <sub>lim</sub> at 50% $\Delta$ 20min alternating 30s at 100% & 30s at 50% vVO <sub>2max</sub> , 15min rest, T <sub>lim</sub> at 50% $\Delta$	NS	NS
Jones et al. (2003)	7 M: MT	No prior exercise 6min 50%Δ, T <sub>lim</sub> at 100, 110 & 120% VO <sub>2peak</sub>	- Significantly greater at 120% VO <sub>2peak</sub> (5.8%)	NS
Judelson et al. (2004)	10 M cross country skiers: WT	incremental run to fatigue preceded by HI submax run & 20min rest period incremental run to fatigue preceded by HI submax run with no rest	NS	NS
Bishop et al. (2003)	7 M, 500m kayak, state rep	continuous: 15min 50% $\Delta$ intermittent: same 1st 10mins, 5x10s sprints 200% VO <sub>2max</sub> , with 50s recovery at AeT	NS	1 1
Burnley, Doust, & Jones (2002)	9 M: MT	<ol> <li>heavy exercise, HE: 2 x 6min cycle, 50%∆</li> <li>sprint exercise: 30s at 17km/hr cycle preceding HE</li> <li>40min passive warming lower limbs, preceding HE</li> </ol>	Significantly less than 2) Significantly greater than 1) (3.3%, 0.11 L·min <sup>-1</sup> ) NS	NS

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Reference	Participants	<b>Priming Strategy</b>	Effect on VO <sub>2peak</sub> Between Intensities	Effect on VO <sub>2peak</sub> from Incremental/Control
		EX-0: 6min 90% VO <sub>2peak</sub> , no prior exercise	Greater than EX-50 $(2.7\%, 0.09 \text{ L} \cdot \text{min}^{-1})$	NS
Koppo & Bouckaert	12 (9M, 3FM): MT	EX-90: 2nd bout of 6min 90% VO <sub>2peak</sub>	NS	NS
		EX-50: 12min 50% VO <sub>2peak</sub> preceding 6min 90% VO <sub>2peak</sub>	Lower than EX-0	Significantly lower
		Easy warm-up, EWU: 5min segments at 70, 80 & 90% VT		
Hajoglou et al. (2005)	8 M, WT cyclists & triathletes, local class	Hard warm-up, HWU: 5min segments at 70, 80 & 90% VT, + 3min at RCT, 6min rest	NS	NS
		a) 6min 80% LT preceded by 6min 80% LT		VO <sup>2</sup> absolute value at end of
Burnley et al. (2000)	10 (8M, 2FM): MT	<ul> <li>b) 6min 50% Δ preceded by 6min 80% LT</li> <li>c) 6min 80% LT preceded by 6min 50% Δ</li> </ul>	NS	exercise 84% VO <sub>2peak</sub> (75- 95% VO <sub>2peak</sub> ), significance
		d) 6min 50% $\Delta$ preceded by 6min 50% $\Delta$		not stated
		15min AeT		
Bishop et al. (2001)	8 elite kayak squad members	15min 50%Δ	NS	NS
		15min AnT		
				200 + 602 V/O
	7 males: MT, recreationally	no prior exercise, T <sub>lim</sub> 105% VO <sub>2peak</sub>	Significantly less	00 ± 0.0 V Ozpeak, Sugnificance not stated
W like soli et al. (2004)	active	3x30s all-out cycling & 15min rest, T <sub>lim</sub> 105% VO <sub>2neak</sub>	Significantly greater (7.0%, 0.25 L·min <sup>-1</sup> )	$94 \pm 10\%$ VO <sub>2peak</sub> , significance not stated

Reference	Participants	Priming Strategy	Effect on VO <sub>2peak</sub> Between Intensities	Effect on VO <sub>2peak</sub> from Incremental/Control
		6min leg cycling (90% cycle VO <sub>2peak</sub> ), no prior exercise (LE-C)	Significantly greater than LE-C (1.6%. 0.064 L.min <sup>-1</sup> )	
Koppo et al. (2003)	10 M: MT - WT	2nd bout of heavy leg cycle exercise: LE-L heavy leg cycle preceded by heavy arm crank (90% arm VO <sub>2peak</sub> ): LE-A	Significantly less Significantly greater than LE-C (2.3%, 0.093 L·min <sup>-1</sup> )	Significance not stated
		6min cycle at 90% VO <sub>2peak</sub> , no prior exercise		
Koppo et al. (2002)	6 PE students: MT	2nd bout of 6min cycle at 90% VO <sub>2peak</sub>	NS	Significance not stated
		passive warming, 6min cycle at 90% $VO_{2peak}$		
	My	3min knee extension, no prior exercise	NC	
Daulgsou et al. (2001)	MT O	2nd bout of 3min knee extension	2	·
Burnley, Doust, Ball et al. (2002)	8 M: MT	Square-wave cycling exercise: 2 x 6min heavy exercise (70%∆ between VT & VO <sub>2peak</sub> )	NS	ı

VT/LT: ventilatory/lactate threshold

AeT/AnT: aerobic/anaerobic threshold

50% the half-way between lactate threshold & VO2peak (Burnley et al., 2002), gas exchange threshold & VO2peak (Jones et al., 2003) or aerobic and anaerobic thresholds (Bishop et al., 2001)

MT, ET, WT: moderately, endurance and well-trained, respectively

# 3.2 Effects of prior-exercise on lactate concentrations and metabolic acidosis

Lactate has been defined as a "left-over by-product of anaerobic metabolism that is converted back into ATP" (Pearson, 2001, p. 145). It has commonly been considered in the past to be a "waste-product", and has widely been considered (especially in the coaching circles) the cause of muscle fatigue and detrimental to performance. However, recent research exists providing evidence that lactate may have a greater and more positive role in the regulation of energy and metabolism during exercise (for examples see Cairns, 2006; Gladden, 2000).

Gladden (2000) in a review of lactate metabolism during exercise cited studies that showed the uptake of lactate to be oxidised or reconverted by the glycolytic muscles fibres back into glycogen through the process of glyconeogenesis, or shuttled intra and extracellularly for oxidation. In a review by Westerblad, Allen and Lannergren (2002), and more recently by Cairns (2006), lactate has been questioned as a true cause of fatigue, and even considered beneficial to performance. Both reviews drew attention to the quality of 'evidence' suggesting lactate causes fatigue, which were mainly correlational studies, and show only that there is an increase in [lactate] with exercise, not that it causes the onset of fatigue. Rather, as Cairns (2006) pointed out, lactate and acidosis (increased  $H^+$ ) may prolong fatigue, as evidenced by induced acidosis. One cause of fatigue that was proposed from the evidence complied by Cairns (2006) was  $K^+$ , or  $K^+$  combined with  $H^+$ . However, it was clear that  $H^+$  was not the sole cause of fatigue, as has been the common belief in recent years; in agreement with the observation of Nielson et al. (2001) that acidification offsets the fatiguing effects of elevated  $K^+$  in the muscle. Westerblad et al. (2002) proposed that, of the factors involved in anaerobic metabolism, in fact it may be the increased concentrations of inorganic phosphate (P<sub>i</sub>) from the breakdown in creatine phosphate (CrP) that cause the fatigue, rather the acidosis. One suggested cause of force reduction as caused by increased P<sub>i</sub> was inhibited cross-bridge function by reducing sensitivity to calcium. These two reviews have therefore presented the abundant evidence against the previously held popular belief that muscle [lactate] ([MLa]), blood [lactate] ([BLa]) and  $H^+$  are the cause of fatigue. It now appears that  $P_i$  or  $K^+$  may in reality be causing the decrease in force generating capacity, possibly in combination with increased H<sup>+</sup>.

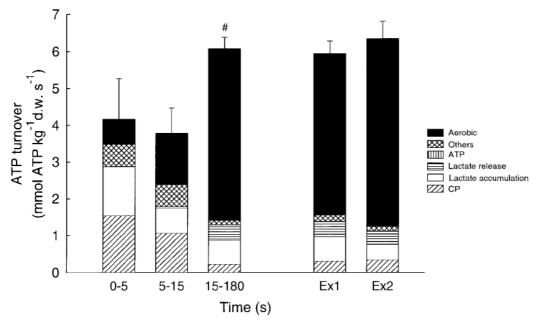
Despite the controversies surrounding the role, removal (Brooks, 2001) and effect of elevated lactate during exercise (Cairns, 2006), measurement of [BLa] is widely used as a 'marker' in routine laboratory and field assessments of athletes. The strong correlation and predictive ability from lactate thresholds (and turn-points) support its continued use, especially for endurance sports (Bosquet, Léger, & Legros, 2002; Coyle, Feltner, Kautz et al., 1991; Edwards, Clark, & Macfadyen, 2003; Midgley, McNaughton, & Wilkinson, 2006). The differences in the [BLa] clearance rate compared with clearance rate of other fatigue causing metabolites, and the speed which the electrochemical gradient across the muscle membrane is restored may be an important determinant in the augmentation of performance (Jones et al., 2003). The production, accumulation and subsequent clearance of [BLa] during and following exercise may be influenced by different priming strategies, which would in turn directly affect performance.

### 3.2.1 Greater metabolic response with increased exercise intensity

In the majority of studies, more intense priming exercise has unsurprisingly resulted in a greater metabolic ([BLa], [MLa], pH) response (Billat, Bocquet et al., 2000; Bishop et al., 2001; Bishop et al., 2003; Burnley, Doust, & Jones, 2002; Campbell-O'Sullivan et al., 2002; Hajoglou et al., 2005; Jones et al., 2003; Koppo & Bouckaert, 2000; Wilkerson et al., 2004) (Table 2-3). However, with the exception of only a few studies (Bishop et al., 2001; Fukuba et al., 2002; Koppo et al., 2003; Wilkerson et al., 2004), this did not result in an elevated response following the trial in comparison to the lesser intensity priming conditions or the control. As expected there was a greater [BLa] response in many of the studies due to the increased intensity of the prior exercise, but the same absolute work time (Billat, Bocquet et al., 2000; Bishop et al., 2001; Bishop et al., 2003; Burnley, Doust, & Jones, 2002; Campbell-O'Sullivan et al., 2002; Hajoglou et al., 2005; Koppo & Bouckaert, 2000), or high intensity compared with no prior exercise (Burnley, Doust, Ball et al., 2002; Fukuba et al., 2002; Hajoglou et al., 2005; Jones et al., 2003; Koppo & Bouckaert, 2000; Koppo et al., 2003; Wilkerson et al., 2004). This would result in an increase in total work completed prior to the trial, which logically should result in an increase in metabolic cost. This can be further evidenced by the significantly higher HR following the more intense prior exercise and also at the completion of the trial (Bishop et al., 2001; Burnley, Doust, Ball et al., 2002; Koppo et al., 2003).

Not all studies have reported significant differences in the metabolic response following different 'warm-up'/priming conditions (Bangsbo et al., 2001; Burnley, Doust, Ball et al., 2002; Fukuba et al., 2002; Koppo et al., 2003). In the study of Burnley, Doust, Ball et al. (2002), although [BLa] and blood pH both changed significantly between the first and second bouts of heavy cycling exercise, muscle pH (in the active muscle group) was not different between the two conditions. This suggests that it may be necessary to directly measure muscle pH in addition to blood pH and [BLa] as they can respond differently, and that the true metabolic response may in fact lie in the active muscles and may not be represented accurately in the analysis of blood acidosis. However, this is an invasive procedure to conduct.

The study of Bangsbo et al. (2001) was of similar design to that of Burnley, Doust, Ball et al. (2002) in that it investigated the different metabolic response between the first and second sets of identical exercise. Three minutes isoinertial knee extension exercise was performed twice, separated by six minutes passive recovery time. The metabolic response to each condition is illustrated in Figure 2-5 and shows how the concentration of different muscle metabolites may change both over the time course of short-term moderate to heavy intensity exercise and between conditions. Consistent with Burnley, Doust, Ball et al. (2002), [MLa] was found to be the same at the end of the second bout of exercise (EX2) as at end of the first (EX1). However [MLa] prior to the start of EX2 was higher than at the start of EX1 (elevated [MLa] at baseline following the six minutes of recovery). This resulted in a reduced  $\Delta$ [MLa] following EX2 and accordingly, [MLa] accumulation (measured as a function of time) was lower (P < 0.05) in EX2 than EX1. The [MLa] response was to be expected, as it was likely that the subjects were at the peak [lactate] expected to be achieved for that exercise intensity (~60w) following both bouts. Of interest however was the response of other metabolites and physiological responses. Along with the non-significant difference in muscle  $\dot{V}O_2$  at the end of each bout, muscle creatine phosphate returned to resting level before EX2 and decreased to the same level as during EX1, muscle ATP significantly decreased during EX1 and the mean rate of anaerobic ATP production was significantly less in EX2 than EX1, and EX2 caused significantly greater blood flow.



*Figure 2-4:* Rate of muscle ATP turnover during 0–5, 5–15, and 15–180 s of EX1 as well as during EX1 and EX2.

Estimated as the sum of muscle anaerobic energy production determined as energy release related to utilization of CP (hatched part of bar), net lactate production determined as the sum of accumulation in muscle (open bar) and release to the blood (horizontally lined bar), net ATP utilization (vertically lined bar), others (crosshatched bar), and aerobic energy production (filled bar), determined from muscle oxygen uptake and estimated utilization of oxygen from myoglobin. Values are means  $\pm$  SE. #Significantly (P < 0.05) different from 0–5 and 5–15 s (Bangsbo et al., 2001).

These studies show that total work performed in the priming exercise affects the associated metabolic response. However, it does not show how priming strategies of different intensity, but equal total work (i.e. of differing duration) may affect metabolic responses. Thus, future research investigating the physiological response and metabolic cost of different priming strategies may want to consider manipulating either the duration or intensity of the different priming conditions so that the total work performed during the priming exercise is equal. Some priming studies may have achieved similar total work performed through greater intensity priming exercise being of shorter duration (e.g. Burnley, Doust, & Jones, 2002), although it may be necessary to calculate (and report) the true mechanical work of the task to ensure it is equal between conditions, something that is lacking in most previous studies. This would allow for investigation of the true physiological response of a priming strategy of different or unique design, irrespective of the expected increases in metabolic cost due to an increase in total work performed.

# 3.2.2 Passive warming strategies and priming with a different muscle group

Of primary interest are those studies that compared passive warming strategies with active priming, or those that compared priming with different muscle groups in the 'warm-up'. These strategies allow the investigation of the effects of  $T_C$  and local  $T_M$ , or increasing [BLa] through a different muscle group, on the [BLa] response in the active muscles and on subsequent performance irrespective of other contributing factors.

### Passive warming

Similar [BLa] responses to the effects of a decreased magnitude of response with regards to the  $\dot{V}O_2$  slow component and HR have been noted when utilising passive warming techniques. Prior warming of the lower limbs by using hot water (Burnley, Doust, & Jones, 2002) and wrapping an electric heat blanket around the legs (Gray et al., 2002) was shown to result in significantly less [BLa] than an active 'warm-up' of high intensity, which resulted in the same T<sub>M</sub>. Additionally, while prior heavy exercise (six minutes at  $50\%\Delta$ ) was shown to result in a significantly lower [BLa] than a 30 second maximal effort sprint priming exercise, passive warming produced significantly less [BLa] than both active 'warm-up' conditions (Burnley, Doust, & Jones, 2002). In addition to the greater [BLa] following 'warm-up' conditions, the heavy and passive 'warm-ups' elicited significantly greater *changes* in [BLa] ( $\Delta$ [BLa]) than sprint 'warmup' following the final six minute heavy exercise trial, which was most likely due to the already significantly elevated baseline [BLa] at the onset of the trial in the sprint 'warmup' condition in comparison to the other conditions. These results were consistent with Gray et al. (2002) who found that prior passive warming resulted in significantly less [BLa] than the active 'warm-up', despite generating the same  $T_M$  immediately before the exercise trial, a difference that was still evident immediately following the 30 second sprint effort (120% W<sub>peak</sub>). Gray et al. (2002) also found that following the trial of 30 second sprints at 120% of maximum aerobic power on a cycle ergometer, there was no difference between conditions with respect to epinephrine, norepinephrine, ATP, [MLa] or phosphocreatine (PCr) concentrations. These results suggest that the increase in [BLa] from priming exercise is not wholly due to increases in muscle temperature and that prior [BLa] is not necessarily a determinant of [BLa] following exhaustive, very high-intensity, short duration exercise. Of interest also was the response of acetylcarnitine, which is an indicator of the relative contribution of anaerobic and oxidative ATP regenerating pathways to energy production at the start of high intensity

skeletal muscle contractions (Gray et al., 2002). Increased concentrations of acetylcarnitine may also facilitate recovery from high intensity muscular contractions like those required to perform the maximal short-duration (30 second) sprint efforts (Volek, Kraemer, Rubin et al., 2002). This was shown to have a significant time effect (p < 0.05) in the active 'warm-up', whereas there was no change in the control group following the passive warming. This may be interpreted as evidence that there were processes occurring to maintain the ATP levels in the active 'warm-up' group which were not necessary in the passive warming group. The findings with regard to acetylcarnitine, PCr and ATP were authenticated in two recent studies which reported 1) these factors all increased significantly more in the active 'warm-up' than the control (Sahlin et al., 2005), and 2) that the kinetics of PCr are accelerated (decreased  $\tau$  and slow component of PCr) during a second bout compared to an initial bout (Rossiter et al., 2001).

## Different muscle group

As with increased T<sub>C</sub>, [BLa] following the priming exercise may be the key factor that facilitates the subsequent performance and its associated metabolic response, irrespective of how it is achieved (i.e. with a different mode of exercise in a different muscle group). Interestingly, the two studies investigating the effects of priming with a different muscle group were also the two studies (in Table 2-3) that demonstrated a significant difference in lactate levels between priming conditions post exercise (Fukuba et al., 2002; Koppo et al., 2003). However, the results between these two studies were not consistent. Although demonstrating no significant difference in [BLa] between conditions following priming, Fukuba et al.(2002) reported that both priming conditions had significantly greater [BLa] at the termination of the exercise trial than the no prior exercise condition. These findings were not in complete agreement with those of Koppo et al. (2003), who reported that the exercise group that were primed with arm cranking before cycling had significantly greater end-exercise [BLa] than both the prior heavy cycling and no prior exercise groups, even though [BLa] was not significantly different between conditions post priming. These findings provide evidence that the lactate and  $\dot{VO}_2$  kinetics may not always respond similarly to the same stimulus during exercise. This is perhaps due to a smaller muscle group being used during arm cranking exercise, and therefore eliciting a smaller metabolic response. In support of this is reported data demonstrating that the  $\dot{V}O_2$  kinetic response was similar, although smaller in magnitude, when primed by a different muscle group (Koppo et al., 2003). Some studies however did not report significant effect between different muscle groups utilized in the priming exercise (Fukuba et al., 2002). It is also possible however that the subjects in this study found that the arm cranking exercise was harder than the cycling. Cycling, in contrast to arm cranking, would be an exercise that most of the participants would have participated in quite regularly, and coupled with the larger muscle groups employed, it would have likely been a much easier and less taxing exercise. This would be in contrast to the novel task of arm-cranking, where even at equal relative intensities (e.g. as a percentage of arm crank  $W_{peak}$  or power at LT), it would be harder to perform and  $T_{lim}$  would be potentially decreased, with the absence of local musculature adaptations.

In summary, high intensity priming exercise results in greater [BLa] (and other metabolites) in comparison to priming exercise and 'warm-ups' of lower intensity. This difference however is not present at the end of the subsequent trial in the majority of studies. This brings into question the role of [BLa] in fatigue and performance enhancement. Performing prior exercise with a different muscle group results in a different lactate response than when the exercise modes are the same in the priming phase and trial. How the differing concentrations of these metabolites affect the ensuing exercise performance will be discussed in subsequent sections.

Table 2-3: Effects on the lactate and acidosis response	lactate and acidosis re	ssponse			
Reference	Variable	Participants	Priming Strategy	[BLa] Following Priming	[BLa] Following Test
Billat et al. (2000)	[BLa]	8 M, ET: 60 ± 16km/week training volume	20min AT 50% vVO <sub>2max</sub> , 5min rest, T <sub>lim</sub> at 50%Δ 20min alternating 30s at 100% & 30s at 50% vVO <sub>2max</sub> , 15min rest, T <sub>lim</sub> at 50%Δ	Significantly higher $(75.7\%, 5.6 \text{ mmol.L}^{-1})$	NS
Jones et al. (2003)	[BLa]	7 M: MT	No prior exercise 6min 50%Δ, T <sub>lim</sub> at 100, 110 & 120% VO <sub>2peak</sub>	ı	NS
Bishop et al. (2003)	[BLa]	7 M, 500m kayak, State rep	15min 50%∆ 10min 50%∆, 5x10s sprints 200% VO <sub>2max</sub> , with 50s recovery at AeT	Significantly lower Significantly higher (exact values not stated)	NS
Burnley, Doust, & Jones (2002)	[BLa]	9 M: MT	<ol> <li>heavy exercise, HE: 2 x 6min cycle bout at 50%Δ</li> <li>sprint exercise: 30s at 17km/hr (120rpm) cycle preceded by HE</li> <li>40min passive warming lower limbs, preceded by HE</li> </ol>	Significantly higher than 3 (79.4%, 2.7 mmol.L <sup>-1</sup> ) Significantly higher than 1 & 3 (46.9 & 89.1%, 3.0 & 5.7 mmol.L <sup>-1</sup> ) Significantly lower	- Significantly higher than 1 (25.0%, 1.7 mmol.L <sup>-1</sup> ) -
Gray et al. (2002)	[BLa]	6 FM: MT	AW: 30s at 120% W <sub>peak</sub> , preceded by 5min 40% W <sub>max</sub> , 1min rest, 4 x 15s 120% W <sub>max</sub> Passive heating to same pre-ex T <sub>M</sub> as AW; 30s at 120% W <sub>peak</sub> ,	Significantly higher (80.8%, 4.2 mmol.L <sup>-1</sup> ) Significantly lower	Significantly higher (29.6%, 1.6 mmol.L <sup>-1</sup> ) Significantly lower

Reference	Variable	Participants	<b>Priming Strategy</b>	[BLa] Following Priming	[BLa] Following Test
Koppo & Bouckaert (2000)	[PLa]	12 (9M, 3FM); MT	<ul> <li>Ex-0: 6min at 90% VO<sub>2peak</sub> with no prior exercise</li> <li>EX-90: 2nd bout of 6min at 90% VO<sub>2peak</sub></li> <li>EX-50: 12. 1 ± 0.8min at 50% VO<sub>2peak</sub> followed by 6min at 90% VO<sub>2peak</sub></li> </ul>	Significantly greater (77.4%, 4.8 mmol.L <sup>-1</sup> ) Less than EX-90, NS from baseline (EX-0)	SN
Hajoglou et al. (2005)	[BLa]	8 M, WT cyclists & triathletes, local class	<ol> <li>No warm-up: 6min seated on erg</li> <li>5min segments at 70, 80 &amp; 90% VT</li> <li>5min segments at 70, 80 &amp; 90% VT, 3min at RCT</li> </ol>	- Significantly lower than HWU (exact values not stated) Significantly greater the EWU	SN
Wilkerson et al. (2004)	[BLa]	7 M: MT, recreationally active	MAX1: no prior exercise MAX2: 3x30s all-out cycling & 15min rest	Significantly lower Significantly higher (83.1%, 6.4 mmol.L <sup>-1</sup> )	Significantly lower Significantly higher (29.1%, 2.5 mmol.L <sup>-1</sup> )
			6min leg cycling (90% cycle VO <sub>2peak</sub> ) with no prior exercise (LE-C)		Significantly lower than LE-A
Koppo et al. (2003)	[BLa]	10 M: MT - WT	2nd bout of heavy leg cycle exercise: LE-L	NS	NS
			Heavy leg cycle preceded by heavy arm crank exercise (90% arm VO <sub>2reak</sub> ): LE-A	NS	Significantly higher than LE-C (16.8%, 1.7 mmol.L <sup>-1</sup> )

Reference	Variable	Participants	Priming Strategy	[BLa] Following Priming	[BLa] Following Test
Bangsbo et al. (2001)	[MLa]	6 M	3min knee extension with no prior exercise 2 <sup>nd</sup> bout of 3min knee extension	NS	NS
	[BLa]		15min AeT (W1)	[BLa] significantly lower. pH significantly higher (exact values not stated)	[BLa] significantly lower than W3. pH NS (exact values not stated)
Bishop et al. (2001)	ווהנכסות	8 M, elite kayak squad members	15min 50%Δ (W2)	[BLa] significantly higher than W1, pH significantly lower	NS
	FI 00010		15min AnT (W3)	[BLa] significantly higher & pH significantly lower than W1 & W2	[BLa] significantly higher than W1. pH NS
			A) cycle 10min at 55% VO <sub>2max</sub> , 3min rest, 10min at 75% VO <sub>2max</sub>		NS from B)
Campbell-	Muscle lactate		B) cycle 10min at 75% VO $_{2max}$	ı	NS from A)
U Sulfiyan et al. (2002)	[MLa]	/ M: M1, recreationally active	C) cycle 10min at 55% VO <sub>2max</sub> , 3min rest, 1min at 75% VO <sub>2max</sub>	ŗ	Significantly less than D) $(48.0\%, 15.9 \text{ mmol.L}^{-1})$
			D) cycle 1min at 75% VO <sub>2max</sub>	·	Significantly higher than C)
Burnley, Doust, Ball et al. (2002)	Muscle pH Blood pH [BLa]	8 healthy males: moderately trained	2 x 6min heavy ex (70% Δ between VT & VO <sub>2peak</sub> ), no prior exercise	[BLa] greater (75.5%, 3.7 mmol.L <sup>-1</sup> ), blood pH lower $(-2.3\%, -0.17)$ in $2^{nd}$ bout, muscle pH NS	

Reference	Variable	Participants	Priming Strategy	[BLa] Following Priming	[BLa] Following Test
			L1-ex: 6min leg cycling (LT + $50\%\Delta$ ) with no prior exercise		Significantly lower
Fukuba et al. (2002)	[BLa]	6 M	L2-ex: 2 <sup>nd</sup> bout supra-LT leg cycling	NS from end of A1-ex (start of A2-ex)	NS
			Al-ex to A2-ex: 6min heavy arm cranking (1W/kg BW), 6min supra-LT leg cycling	NS from end of L1-ex (start of L2-ex)	Significantly higher than L1-ex (24.4%, 2.2 mmol.L <sup>-1</sup> )

RCT: respiratory compensation threshold

VT/LT: ventilatory/lactate threshold

AeT/AnT: aerobic/anaerobic threshold

50% half-way between lactate threshold & VO2peak (Burnley et al., 2002), gas exchange threshold & VO2peak (Jones et al., 2003) or aerobic and anaerobic thresholds (Bishop et al., 2001)

MT, ET, WT: moderately, endurance and well-trained, respectively

### 4. Effects of priming on performance measures

Whilst it is important to consider the physiological and metabolic responses to different intensity exercise, the most important measure for athletes and coaches to consider is the 'actual' performance after prior exercise. This gives insight into the real effectiveness of the chosen priming strategy and how it may transfer to a competitive setting. In contrast to many studies that have investigated prior exercise on physiological measures, surprisingly few studies have considered performance measures.

# 4.1 Measures of performance in cycling and maximal endurance exercise: validity, reliability and specificity

There are many ways to assess cycling performance. An appropriate test must be chosen which reflects (i.e. displays a strong relationship with) the actual demands of the competition/event. Cycling events vary greatly in duration (and intensity), from track sprints lasting approximately ten seconds to road races that last over six hours, and thus a variety of reliable and valid assessments are required to accurately quantify a cyclist's physical abilities in relation to the demands of their event.

Numerous types of tests have been utilized to assess cycling performance and endurance capacity. These include, but are not restricted to, constant load tests (Berthoin, Manteca, Gerbeaux, & Lensel-Corbeil, 1995; Heubert et al., 2005), laboratory-based time trials (TT; Bishop et al., 2001; Hajoglou et al., 2005), peak power tests (Kang, Chaloupka, Mastrangelo, Biren, & Robertson, 2001; Wilkinson, Fallowfield, & Myers, 1999), and any combination of these tests (Doherty, Balmer, Davison, Robinson, & Smith, 2003; Doyle & Martinez, 1998). Constant load tests can be sub-maximal for a set duration, in order to assess physiological responses to a set workload pre- and post-intervention and to monitor progress, i.e. a sub-maximal blood lactate or exercise economy assessment. These assessments typically require participants to exercise at a set relative intensity corresponding to some physiological variable (e.g. lactate threshold (LT) or a percentage of  $\dot{V}O_{2peak}$ ) until exhaustion, and is called the time to limit of exhaustion (T<sub>lim</sub>). Lab-based TT's on the other hand require subjects to perform a set amount of work (i.e. total distance, wattage or kilojoules of energy) in as little time as possible, or perform as much work as

they can within a set time frame. Finally, incremental tests for the determination of peak aerobic power ( $W_{peak}$ ) are commonly used in the physical profiling of an athlete, either incremental ramp (Whipp, Davis, Torres, & Wasserman, 1981) or step test (Grant, McMillan, Newell et al., 2002) protocols can be used.

#### Time-trials

Paton and Hopkins (2001), in an extensive review on the reliability of various ergometers and performance tests in cycling, reported that the two most reliable predictors of cycling performance were W<sub>peak</sub> to predict simulated 16.1 kilometre time trial (16km-TT) performance (typical error (TE) = 0.7%) and 40 kilometre time trial (40km-TT) performance, as represented by mean power ( $W_{mean}$ , TE = 1%). Interestingly, the assessment that exhibited the value with the greatest reliability and the strongest relationship with W<sub>peak</sub> in Paton and Hopkins' review, 16.1km-TT W<sub>mean</sub>, was that of Balmer, Davison, & Bird (2000) which also exhibited a very weak relationship when the 16.1km-TT field test was represented as 'actual' performance. 'Actual' performance was measured by time to complete the distance, and had a correlation of only -0.46 (p = 0.07) with W<sub>peak</sub>, which translated to a larger TE of 4.7% compared to that reported by Paton and Hopkins (2001). The authors reported that these relationships were significantly improved when  $W_{\text{peak}}$  and  $W_{\text{mean}}$  values were made relative to body weight; however, the correlations of r = 0.64 and 0.66 were still only 'moderate' at best, and not 'nearly perfect' relationships, as they were with laboratory-based W<sub>peak</sub> and W<sub>mean</sub> (Hopkins, 2002). When considering the poor relationship the W<sub>peak</sub> exhibited with 16.1km-TT time (in contrast to W<sub>mean</sub>), the inability of W<sub>peak</sub> in a laboratory to predict 16.1km-TT time in a field assessment or race is highlighted. Both Paton and Hopkins (2001) and Balmer et al. (2000) acknowledged that the poor relationship with 16.1km-TT time compared to with W<sub>mean</sub> reflected the marked effect of aerodynamics due to body size, racing position, bicycle design and the effects of wind and ambient temperature on cycling performance. Clearly, this highlights the need to standardise the environmental conditions to ensure reliable results, which will increase the chance of identifying the true effect(s) (Hopkins, Hawley, & Burke, 1999).

One hour cycle TT's have been shown to be both reliable (Bishop, 1997) and valid (Coyle et al., 1991). Coyle et al. (1991) tested for validity by examining the relationship that

various mechanical and physiological variables had with 'actual' 40 kilometre TT (40km-TT) performance. It was found that there was a high correlation between average absolute power during the one-hour laboratory-based TT and both 40km-TT performance (r = -0.88; p < 0.001) and  $\dot{VO}_2$  at LT (r = 0.93; P < 0.001). These relationships provide evidence of the validity of a one-hour laboratory based TT, however, it does not provide any evidence of its reliability. To address this, Bishop (1997) subsequently investigated the reliability of the one-hour TT. A number of interesting findings were presented in their study. Firstly, heart rate (HR) and ratings of perceived exertion (RPE) were reliable (ICC = 0.91 and 0.75, respectively). However, they were not as reliable as the  $W_{mean}$  (ICC = 0.97), the variable which was previously determined by Coyle et al. (1991) to be valid. The change in HR between trials was not dependant on changes in W<sub>mean</sub> between trials, as evidenced by a very weak relationship between the two variables (r = 0.17, p > 0.05). This not only highlights the superior reliability of  $W_{mean}$  over HR, but also that the variance in HR between trials cannot be as a result of variance in  $W_{mean}$  between trials. In addition, the absence of any significant relationship between variation in TT performance (expressed as  $W_{\text{mean}})$  and best TT performance,  $\dot{V}O_{2\text{peak}},$  or LT indicated that the reliability (i.e. inter-trial variance) was not affected by the level of conditioning of the individual subjects (i.e. fitness level does not affect reliability). It appears from the results of these two studies that a onehour TT is both a valid (Coyle et al., 1991) and reliable (Bishop, 1997) method of assessing endurance cycling performance in both the laboratory and as a field measure, with the most reliable variable being W<sub>mean</sub>.

The duration of a TT could influence the reliability of measured obtained. To address this, Hickey et al. (1992) investigated how duration affected reliability in endurance-trained cyclists. Three distances were examined on four occasions: long (TT<sub>long</sub>; 64km), moderate (TT<sub>mod</sub>; 8km) and sprint (TT<sub>sprint</sub>; 0.8km) distances. The mean CV's were 1.01%, 0.95% and 2.43% for TT<sub>long</sub>, TT<sub>mod</sub>, and TT<sub>sprint</sub>, respectively. Whilst the CV's are low, mean CV for TT<sub>sprint</sub> was much larger than for the other conditions. For the measures of time, total kJ of energy and mean power, this difference was significant (p < 0.05). The authors cited this as evidence of the inability of endurance-trained cyclists to reproduce sprint performance with the same fidelity as either medium or long duration endurance performance. This is a plausible explanation, given that it is expected that cyclists whom are familiar with only riding longer distances would find it difficult to reproduce performances over a distance, and at an intensity, that they are not accustomed to. However, trial four of  $TT_{long}$  was significantly different than the first three trials, with greater mean percentage of  $\dot{V}O_{2peak}$  and faster performance time (~1%) which resulted in a sizeable improvement of one minute. The authors stated that a lack of difference in RER responses between trials indirectly supported the assumption that this did not represent any alteration in fuel availability or substrate utilisation relative to the first three trials, but rather the athletes worked harder knowing that it was final trial, and these TT performances were reproducible. The improved performance in the final  $TT_{long}$  also highlights the consistent level of motivation that is required of athletes to minimise variability and ensure reliable and valid data, however difficult this is to regulate.

#### Constant load $(T_{lim})$ and Pre-loading TT

A novel method of testing endurance performance is to use a 'pre-loading' assessment, which incorporates a combination of constant load exercise for a set period of time which is then followed by a TT, where the subjects either perform as much work as they can within a set amount of time (Doherty et al., 2003), or where they complete a set amount of work (or distance) as fast as possible (Doyle & Martinez, 1998). Assessments using this concept have been designed and assessed for reliability in two studies who suggested that, by themselves, exercise capacity tests (i.e.  $T_{lim}$ ) were not reliable, but combined with a TT, constant load tests may result in even greater reliability (Doherty et al., 2003; Doyle & Martinez, 1998).

Doyle et al. (1998) investigated the reliability of this type of test to assess endurance capacity in both cycling and running. Ten runners and ten cyclists were required to perform a trial, four times, in which they exercised for 90 minutes at a power output corresponding to 70% of  $\dot{V}O_{2peak}$ , followed immediately by a fixed distance as fast as possible which equated to the distance covered in 30 minutes of the constant-load part of the test, in their respective exercise mode. The performance times test-retest reliability was r = 0.59 (CV = 10.1%) and r = 0.92 (CV = 4.0%) for the runners and cyclists, respectively. The authors then removed the first trial and excluded it from the analysis to examine the effect of considering it a learning trial. This resulted in an increase in the reliability of the

cycling (r = 0.93, CV = 3.5%) and a substantial increase in reliability of the running performance (r = 0.93, CV = 4.4%). These results showed that this combined constant-load and TT endurance protocol was very reliable when either running or cycling was performed repeatedly under the same carefully controlled conditions. However, these are not preferable over those reported for a one hour ride (Bishop, 1997) or 64km-TT (Hickey et al., 1992). The findings of Doyle et al. (1998) also highlight the need for a familiarisation pre-loading test, especially when assessing runners, who it seems are less able to regulate their performance in a prolonged exercise test than cyclists.

A more recent study investigating this type of assessment focused on high intensity, shorter duration performance (Doherty et al., 2003). Participants were required to cycle at their individual  $W_{peak}$ , as determined in an incremental assessment, for two minutes, and then immediately perform as much work as they could for one minute. Reported CV values of performance measures in this assessment were even more favourable than those of the endurance pre-loading assessment (Doyle & Martinez, 1998). Peak performance power ( $P_{peak}$ ), peak cadence ( $C_{peak}$ ) and total distance completed were 3.6%, 0.9% and 3.0%, respectively, with no significant difference between trials for either  $P_{peak}$  nor  $C_{peak}$ , as determined by repeated measures ANOVA. In addition to this,  $P_{peak}$ ,  $C_{peak}$  and total distance completed resulted in ICC of 0.96, 0.99 and 0.98, respectively, thereby providing a highly acceptable level of test-retest reliability. The authors concluded that for this test, the high ICC, lack of systematic bias between successive trials, and an acceptable within-subject error collectively verified that it was indeed a reliable measure of power output.

Of the TT assessments discussed in this review, the one that would merit the greatest comparison with the high intensity, shorter duration pre-loading protocol of Doherty et al. (2003) would be the  $TT_{sprint}$  (0.8km) of Hickey et al. (1992). As mentioned above, the  $TT_{sprint}$  was proven very reliable, with a CV of only 0.95%, much more favourable than the 3.0% CV for total distance completed in this pre-loading protocol. The authors of both of these pre-loading studies (Doherty et al., 2003; Doyle & Martinez, 1998) however stated that the purpose of these assessments was to provide a assessment that would be more reliable than stand-alone exercise capacity tests. Exercise capacity, as measured by  $T_{lim}$ , has been previously reported to be of low reliability (Atkinson & Nevill, 2005; Jeukendrup & Currell, 2005; Jeukendrup, Saris, Brouns, & Kester, 1996) of which this form of

assessment the CV improves dramatically (e.g. versus 26.6%; Jeukendrup et al., 1996). The debate surrounding CV as an appropriate measure of reliability of  $T_{lim}$  is contested however (Atkinson & Nevill, 2005; Hinckson & Hopkins, 2005a, 2005b; Hopkins & Hinckson, 2005; Jeukendrup & Currell, 2005).

Despite the range of assessments available, few studies have compared test protocols. A study performed by Jeukendrup et al. (1996) compared the reliability of three different protocols: 1)  $T_{lim}$  at 75% MAP (protocol A), 2) combination constant load of 45 minutes at 70% MAP and a 15 minute TT (protocol B), and 3) a one-hour TT where subjects were required to perform a certain amount of work (equal to ~one-hour of cycling) as fast as possible (protocol C). To allow for comparison of physiological responses and standardised energy expenditure, all three protocols were designed to last approximately one hour, with each performed six times (including a learning trial). The mean CV (individual CV range) for protocols A, B and C were 26.6% (17.4% - 39.5%), 3.5% (1.7% - 5.8%) and 3.4% (0.8% - 5.8%), respectively. While the CV for protocols B and C were not significantly different (p = 0.88), both were substantially lower than protocol A (both p values < 0.0001).

The authors stated that the high CV associated with protocol A supported their agreement with Krebs and Powers (1989, as cited in Jeukendrup et al., 1996) that  $T_{lim}$  is not a reliable assessment, especially in comparison to tests with a known endpoint; a belief that is held by many researchers (Atkinson & Nevill, 2005; Doyle & Martinez, 1998; Jeukendrup & Currell, 2005; Jeukendrup et al., 1996). However, Jeukendrup and Currell (2005) and Atkinson & Nevill (2005) do not account for the signal to noise ratio, as explained in detail by Hinckson and Hopkins (Hinckson & Hopkins, 2005a, 2005b; Hopkins & Hinckson, 2005). Briefly, small increases in power output will often result in small, but significant, changes in TT performance. That same increase in power (or change in 'signal') will result in a much more substantial change in  $T_{lim}$ , one that is far greater than the CV (or 'noise') of the  $T_{lim}$  protocol of assessment. This signal to noise ratio is likely to be of a similar magnitude to that of the TT, and shows why it is also an acceptable test to assess changes in performance. It has been recommended to perform two  $T_{lim}$  tests to account for the differences in the power-duration curve, just as one would perform a TT of both long and shorter distances (Hinckson & Hopkins, 2005b).

In summary, when examining all the studies mentioned above, it is apparent that there are many different ways of assessing performance in cycling. The different assessments have varying levels of validity and reliability. Although reliability, and the measures used to represent it, is contested, the most reliable performance measure appears to be a TT. It is important that the validity of the test is considered with respect to the demands of the competition. It is important to take into account the level of error associated with each type of assessment when considering changes in performance. The changes in performance must be greater than that error to exhibit a true 'meaningful' change, and therefore, the assessor must note the smallest worthwhile change to be sure of any performance enhancement or notable difference in performance between conditions (Hopkins et al., 1999).

### 4.2 Effect of prior exercise on power output and time-trial performance

It can be observed in *Table 2-4* that few studies have incorporated a performance measure in their investigations on the effect of prior exercise, and even fewer utilising the most reliable (and seemingly favourable) method of assessing endurance performance – the timetrial (TT) (Bishop et al., 2001; Bishop et al., 2003; Hajoglou et al., 2005). Most have only examined the physiological response, with the majority utilising fixed workload exercise such as square-wave protocols (e.g. Koppo, Bouckaert, & Jones, 2004; Rossiter et al., 2001). Whilst focussing on the physiological response is necessary from a mechanistic perspective, considering the important outcome measure of performance for the athlete is equally important.

Most of the studies that have investigated prior exercise have used cycling (e.g. Burnley, Doust, Ball et al., 2002; Hajoglou et al., 2005; Tordi et al., 2003) and running (e.g. Billat, Bocquet et al., 2000; Judelson et al., 2004). However, a unique pair of performance-focussed studies utilising kayak ergometry were performed by Bishop and colleagues (Bishop et al., 2001; Bishop et al., 2003). Both studies utilised a two minute 'all-out kayak' ergometer time trial (2min-TT) as the performance measure to quantify peak and mean power outputs. This duration is close to the time taken to complete the K1-500 metre sprint kayak event for a state-level kayaker.

The priming conditions used in the first Bishop et al. (2001) study were of a continuous nature, and required participants to paddle for 15 minutes at either kayak-specific aerobic threshold (AeT, low intensity; W1), anaerobic threshold (AnT, high intensity; W3) or at an intensity half way between the two (50% $\Delta$ , moderate intensity; W2). The latter study by Bishop et al. (2003) compared the most effective condition from the first study (W2; ~65%  $\dot{VO}_{2peak}$ ) with a high intensity intermittent priming protocol. This intermittent protocol consisted of the same first ten minutes as the continuous condition, but in the final five minutes, the subjects performed five sprints at a power output corresponding to ~200% of  $\dot{VO}_{2peak}$ , separated by 50 seconds at ~55%  $\dot{VO}_{2peak}$ . The protocols of the priming conditions of these two studies were novel in that not only were they of shorter duration than typically used in preparation for a supramaximal endurance event, but were also much higher intensity; in particular the intermittent sprint nature in the latter study. Although there were differences between the designs of the two studies, they did exhibit similar findings.

As discussed in previous sections, no significant differences in gas analysis values existed between conditions with either study by Bishop et al. Additionally, the earlier study reported each successful exercise intensity produced significantly greater [BLa] than the last (W1<W2<W3). These physiological responses are more relevant when considered along with the resulting mean power output. *Figure 2-6* displays the power output between the three priming conditions at 15s intervals throughout the two minute TT, reported by Bishop et al. (2001).

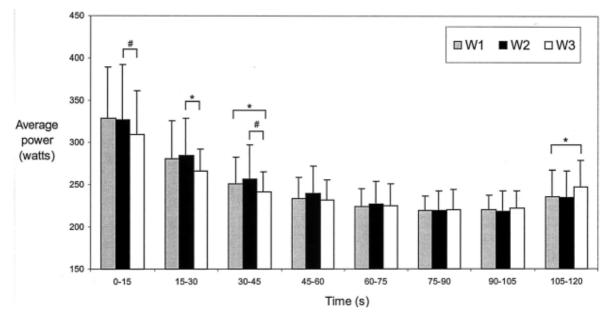


Figure 2-5: Power output responses to three kayak 'warm-up' intensities (Bishop et al., 2001)

These results demonstrated that 2min-TT performance did not significantly differ between The moderate priming intensity (W2) resulted in 1.8% and 2.9% greater conditions. average power output than the lowest priming intensity (W1) and the highest priming intensity (W3), respectively, and W1 displayed 1.1% greater average power than W3. However, when each half of the 2min-TT was considered separately, improved kayak performance was evident in the first minute of W2 (277.0  $\pm$  56.1 W), with significantly improved  $W_{mean}$  (5.4%; p < 0.05) when compared to the W3 (262.1 ± 44.6 W). Additionally, the greater W<sub>mean</sub> and W<sub>peak</sub> for the whole 2min-TT in W1 and W2 compared to W3 approached significance (p = 0.09 and 0.1, respectively), and all conditions were very similar in the second half of the test (1.6 - 1.7% difference). The authors suggested that the large individual variance in W<sub>mean</sub> lessened the ability of the ANOVA to detect significant effects. However, these results suggest that performance may be improved with the more intense preparation of the moderate than the low-intensity priming condition, but that the greater metabolic stress of the most intense priming condition impairs performance. This is especially relevant when one considers that first and second place the K1-500m sprint kayak event are only separated by 0.01s at an Olympic level (Bishop, Bonetti, & Dawson, 2002). This relates to the authors concluding statement that, although the increased [BLa] response with each successive priming condition had no significant effect on the corresponding  $\dot{V}O_2$  in this particular study but did result in a trend towards increased power output, a degree of metabolic acidemia may be necessary to accelerate VO<sub>2</sub> kinetics. Indeed, the relationship between acidemia and  $\dot{V}O_2$  kinetics has been supported by a number of previous studies (e.g. Billat, Bocquet et al., 2000; Burnley, Doust, & Jones, 2002; Gerbino et al., 1996). This accelerated  $\dot{VO}_2$  kinetic response resulting from increased [BLa] would then be expected to result in improved performance through greater contribution of aerobic energy pathways. However, the reduction in 2min-TT performance following the most intense priming condition (although non-significant) provides evidence that the metabolic acidemia associated with too intense a priming exercise may impair the supra-maximal performance by too great a reduction in the contribution of anaerobic energy sources, and/or by interfering with muscle contractile processes and/or inhibition of anaerobic glycolysis. These factors were among those which the authors cited to be in agreement with Hermansen (1981) and factors which are likely to have a particularly strong influence in an event such as the 2min-TT or K1-500m which has a significant anaerobic contribution. Indeed, Bishop et al. (2001) demonstrated that anaerobic and aerobic contributions during the 2min-TT were ~35% and ~65%, respectively.

Bishop et al. (2001) demonstrated that continuous active priming exercise of moderate intensity significantly improved performance in the first half of the 2min-TT compared to the heavy intensity priming exercise. However, this difference was not significant between conditions at the end of the whole trial, suggesting that performance was unchanged. The subsequent study by Bishop et al. (2003) sought to determine the difference between the favourable moderate intensity continuous priming exercise with that of a high-intensity intermittent priming strategy. However, there was no effect on the peak value of  $\dot{V}O_2$ following the two minute trial, or the total  $\dot{V}O_2$ , although the  $\dot{V}O_2$  was significantly higher for the last five minutes of the high intensity intermittent priming condition than the continuous (Bishop et al., 2003). As a result of the maintained  $\dot{V}O_2$  and increased [BLa] responses, performance was improved in the trial that was preceded by the intermittent priming exercise (see Table 2-4). Again, these findings provide strong argument for the increase in [BLa] as an influencing factor that helps increase power output and improve overall performance. Because the [BLa] was also already elevated, this may have improved performance through smaller increases in [BLa] and a greater ability to maintain the anaerobic contribution to the supra-maximal task. In addition, the authors suggested

that the significantly greater peak power after the high intensity prior exercise could be attributed to the subjects being more able to recruit additional motor units for the subsequent trial, and that it was possible that the large voluntary contractions required increased voluntary neuromuscular activation. This concept of improved muscle unit recruitment has been previously shown to be related to the  $\dot{V}O_2$  slow component (Krustrup et al., 2004). Additionally, a reduction in the slow component with increased priming intensity has been shown to result in greater  $\dot{V}O_2$  closer to the onset of exercise (Billat, Bocquet et al., 2000). Collectively, this supports the suggestion by Bishop et al. (2003) that additional muscle unit recruitment, and thus increased  $\dot{V}O_2$  response, closer to the onset of exercise may be the primary factors influencing performance following the priming exercise.

More recently, Hajoglou et al. (2005) investigated the effects of two 'warm-up' conditions (EWU and HWU; easy and hard warm-up, respectively) on three kilometre cycle ergometer time trial (3km-TT). Average power output and time to completed three kilometres were measured in the trial. Following the HWU, W<sub>mean</sub> was improved in the first 500m compared with EWU, and both were significantly greater than the control. The W<sub>mean</sub> in the EWU condition remained significantly greater than the no 'warm-up' control (CON) for the next 500m also (see *Table 2-4*). Despite the significant differences in  $W_{mean}$  early in the 3km-TT, EWU and HWU had very similar 3km-TT times (266.8  $\pm$  12.0 and 267.3  $\pm$  3s, respectively). These 3km-TT times were both significantly less than the CON condition  $(274.4 \pm 12.1s)$  by 2.8% (EWU) and 2.6% (HWU). The observed data provides evidence of the benefits of priming exercise before high intensity maximal exercise lasting approximately 4.5 minutes. It did not however provide evidence of greater benefits of high intensity priming over moderate intensity, which supports the non-significant overall difference in W<sub>mean</sub> between conditions also reported by Bishop et al. (2001). The authors stated that accelerated  $\dot{V}O_2$  kinetics and the subsequent augmentation of aerobic metabolism is the most feasible explanation for improved performance, as no differences in anaerobic energy contribution were reported. Aerobic contribution to mechanical work was calculated by Hajoglou et al. (2005) by multiplying metabolic work and efficiency, thereby allowing anaerobic energy contribution to be calculated by subtracting work attributable to aerobic metabolism from total work accomplished. [BLa] is likely to have played some

role in the improved performance also, as it was significantly greater than the control for both priming conditions. Increased [BLa] was also exhibited in a number of previous studies, where it appears to have a positive effect (Bishop et al., 2001; Bishop et al., 2003; Jones et al., 2003). The influence of [BLa] does not seem as convincing in the work of Hajoglou et al. (2005), due to the HWU not performing any better than the EWU condition, regardless of increased [BLa]. The role of lactate in exercise is heavily debated and its use as a measure of performance has been questioned (Cairns, 2006). Further work to establish its contribution as a 'primer' is required.

### 4.3 Time to exhaustion

Four of the reviewed studies (see *Table 2-2*) used time to exhaustion  $(T_{lim})$  as the performance measure when investigating the effects of different prior exercise preparations (Billat, Bocquet et al., 2000; Jones et al., 2003; Stewart & Sleivert, 1998; Wilkerson et al., 2004). The results surrounding these studies appear somewhat inconsistent, with one reporting improved performance compared to no prior exercise (Jones et al., 2003), and others reporting decreased performance in comparison to no prior exercise (Wilkerson et al., 2004) and lower intensity exercise (Billat, Bocquet et al., 2000). Stewart et al. (1998) reported improved performance in contrast to no prior exercise following priming conditions of 15 minutes at both 60 and 70%  $\dot{V}O_{2peak}.$  However, following a very high intensity priming strategy (80% VO<sub>2peak</sub>) performance was reduced compared to 70% VO<sub>2peak</sub>, but not significantly different from the non-priming control. The study of Marles et al. (2006) should be acknowledged, which also demonstrated reduced T<sub>lim</sub> following a prior bout of constant-load very heavy exercise (90% VO<sub>2peak</sub>) compared to the no-exercise control. However, this was not a 'true' T<sub>lim</sub> assessment, in that it was an incremental ramp protocol to exhaustion, rather than exercise of a constant-load to exhaustion. Nevertheless, such very high intensity priming strategies appear to be detrimental to subsequent performance.

Billat, Bocquet et al. (2000), in their examination of the effects of continuous sub-LT and maximal intermittent priming exercise on  $T_{lim}$  in long-distance runners, reported that MRT and  $\dot{V}O_2$  slow component were decreased, [BLa] was increased, and time spent at  $\dot{V}O_{2peak}$ 

was maintained in the intermittent condition. Surprisingly however, the result of these effects was a significant (24.2%, p = 0.02) decrease in T<sub>lim</sub> in the higher intensity priming exercise group. This finding suggests that the higher intensity intermittent priming exercise may not be appropriate before competition. The observed reduction in performance is in contrast to the improved kayak performance following the high intensity intermittent priming strategy of Bishop et al. (2003). However, this reduction in performance is potentially due to the greater duration of the priming exercise used by Billat, Bocquet et al. (2000) (20 versus 15 minutes) as well as the time spent in the intermittent phase of the priming exercise (20 versus five minutes).

It appears that the duration of high intensity exercise has an effect on performance. Many previous priming related studies have adopted a square-wave design of fixed duration (Behnke, Kindig, Musch, Sexton, & Poole, 2002; Brittain, Rossiter, Kowalchuk, & Whipp, 2001; Gerbino et al., 1996; Koppo & Bouckaert, 2000), making 'actual performance' impossible to measure. One unique study however incorporated two cycle exercises at 105% VO<sub>2peak</sub> to exhaustion. The first bout was performed with no prior exercise. The second bout was performed after 60 minutes rest following the first bout and was preceded by three bouts of 30 second maximal sprints. The study thereby utilised a square-wave design of sorts, whilst still managing to have a performance measure:  $T_{lim}$  in each 105% VO<sub>2peak</sub> trial (Wilkerson et al., 2004). Wilkerson and colleagues (2004), like Billat, Bocquet et al. (2000), demonstrated decreased MRT and increased [BLa] with the more intense preparation to exercise, as well as an increased  $\dot{V}O_{2peak}$  attained at the end of In agreement with Billat, Bocquet et al. (2000), performance (T<sub>lim</sub>) was exercise. significantly reduced when compared to the control condition. One possible explanation for the decrease in performance in these studies that was not seen in others is the priming strategy may have been too intense for the intensity of the subsequent exercise trial, causing a 'fatigue effect' rather than a 'warm-up/priming effect'. Clearly, some studies are investigating mechanistic aspects rather than having a realistic performance focus. This mechanistic approach is especially evident in the study of Wilkerson et al. (2004) where they attempted to include a performance measure, but at two exhaustive bouts at 105%  $\dot{V}O_{2peak},$  and one preceded by three maximal sprint efforts, all on the same day, it is clear that performance improvements were not the goal of the study. Rather it seems they were

interested in examining the  $\dot{V}O_2$  kinetic response to the two bouts of exercise, with  $T_{lim}$  an additional variable to include in their results.

Although the  $T_{lim}$  at 150%  $\dot{V}O_{2peak}$  might not be expected to influence physiological responses after such a long passive rest (60 minutes), it is possible that because the tests were performed on the same day, there was some carry-over effect on the [BLa] and  $\dot{V}O_2$  kinetic values following the second bout, contributing to the 'fatiguing effect' of the very high intensity intermittent priming strategy. When one considers the positive response to high intensity intermittent sprint priming for an event of similar duration (Bishop et al., 2003) and potentially relative intensity, it also seems plausible that the duration and intensity of the sprint efforts was too great for the subsequent exercise. If so, this would again contribute to the potentially fatigued state that the participants may have displayed for the second bout of supra-maximal exercise, in spite of the longer rest periods between sprint efforts (five minutes versus 55 seconds) and between priming and performance trial (15 versus 5 minutes) in the Wilkerson et al. (2004) study compared with Bishop et al. (2003).

In the study by Wilkerson et al. (2004), the results provided some indication that physiological variables may be important determinants of performance, as evidenced by the inhibited performance accompanying the altered physiological response in the second bout. Jones et al. (2003) supported the notion that the differences in the [BLa] clearance rate and the clearance rate of other fatigue causing metabolites, and the speed which the electrochemical gradient across the muscle membrane is restored may be important determinants in the augmentation of performance. It did appear that the increase [BLa] improved performance as the high intensity priming condition had a significantly greater  $T_{lim}$  than the no priming condition. The accelerated  $\dot{V}O_2$  kinetics no doubt also assisted in the improved performance by increasing  $O_2$  availability at the onset of the exercise trial, thereby enabling a steady-state  $\dot{V}O_2$  to be reached much earlier on. The increased HR at the termination of the exercise trial indicates a greater demand on the cardiovascular system, resulting in increased cardiac output, and therefore increased  $O_2$  delivery to the working muscles.

### Anaerobic performance

One priming study has investigated the effects of prior exercise on performance in a protocol that was almost completely anaerobic (Stewart & Sleivert, 1998). Stewart and Sleivert (1998) investigated the effects of 15 minute priming strategies at 60%, 70% and 80%  $\dot{V}O_{2peak}$  in comparison to no priming (control) on anaerobic running performance, as measured by  $T_{lim}$  when running at 13 km·hr<sup>-1</sup> on a treadmill inclined to 20%. It can be observed in *Table 2-2* that anaerobic running performance was most improved with the moderate intensity priming strategies of 60% and 70%  $\dot{V}O_{2peak}$ . The  $T_{lim}$  after the control and 80%  $\dot{V}O_{2peak}$  conditions, while not significantly different from each other, were significantly less than both of the moderate conditions, indicating reduced performance. The need for a priming strategy that is of moderate-heavy (but not too heavy) intensity is once again advocated by these findings.

These findings of Stewart and Sleivert (1998) are somewhat in agreement with those of Bishop et al. (2001), who also reported that low (AeT) and moderate (50% $\Delta$ ) prior exercise resulted in no significant difference in high-intensity exercise performance compared to each other, but too great intensity prior exercise condition (AnT) impaired performance when compared to the moderate priming condition. It was reported by Stewart and Sleivert (1998) that all priming conditions had significantly greater mean body temperature than the control condition, and 80% VO<sub>2peak</sub> was significantly greater than both other priming intensities. It was also reported that HR was significantly greater in each successive intensity compared to the last at each time point, i.e. control < 60% < 70% < 80%. It would therefore appear that exercising at 80%  $\dot{VO}_{2peak}$  produces too great a HR response and increase in body temperature for succeeding high intensity anaerobic running, whereas no prior exercise does not physiologically prepare the athlete sufficiently for the high demand exercise. Moderate intensity exercise however, performed between 60% and 70%  $\dot{V}O_{2peak}$ appears to be the ideal preparation for exercise of that nature. Stewart and Sleivert (1998) stated that they were investigating 'anaerobic performance', and at 13 km·hr<sup>-1</sup>, 20% incline, and lasting little more than 70 seconds in the most effective priming condition, it can be considered to have quite a significant anaerobic contribution. Indeed, in a review on energy system interaction it was reported that equal contribution of aerobic and anaerobic energy systems occurred ~75s into maximal effort exercise (Gastin, 2001), which is longer than the

trials in the study of Stewart and Sleivert (1998). In a high intensity task such as that of Stewart and Sleivert (1998), which of short duration and of an anaerobic nature, it is not likely sufficient oxidation will occur. This in-turn would result in decreased contribution of aerobic metabolism towards energy production and utilisation of gluconeogenesis (Billat, Sirvent, Py, Koralsztein, & Mercier, 2003). This is a probable explanation for the decreased performance with the elevated [BLa] in the highest exercise intensity in contrast to many of the previously discussed continuous endurance trials.

Table 2-4: Effe	Table 2-4: Effects on power output				
Reference	Participants	Variables	<b>Measurement Method</b>	Prior Exercise	Results
Bishop et	7 M 500m	MPO	2min all-out kayak erg	continuous: 15min 50% $\Delta$	W <sub>mean</sub> & W <sub>peak</sub> higher after intermittent priming
al. (2003)	kayak, State rep	Odd	TT	intermittent	(2.1 & 4.5%, 7 & 28W)
Mohr et	16 sub-elite M	2	friendly soccer match	Control: very low intensity activities	Pre-2nd half & post-match sprint performance significantly reduced compared with pre-match (2.4 & 2.0%, 0.11 & 0.09s)
al. (2004)	soccer players	KSA	3 x 30m half-time sprints, 25s recovery between	7min passive; 7min mod intensity, finishing ~1 min before 2nd half	Similar pre-match, post 1st half & pre 2nd half, significantly lower post-match (2.3%, 0.1s)
				CON	Significantly increased
Hajoglou et al.	8 M well- trained cyclists	3km-TT time	3km cycle erg TT	EWU	Significantly less than CON (2.8%, -7.6s)
(0007)	& Illauneres			NWH	Significantly less than CON (-2.6%, -7.1s)
				W1) 15min at aerobic threshold	
Bishop et al. (2001)	8 M elite kayak squad members	MPO	2min all-out kayak erg TT	W2) 15min 50%Δ	NS
~				W3) 15min at anaerobic threshold	
RCT: respirato VT/LT: ventilat	RCT: respiratory compensation threshold VT/LT: ventilatory/lactate threshold	I			
50%A: mid-wa	50%dl: mid-way between aerobic & anaerobic threshold מאסר א אדוסי יישר 2 שישים יישיעי שישייליטאי	robic threshold			
RSA: repeated sprint ability	peur a meun power ompu sprint ability	u, respectively	-		
MI, EI, WI: n	MI, EI, WI: moderately, endurance and well-trained, respectively	well-trained, respe	ectively		

Reference	Participants	Measurement Method	Prior Exercise	Results
Billat. Bocquet et	8 M: ET; 60 ±	$v\Lambda50 T_{im}$ mreceded by $2 \times 20min$	20min AT 50% vVO <sub>2max</sub> , 5min rest, $T_{lim}$ at 50% $\Delta$	Significantly greater (24.2%, 121s)
al. (2000)	l6km/week training volume	runs at diff intensities	20min alternating 30s at 100% & 30s at 50% vVO <sub>2max</sub> , 15min rest, $T_{lim}$ at 50% $\Delta$	Significantly reduced
Jones et al.	7 M: MT	Square-wave transitions from unloaded cycling to work rates of	6min bout at $50\%\Delta$	Significantly greater for 100, 110 & 120% VO <sub>2peak</sub> (37.0, 30.1 & 22.8%, 227, 66 & 41s)
(0007)		100, 110 & 120% VO <sub>2peak</sub>	no prior exercise (control)	Significantly reduced
Wilkerson et al.	7 M, recreationally	Square-wave tests: 3min baseline	MAX1: no prior exercise, 105% VO <sub>2peak</sub>	Significantly greater (18.5%, 28s)
(2004)	active	pedaling (20W), 105% VO <sub>2peak</sub> T <sub>lim</sub>	MAX2: 3x30s all-out cycling & 15min rest, 105% VO <sub>2peak</sub>	Significantly reduced
Stewart &	9 M senior rugby	Treadmill anaerobic capacity test	No warm-up (control) 60% VO <sub>2max</sub> , 15min rest	60% & 70% > control, 70% > 80%,
Sleivert (1998)	union players	(T <sub>lim</sub> at 13km/hr & 20% grade)	70% VO <sub>2max</sub> , 15min rest 80% VO <sub>2max</sub> , 15min rest	80% not different to control (actual values not given)

50%Δ: V2 way between GET & VO<sub>2peak</sub> νΔ50: V2way between vLT & vVO<sub>2peak</sub> T<sub>lm</sub>: exercise to exhaustion 64

# 5. Conclusions

Two of the key mechanisms that have been shown to change with increased intensity of prior exercise are [BLa] and  $\dot{V}O_2$  kinetics. This was seen in the increased [BLa] and decreased  $\tau$ , indicating a shorter MRT and thus accelerated  $\dot{V}O_2$  kinetics. The increased delivery and utilisation of  $O_2$  nearer the onset of exercise was beneficial in providing contribution of the  $O_2$  dependant energy systems sooner in the exercise, and the increased [BLa] also provided increased fuel for energy metabolism. However, if the priming intensity is excessive, it will be fatiguing, rather than assisting. Rather, the induced metabolic responses such as muscle/blood acidosis and high levels of [BLa] may actually inhibit muscle contractile ability and therefore, subsequent performance. However, if the priming intensity is too low and the duration is too short, or the duration of recovery between the priming exercise and performance is too great, then the effect of the priming may be lost.

Considering the data presented, there appears to be a fine line between a beneficial 'warmup' and one that is either not sufficient or one that causes too great a metabolic response and is fatiguing. Therefore it seems beneficial that the duration and intensity of the priming exercise is sufficient to obtain physiological responses that are elevated to near the magnitude of those to be expected during performance.

Directions for further research may also be considered from the evidence presented in this review. It would seem beneficial for further research to investigate not only the effect that increased temperature change has on performance, but also how much change in  $T_M/T_C$  is required for a positive change, and what magnitude of increase much would result in diminished performance. This would be achieved through more carefully controlled studies, with temperature changes (or the methods used to obtain temperature changes) as the independent variable, such as those by Burnley, Doust and Jones (2002) and Gray and Nimmo (2001). Similarly, the influence of [BLa] as a key determinant of performance does not as convincing with contrasting evidence presented in the review (Bishop et al., 2001; Bishop et al., 2003; Hajoglou et al., 2005; Jones et al., 2003), in addition to the evidence presented by Cairns (2006) questioning the role of lactate in exercise and its use as a

measure of performance. Further work to establish its contribution as a 'primer' is required.

It becomes apparent from this review that physiological responses can in fact differ greatly with respect to priming exercise. It would also be noted from this review that the specific duration, mode and intensity of the priming exercise would differ depending on the duration of the subsequent task, be it a largely anaerobic task (Stewart & Sleivert, 1998), perimaximal (Bishop et al., 2001; Bishop et al., 2003; Jones et al., 2003) or one that places high demand on not only the anaerobic energy pathways, but also large aerobic contribution (Hajoglou et al., 2005). Much of the priming exercise research to date however has neglected this performance element, highlighting the need for more research to help develop priming strategies that can be directly applied to specific performance, such as K1-500m Kayak (Bishop et al., 2001; Bishop et al., 2003) and track cycling distances (Hajoglou et al., 2005). Additionally, future research further investigating the intermittent nature priming strategies on specific performance events would also be warranted.

# **Chapter Three: Methods**

## 1. Participants

Ten well-conditioned endurance-trained male cyclists were recruited for this study from various cycling clubs within the greater Auckland area. Their characteristics are presented in *Table 3-1*. The majority of participants regularly competed in both road and 'endurance track' events, i.e. pursuit distances or greater. Participants were training regularly at the time of testing, with a minimum training volume of 200 kilometres per week. All participants were informed of the risks associated with the testing and the requirements of participation both verbally and in written form, and were given the opportunity to have any questions answered. Prior to participation, all participants gave their written informed consent in accordance with Auckland University of Technology's ethics committee, and completed a medical screening questionnaire.

Table 3-1: Participant anthropometric and physiological characteristics

Measure	Mean ± SD
Age (yr)	$28.3\pm8.4$
Body Mass (kg)	$81.8\pm11.6$
Height (m)	$1.8 \pm 0.1$
W <sub>peak</sub> (W)	$359.7\pm35.4$
$W_{peak} \cdot kg^{-1} (W \cdot kg^{-1})$	$4.5\pm0.6$
$\dot{V}O_{2peak} (L \cdot min^{-1})$	$4.6\pm0.5$
$\dot{VO}_{2peak} (mL \cdot kg^{-1} \cdot min^{-1})$	$57.5\pm8.6$
$HR_{peak}$ (b·min <sup>-1</sup> )	$185 \pm 12$
$[BLa]_{peak} (mmol \cdot L^{-1})$	$7.7 \pm 1.4$
Power at LT (W)	$228.0\pm21.0$
Power at LT (%W <sub>peak</sub> )	$63.7\pm6.7$
$\dot{V}O_2$ at LT (L·min <sup>-1</sup> )	$3.4 \pm 0.3$
$\dot{V}O_2$ at LT (% $\dot{V}O_{2peak}$ )	$74.3\pm9.9$
HR at LT $(b \cdot min^{-1})$	$149\pm10$
HR at LT (%HR <sub>peak</sub> )	$80.5\pm6.1$
Power at LTP (W)	$286.5\pm28.7$
Power at LTP (%W <sub>peak</sub> )	$79.8\pm5.2$
$\dot{V}O_2$ at LTP (L·min <sup>-1</sup> )	$4.1\pm0.3$
<sup>.</sup> VO <sub>2</sub> at LTP (% <sup>.</sup> VO <sub>2peak</sub> )	$88.4\pm5.5$
HR at LTP $(b \cdot min^{-1})$	$167.4\pm7.9$
HR at LTP (%HR <sub>peak</sub> )	$90.7\pm2.9$

## 2. Equipment

#### Ergometer

An electro-magnetically braked cycle ergometer (Velotron, Racermate Inc, Seattle, USA) was used for all physiological assessments, priming exercise and three kilometre time-trials (3km-TT). This model of ergometer utilises an electro-magnetic braking system to increase the amount of force applied to the rear wheel of the machine, where the assessor can predetermine the distance required to complete (as with the 3km-TT), or the level of resistance (i.e. power output) on the flywheel, independent of pedalling cadence (as with the incremental step test). This ergometer allowed for the design of different 'protocols' or 'courses', which in this study were the incremental step test and 3km-TT, respectively. It can also be in operated 'manual mode' whereby the participant is able to self-select the level of resistance, cadence and duration of exercise, as was the case in the priming portion of assessments.

### Pulmonary Gas Exchange

An automated breath-by-breath system (Metamax 3B, Cortex, Leipzig, Germany) was used to record pulmonary gas exchange measures during the incremental peak power test, the priming exercise and 3km-TT. This system has a proven reliability and validity when compared to the Douglas bag as the traditional gold standard measurement (Larsson, Wadell, Jakobsson, Burlin, & Hendriksson-Larsen, 2004).

Calibration of the gas analysis system was performed prior to all assessments, using a twopoint calibration procedure. This involved calibrating the apparatus to ambient air, which was assumed to be 20.93%  $O_2$  and 0.03%  $CO_2$ ; and then to a known mixture of hightolerance calibration gas (BOC, Auckland, NZ), composing of 14.82%  $O_2$  and 4.80%  $CO_2$ . Further 'checks' were then carried out following the two-point calibration to ensure the values of the surrounding environment were in the required range. The two-point calibration and ambient air 'checking' procedure was repeated until acceptable values ( $\pm$ 0.02%) were reached. Following the gas calibration, the flow-volume transducer was calibrated using a three litre syringe (Hans Rudolph, US). This calibration was verified with three different ventilation rates, according to the manufacturer's instructions.

#### Heart rate

Heart rate (HR) measurements during all testing sessions were carried out using a S625X Polar heart rate system (Polar Electro Oy, Kemplele, Finland). This device measures and records HR data at an interval of every five seconds. Manufacturers claim accuracy of heart rate measurements to be  $\pm 1\%$  or  $\pm 1$  b·min<sup>-1</sup> (which ever is larger) during steady-state conditions. The accuracy and reliability of polar heart rate monitors have been proven when compared to ECG measurements (Achten & Jeukendrup, 2003; Laukkanen & Virtanen, 1998).

#### Blood lactate

Blood, collected in 25  $\mu$ L capillary tubes of blood samples, was analysed for blood lactate concentration [BLa] using a YSI 1500 Sport analyser (Yellow Springs, Ohio). This device has proven reliability when compared with the Accusport and Lactate Pro lactate analysers in the analysis of various blood lactate transition thresholds (Buckley, Bourdon, & Woolford, 2003).

Prior to each testing session, calibration of the YSI lactate analyser was performed using a synthetic lactate solution of 5 mmol·L<sup>-1</sup> (YSI 2327). A check was then performed using the same 5 mmol·L<sup>-1</sup> solution to ensure that the values given were within an acceptable range  $(5.0 \pm 0.2 \text{ mmol·L}^{-1})$ . If the values were outside of this range, another calibration and check procedure was performed until the device was calibrated sufficiently to give acceptable values within the specified range. Checks of this nature were also carried out intermittently throughout assessments to ensure there was no drift in the calibration.

### Core Temperature

Core temperature ( $T_C$ ) was measured using a FirstTemp Genius tympanic thermometer (Intelligent Medical Systems, Carlsbad, California). This device is reported to be among the best of the infrared ear thermometers (IRET), providing the greatest ease in calibration and measurement, and uncertainty of only ±0.01%. It is the only IRET with the maximum permissible error in compliance with the requirements of the relevant standards in medical diagnostics (Pušnik & Drnovšek, 2005). To measure  $T_C$ , the probe was placed into the left ear cavity and the handle twisted inwards and towards the jaw to ensure a good seal, according to manufacturer's recommendations. A fan was not used during testing and cotton wool was placed into the ear between samples to prevent convective cooling (Hansen, Daley, & Leelarthaepin, 1993).

# 3. Experimental Procedures

All tests were performed at AUT's Human Performance Laboratory located at the Millennium Institute of Sport and Health. The laboratory is well ventilated and environmental conditions were standardised. The laboratory was temperature-controlled within the range 19-21°C, and humidity was consistently in the range of 60-75%.

In total, participants were required to visit the laboratory six times. The first visit involved an incremental step test and an initial 3km-TT familiarisation. The second visit was another 3km-TT familiarisation. Visits three to six were the experimental 3km-TT's, each preceded by a different priming exercise condition (randomised order). Prior to the first assessment, the participants own bike dimensions were measured and recorded so that the cycle ergometer resembled the rider's preferred set-up as closely as possible. This included crank length, and height, reach and angle of the handle bars and the height, fore and aft position and angle of the seat. Additionally, the participants own pedals were fitted to the cranks of the ergometer to allow them to ride in their own cycling shoes. Participants were then instructed to ride at a low to moderate resistance on the newly arranged set-up for one to two minutes to request any final minor adjustments before confirming the set-up. Once confirmed, this set-up was then recorded and held constant for all future testing sessions.

#### Incremental Step Test

The purpose of this assessment was to determine each participant's peak oxygen uptake  $(\dot{V}O_{2peak})$ , lactate threshold (LT), lactate turn-point (LTP) and peak aerobic power ( $W_{peak}$ ). Prior to the test, participants were required to sit down for five minutes so that both resting [BLa] and HR could be determined. Participants then started a ten-minute bout of priming exercise. Participants were instructed to start pedalling at a low power output (<100 W), and gradually increase power to, but not exceeding, 150 W. Following this priming exercise, participants were allowed five minutes in which to perform any last stretches, get a drink, etc.

The incremental step test started at 150 W and consisted of a "five-on, one-off" protocol. This protocol required participants to exercise at the required power output for five minutes, with one minute at 100 W in between each stage to allow for blood sampling for lactate analysis. HR was recorded in the last 15 seconds of each stage. Power output was progressively increased by 30 W per five minute stage until exhaustion. The test was terminated when the participant could no longer maintain a cadence of at least 60 rpm in a 30-second period or the participant stopped due to volitional exhaustion. Participant's were allowed to stand-up out of the saddle intermittently if a drop in cadence was starting to occur, but were encouraged to remain seated in the saddle for the majority of each stage, as well as maintaining a relatively constant cadence throughout the assessment.

The LT was defined as the first deflection point of 1 mmol·L<sup>-1</sup> or greater above baseline (Yoshida et al., 1987 as cited in Bosquet et al., 2002) in the [BLa] vs. power output curve. LTP was defined as the point in the [BLa] vs. power output curve before the observation of a second sudden and sustained increase in [BLa] that coincided with a [BLa] of between approximately 2-5 mmol·L<sup>-1</sup> (Hofmann, Bunc, Leitner, Pokan, & Gaisl, 1994; Midgley et al., 2006). To confirm the second deflection point, a modified D-max method of Cheng et al. (1992) was utilised. All LT and LTP analysis was performed by two independent examiners, and where discrepancies occurred, a third examiner was used.

 $\dot{V}O_{2max}$  represents the maximum achievable rate of oxygen (O<sub>2</sub>) during maximal exercise that can be inhaled and delivered to and used by the working muscles (Saltin & Astrand, 1967). It is often identified by a plateau in  $\dot{V}O_2$  despite continued increases in exercise intensity. However a plateau in  $\dot{V}O_2$  is not always observed; and along with the finding that different protocols (e.g. discontinuous step versus continuous ramp) and different exercise modes (e.g. cycle versus running) may yield different maximal results; it has been proposed that it may be more accurate to term this the peak, rather the maximum, rate of  $\dot{V}O_2$  (Day, Rossiter, Coats, Skasick, & Whipp, 2003): the  $\dot{V}O_{2peak}$ . Thus, for this study,  $\dot{V}O_{2peak}$  was defined as the peak O<sub>2</sub> value achieved during the incremental assessment, averaged over a 30 second period.

#### *Three kilometre time-trial (3km-TT)*

Laboratory-based time trials of varying distances have been previously shown to be both reliable and valid measures of cycling performance (Bishop, 1997; Coyle et al., 1991; Hickey et al., 1992; Paton & Hopkins, 2001). Additionally, Hajoglou et al. (2005) successfully utilised 3km-TT in a similar priming study. In an attempt to familiarise the participants with the laboratory-based 3km-TT protocol, two familiarisation trials took place. The first one was in the same initial visit in which the incremental step test was performed. Following exhaustion from the incremental test, participants were encouraged to 'spin' against a very low resistance (~50 W) for five minutes, followed by ten minutes at a power output corresponding to 50% of the W<sub>peak</sub>, followed by another five minutes of low resistance spinning. This low-intensity active recovery strategy was designed to facilitate recovery from the incremental assessment to allow effective participation in the 3k-TT familiarisation. This method has been shown to be effective and contribute towards reliable high intensity efforts following exhaustive incremental exercise in a recent study from our laboratory, of which the manuscript is currently being written (D. Bonetti, personal communication, 01 July 06). The second familiarisation session was carried out within a week of the incremental test. Participants were required to perform a priming exercise of self-selected duration and intensity and the 3km-TT with five minutes of passive rest in between the priming exercise and 3km-TT. These familiarisation sessions had the purpose of allowing the participants to become familiar with 1) riding on the Velotron ergometer,: 2) the high intensity nature of the 3km-TT; 3) the pacing strategy of the 3km-TT and 4) to help the participants determine the fixed-gear that they wished to use in all subsequent 3km-TT efforts. During all visits to the laboratory for 3km-TT's, participants were required to sit in an upright position for approximately five minutes so that resting [BLa], T<sub>C</sub> and HR could be measured. To avoid any influence of circadian variance, participants performed all 3km-TT's at the same time of the day.

## Pre-3km-TT priming exercise

The effect of four priming exercise conditions on 3km-TT performance were investigated in this study: one control condition of self-selected duration and intensity (i.e. the riders own preferred pre-competition priming exercise,  $WU_{con}$ ), and three, different 15 minute high intensity intermittent priming exercise conditions. These were similar in design to the priming strategies utilised by Bishop et al. (2003) and are schematically illustrated in *Figure 3-1.* Before the priming exercise commenced, participants were seated on the ergometer whilst resting  $\dot{V}O_2$  was measured. For the first ten minutes of these three priming exercise conditions, the intensity was set at a power output corresponding to half-way between the participant's own previously determined LT and LTP (50% $\Delta$ ). For the last five minutes, participants were required to perform five sprint efforts, each lasting ten seconds and separated by 50 seconds during which the participants were required to cycle at a power output corresponding to, but not exceeding, LT. The set workloads for the ten second sprint efforts in the three conditions were ~100% W<sub>peak</sub> (WU<sub>100%</sub>), ~150% W<sub>peak</sub> (WU<sub>150%</sub>) and 'all-out' (WU<sub>all-out</sub>). We had originally intended to assess a priming exercise condition with sprint intensities of 200% W<sub>peak</sub>, however in pilot work it was found that for most participants 'all-out' efforts were of a similar power output to 200% W<sub>peak</sub>, thus making that condition redundant. Following the intermittent priming exercise bouts, participants were given five minutes of passive rest, seated on the ergometer, prior to the subsequent 3km-TT.

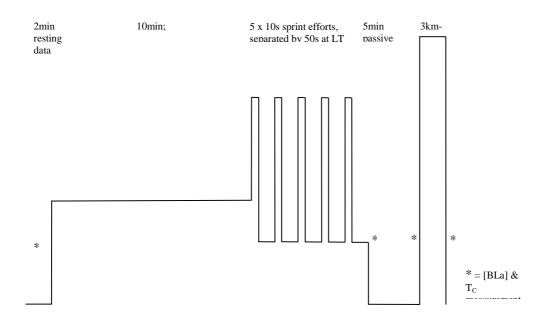


Figure 3-1: Experimental protocol

Participants were then given a one minute verbal warning before the start of the 3km-TT, and then a five second count-down. The start of the 3km-TT was a 'standing start', as would be the case in a track race in a velodrome, with the flywheel completely still. To minimise variation due to learning effects in pacing, an individualised pacing strategy was given to each participant. This required participants to generate as much power as they could in the first 15 seconds (which in reality was ~80% maximum effort for most) and a five to ten second transition to a power output that was 100-110% of W<sub>peak</sub>, which was maintained for the rest of the first minute. Although this 'all-out' sprint start was not necessarily representative of how participants would typically start an individual pursuit, we felt that it was a necessary and effective method to generate power quickly and consistently at the start of the 3km-TT. Pacing strategies similar to this have previously been used successfully and found to result in valid and reliable data (Bishop, 1997; Bishop et al., 2002). In addition to this, there is data on world class pursuit riders that show women and men reach similar power output values (~1000 watts) in the first ten seconds of the 3km and 4km individual pursuits, respectively (Jeukendrup, Craig, & Hawley, 2000), and some that reported that 4km team pursuit riders obtain average power of ~600 watts at the start of the trial (Broker et al., 1999), thereby providing justification for the 'all-out' sprint start of the 3km-TT. No verbal encouragement was given to participants at anytime during the 3km-TT following the initial 15 second build-up. To avoid influence of externally motivating factors the same two assessors were present for each individual participant. The only feedback provided to participants was distance covered at selected points. This was given in an emotionless voice and the feedback was exactly the same every time and included, "that's one kilometre", "that's one point five kilometres, half-way", "one kilometre to go", "last 500 metres", "400 metres", "300 metres", "200 metres", "100 metres" and "50 metres".

### 4. Data Analysis

#### Power calculations

As the stages of the peak power incremental test were five minutes in duration, final peak power ( $W_{peak}$ ) was measured as the participants' fully completed five minute stage plus any additional 30-seconds of the next stage worth an additional three watts. Following the end

of both the priming exercise and 3km-TT, mean power ( $W_{mean}$ ),  $W_{peak}$  and duration were recorded as displayed by the Velotron software.

#### Physiological variables

During all experimental 3km-TT's,  $T_C$  and [BLa] were recorded at rest, immediately post priming exercise, ~one minute and 15 seconds ([BLa] and  $T_C$ , respectively) prior to the start of the 3km-TT, and immediately following the 3km-TT.

All  $\dot{V}O_2$  and  $\dot{V}CO_2$  data was recorded breath-by-breath, and to reduce the effect of breathby-breath variability, outliers were removed using in-built functions in the Cortex software, thereby reducing the 'noise'. For determination of  $\dot{V}O_{2peak}$  data during the incremental assessment was averaged on a 30 second basis. Each participant's absolute  $\dot{V}O_{2peak}$  was recorded as the highest 30-second  $\dot{V}O_2$  value obtained during the test.

### **VO<sub>2</sub> Kinetics**

During all 3km-TT efforts,  $\dot{V}O_2$  was measured on a breath-by-breath basis. Data was collected 'non-stop' from resting values, during the priming exercise, the recovery following the priming exercise, and to the end of the trial. Two minutes of resting data was collected with the participants seated on the ergometer.  $\dot{V}O_2$  and HR values were recorded during and following the ten minute steady-state exercise portion of the priming exercise. Additionally, the maximum values following each ten second sprint, the values just before the start of the next sprint (i.e. how much it had recovered), immediately following the priming exercise, immediately prior to the 3km-TT (i.e. how much it had recovered) and immediately following the 3km-TT were recorded. Following both the priming exercise and 3km-TT the mean and maximum values were noted. Baseline values pre-priming exercise and 3km-TT were averaged on a two minute basis, with the  $\dot{V}O_{2peak}$  values during and the post-values following the priming exercise and 3km-TT averaged on a 30 second basis.

After removing the outliers in the breath-by-breath data, the data was interpolated to one second intervals to allow the fitting of models for examining the  $\dot{V}O_2$  kinetic response. A mono-exponential model, with time delay, was fit to the data. This model from time zero

has been used previously (e.g. Hajoglou et al., 2005; Whipp et al., 1981; Whipp et al., 1982). The reason for utilising a mono-exponential model rather than a multiple term model was that, due to the high intensity nature of the 3km-TT (mean power  $\geq W_{peak}$ ),  $\dot{V}O_2$  values were expected to reach their upper limits (i.e.  $\dot{V}O_{2peak}$ ) very early after the onset of exercise. The  $\dot{V}O_2$  response was modelled using the following equation:

$$\Delta \dot{V}O_2(t) = \Delta \dot{V}O_2(ss) \times (1 - e^{-t - \delta/\tau})$$

where  $\Delta \dot{V}O_2$  (t) and  $\Delta \dot{V}O_2$  (ss) are the changes in oxygen uptake at time (t) and from the previous steady-state control period (i.e. baseline values pre-trial), respectively,  $\delta$  is the time delay and  $\tau$  is the time constant of the  $\dot{V}O_2$  response (Whipp et al., 1981). In addition the mean response time (MRT) was determined by using mono-exponential constraint to start at exercise onset ( $\delta = 0$ ). The O<sub>2</sub> deficit was determined using the following formula: O<sub>2</sub> deficit = MRT ×  $\Delta \dot{V}O_2$  (ss).

#### **Statistics**

Once individual values had been obtained, they were entered into an MS Excel statistical spreadsheet designed by Hopkins (2003). Data was log-transformed and trials were compared using Paired *t*-tests, with priming exercise condition as the main effect, and 3km-TT time,  $P_{mean}$  and associated physiological variables as the independent variables of primary interest. [BLa],  $\dot{V}O_2$ , HR and  $T_C$  during the priming exercise and 3km-TT were also investigated using the *t*-tests to measure their relationship with priming exercise condition. Statistical significance was accepted at an alpha level of  $\leq 0.05$ . Qualitative inferences from the data were also determined, indicating if the effect was substantially positive and negative, or beneficial and harmful. Paton and Hopkins (2001) have estimated smallest worthwhile effects in cycle performance assessments of 0.5-1.5% in W<sub>mean</sub>. This was based on the variability reported in competitive performance of elite cyclists in various time trials where drafting and group tactics did not contribute. Therefore, for the purposes of the current investigation, an estimate of the smallest substantial change in the key variable was required. To make these inferences, this was assumed to be 1%.

# **Chapter Four: Results**

Presented in this section is a description of the main results. A full statistical report on all differences, including means  $\pm$  SD, *p* values, percent change, percentage SD of change and confidence limits can be found in Appendix 1. Unless stated otherwise, all statistical values relating to percentage change and significant difference are log-transformed values.

*Table 4-1:* Power output related variables during the priming phase of each condition (Mean  $\pm$  SD)

	1			/
	WU <sub>con</sub>	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
Time (m:s)	$15:33 \pm 04:43$	15:00	15:00	15:00
Time range	8:26 - 20:00	-	-	-
W <sub>mean</sub> (w)	222.5	250.7 <sup>a</sup>	263.2 <sup>c</sup>	264.0 <sup>a</sup>
W <sub>peak</sub> (w)	496.0	383.6 <sup>a</sup>	553.6 <sup>b</sup>	658.1 <sup>d</sup>
<sup>a</sup> C:		05)		

<sup>a</sup> Significantly different from  $WU_{con}$  (p < 0.05)

<sup>b</sup> Significantly different from  $WU_{100\%}$  (p < 0.05)

<sup>c</sup> Significantly different from WU<sub>con</sub> and WU<sub>100%</sub> (p < 0.05)

<sup>d</sup> Significantly different from all other conditions (p < 0.05)

The priming phase of WU<sub>con</sub> was on average 33 seconds (3.5%) longer than each of the controlled intermittent sprint priming conditions, although it ranged from ~8.5 to 20min (*Table 4-1*). W<sub>mean</sub> (W<sub>peak</sub>) in WU<sub>con</sub> was 11.2 (-29.3), 15.5 (10.4) and 15.7% (24.6%) less than WU<sub>100%</sub>, WU<sub>150%</sub> and WU<sub>all-out</sub>, respectively.

# 1. Performance measures (time, mean power output, W<sub>mean</sub>)

The mean ( $\pm$  SD) power, and its associated variables, for the 3km-TT are presented in *Table 4-2*. No significant differences in 3km-TT performance ( $W_{mean}$  or time) were observed between  $WU_{con}$ ,  $WU_{100\%}$  or  $WU_{150\%}$  for  $W_{mean}$ . This was also apparent for the first half of the trial when considered separately. Conversely, the  $WU_{all-out}$  priming condition resulted in substantial reductions in both  $W_{mean}$  and  $W_{mean}$  for the whole trial and first half of the trial ( $W_{mean} - \frac{1}{2} way$ ) when compared to all other priming conditions. There was a -5.8% change in  $W_{mean}$  between  $WU_{all-out}$  and  $WU_{con}$  (*Table 4-3*). The qualitative inference associated with the 90%CL (-8.3 - -3.1\%) was 'almost certainly' negative.

	WU <sub>con</sub>	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
Time (m:s)	$04{:}18.2\pm00{:}11.3$	$04{:}19.2\pm00{:}12.9$	$04{:}19.6 \pm 00{:}13.6$	$04:23.8 \pm 00:13.6$ <sup>c</sup>
Time (s)	$258.2 \pm 11.3$	$259.2 \pm 12.9$	$259.6 \pm 13.6$	$263.8 \pm 13.5$ <sup>c</sup>
W <sub>peak-TT</sub>	$634.0 \pm 149.9$	$640.7\pm176.4$	$619.5\pm171.7$	$575.4 \pm 117.0$ <sup>b</sup>
W <sub>mean</sub>	$378.6 \pm 44.0$	$376.3 \pm 44.9$	$373.9\pm47.8$	$357.4 \pm 44.5$ <sup>c</sup>
$\mathbf{W}_{ ext{final}}$	$398.7\pm57.1$	$388.8\pm57.8$	$389.2\pm63.9$	$372.4 \pm 68.9$ <sup>a</sup>
Time <sup>1/2</sup> way (m:s)	$02:09.9 \pm 00:05.4$	$02:10.3 \pm 00:06.5$	$02{:}10.0\pm00{:}06.6$	$02:11.9 \pm 00:06.2$ <sup>c</sup>
Time $\frac{1}{2}$ way (s)	$129.9\pm5.4$	$130.3\pm6.5$	$130.0\pm6.6$	$131.9 \pm 6.2$ <sup>c</sup>
$W_{mean-^{1\!\!/_2}way}$	$381.5\pm41.1$	$379.9 \pm 45.7$	$382.3\pm47.5$	$365.9 \pm 42.3$ <sup>c</sup>
W 1/2 way	$366.7\pm41.7$	$367.9\pm45.3$	$363.1\pm43.4$	$345.7 \pm 37.5$ <sup>c</sup>

*Table 4-2:* Power output related variables during the 3km-TT in relation to priming exercise condition (Mean  $\pm$  SD)

<sup>a</sup>Significantly different from WU<sub>con</sub> (p < 0.05)

<sup>b</sup> Significantly different from both  $WU_{con}$  and  $WU_{100\%}$  (p < 0.05)

<sup>c</sup> Significantly different from all other conditions (p < 0.05)

Table	<i>4-3</i> :	Log	transformed	mean	changes	in	performance	(W <sub>mean</sub> )	from	control	(WU <sub>con</sub> )	between
	cor	ndition	ns and chance	s that tl	ne true dif	fere	ence in the cha	inges is s	ubstant	tial		

Condition	WU <sub>100%</sub>	WU <sub>150%</sub>	$\mathbf{WU}_{\mathbf{all-out}}$
Change in mean (%)	-0.7	-1.5	-5.8
<b>SD</b> of change (%)	3.9	4.2	4.7
90% confidence limits (%)	-3.0 - 1.6	-3.8 - 1.0	-8.33.1
P value	0.5713	0.2955	0.0033
Practical inference <sup>‡</sup>	unclear	unclear	almost certainly harmful

<sup>\*</sup> Based on a smallest worthwhile beneficial or harmful change in performance of 1%

The  $W_{mean}$  and time for each 500 m segment of the 3km-TT are reported in *Table 4-4*, with a corresponding graph of  $W_{mean}$  in *Figure 4-1*. No significant differences in  $W_{mean}$  existed at any stage throughout the 3km-TT amongst  $WU_{con}$ ,  $WU_{100\%}$  and  $WU_{150\%}$ . However,  $W_{mean}$  in the  $WU_{all-out}$  condition was significantly reduced compared to the other conditions in all but the second 500m (500-1000m). In the first two 500m segments there was no difference between  $WU_{con}$  and  $WU_{all-out}$  (p = 0.0724 and 0.2143; 0-500m and 500-1000m, respectively), but it can be observed in *Figure 4-1* and *Table 4-4* that this difference is still quite substantial, as demonstrated by a difference in  $W_{mean}$  of 25.9 Watts (6%). These two conditions had the greatest difference in  $W_{mean}$  by the end of the 3km-TT. The final 500m of the 3km-TT was the segment where the greatest difference in  $W_{mean}$  was observed between  $WU_{all-out}$  and the other conditions.

exercise con	$101110n (Mean \pm SD)$			
	WU <sub>con</sub>	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
Segment (m)				
		Mean Power	Output (w)	
0-500	$404.1\pm 66.9$	$406.4\pm77.1$	$406.5\pm85.0$	$378.2 \pm 61.4$ <sup>b</sup>
500-1000	$369.9\pm33.5$	$366.5\pm32.7$	$368.8\pm37.5$	$363.0\pm38.8$
1000-1500	$371.0\pm36.8$	$367.9\pm41.8$	$366.4\pm39.7$	$352.4 \pm 40.0$ <sup>c</sup>
1500-2000	$367.2\pm45.3$	$364.7\pm49.2$	$358.0\pm49.0$	$345.0 \pm 46.5$ <sup>a</sup>
2000-2500	$371.8 \pm 49.3$	$368.3\pm55.2$	$363.5\pm57.1$	$350.2 \pm 56.7$ <sup>a</sup>
2500-3000	$389.3\pm51.8$	$385.7\pm47.5$	$384.9\pm67.8$	$357.7 \pm 55.5$ <sup>c</sup>
Distance (m)		Tim	e (s)	
500	$44.4\pm2.7$	$44.5\pm3.5$	$44.2\pm3.8$	$45.1 \pm 3.0$ <sup>b</sup>
1000	$87.3\pm3.9$	$87.6\pm4.7$	$87.0\pm5.1$	$88.2\pm4.6$
1500	$130.1\pm5.4$	$130.6\pm6.4$	$130.1\pm6.7$	$119.3 \pm 42.3$ <sup>c</sup>
2000	$173.2 \pm 7.3$	$173.8\pm8.6$	$173.6\pm8.8$	$176.1 \pm 8.4$ <sup>a</sup>
2500	$216.1\pm9.4$	$217.0 \pm 10.9$	$217.1 \pm 11.1$	$220.2 \pm 11.0^{a}$
3000	$258.3 \pm 11.3$	$259.3 \pm 12.9$	$259.7 \pm 13.6$	$263.8 \pm 13.5$ <sup>c</sup>
3000	$258.3 \pm 11.3$	$259.3 \pm 12.9$	$259.7 \pm 13.6$	$263.8 \pm 13.5$ °

Table 4-4: Mean power output and time at 500m segments during the 3km-TT in relation to priming exercise condition (Mean  $\pm$  SD)

<sup>a</sup> Significantly different from both  $WU_{con}$  and  $WU_{100\%}$  (p < 0.05) <sup>b</sup> Significantly different from both  $WU_{100\%}$  and  $WU_{150\%}$  (p < 0.05)

° Significantly different from all other conditions (p < 0.05)

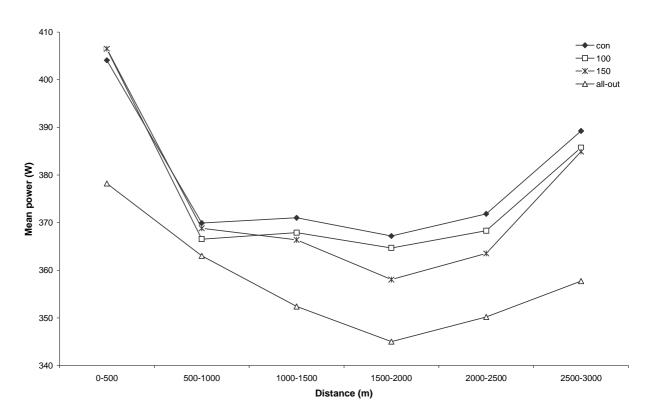
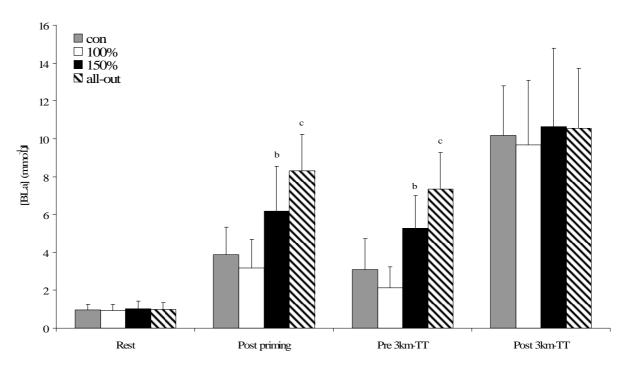


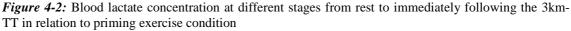
Figure 4-1: Mean power output at 500m segments during the 3km-TT in relation to priming exercise condition. For clarity, SD and individual significant differences are not displayed on this graph but are summarized in Table 4-3.

# 2. Physiological parameters

#### Blood lactate concentration ([BLa])

There were no significant differences between conditions with respect to resting [BLa] (*Figure 4-2*). Immediately following the priming exercise, the [BLa] for WU<sub>con</sub> and WU<sub>100%</sub> were not significantly different (p = 0.2863). However, both WU<sub>150%</sub> and WU<sub>all-out</sub> resulted in significantly greater [BLa] post-priming than both WU<sub>con</sub> (p = 0.0225 and 0.0006, respectively) and WU<sub>100%</sub> (p = 0.002 and 0.0004, respectively). In addition, a greater post-priming [BLa] for WU<sub>all-out</sub> compared to WU<sub>150%</sub> (p = 0.0203) was observed. Following the five minutes passive rest after priming, WU<sub>150%</sub> [BLa] was still significantly elevated in comparison to WU<sub>con</sub> (p = 0.032) and WU<sub>100%</sub> (p = 0.0002), and WU<sub>all-out</sub> was elevated in comparison to all other conditions (p = 0.015, 0.0001 and 0.0151 for WU<sub>con</sub>, WU<sub>100%</sub> and WU<sub>150%</sub>, respectively). No significant differences in [BLa] were apparent immediately post 3km-TT.





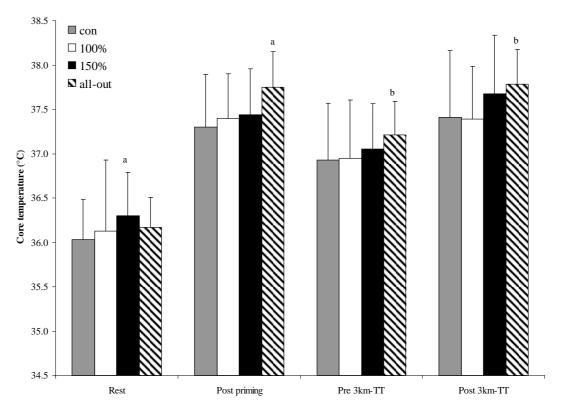
<sup>a</sup> Significantly different from WU<sub>con</sub> (p < 0.05)

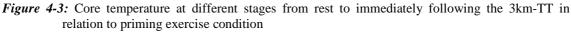
<sup>b</sup> Significantly different from both WU<sub>con</sub> and WU<sub>100%</sub> (p < 0.05)

<sup>c</sup> Significantly different from all other conditions (p < 0.05)

### Core temperature $(T_C)$

Resting T<sub>C</sub> values in WU<sub>150%</sub> were greater than WU<sub>con</sub> (p = 0.0423; *Figure 4-3*). No other differences in resting T<sub>C</sub> existed between conditions. WU<sub>all-out</sub> resulted in significantly (p = 0.0347) greater post-priming T<sub>C</sub> than WU<sub>con</sub>. The pre-3km-TT T<sub>C</sub> for WU<sub>all-out</sub> was also greater than WU<sub>100%</sub> (p = 0.0482). Following the 3km-TT the only significant difference was the T<sub>C</sub> for WU<sub>all-out</sub> compared with WU<sub>100%</sub> (p = 0.017).

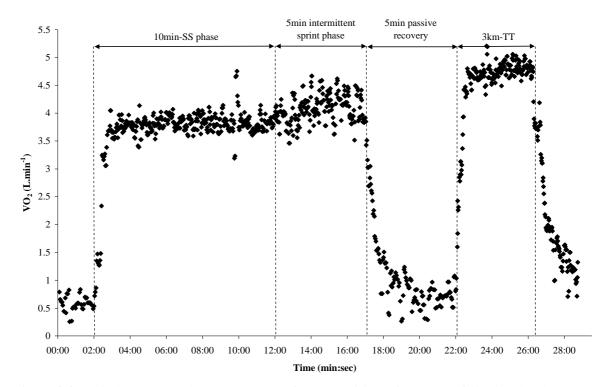




- <sup>a</sup> Significantly different from  $WU_{con}$  (p < 0.05)
- <sup>b</sup> Significantly different from  $WU_{100\%}$  (p < 0.05)

*Oxygen uptake* ( $\dot{V}O_2$ )

An example of a typical  $\dot{V}O_2$  response for one participant to the three intermittent sprint priming conditions can be observed in *Figure 4-4*.

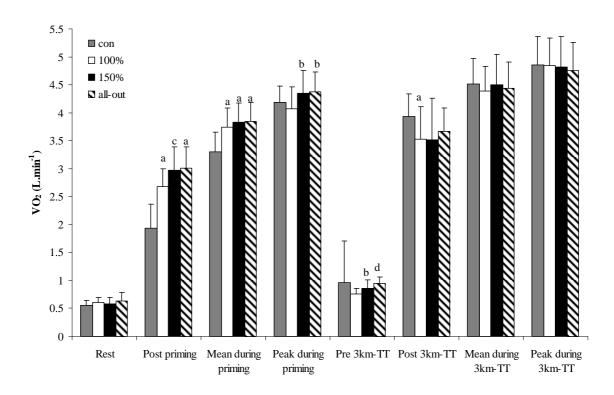


*Figure 4-4:* Typical oxygen uptake (VO<sub>2</sub>) response for one participant from rest to following the 3km-TT (WU<sub>150%</sub>)

Grouped means for  $\dot{VO}_2$  at each stage from rest to following the 3km-TT are displayed in *Figure 4-5*. No differences were observed between conditions for resting  $\dot{VO}_2$  values (p > 0.05). In addition, no differences were observed between conditions for mean  $\dot{VO}_2$ during or following the 10 minute steady-state ( $W_{mean} = \sim 50\%\Delta$ ) portion of each priming strategy.

WU<sub>con</sub> displayed significantly reduced  $\dot{VO}_2$  than all other priming conditions both in the 30 seconds post-priming (p = 0.0005, 0.0005 and 0.0006 for WU<sub>100%</sub>, WU<sub>150%</sub> and WU<sub>all-out</sub>, respectively) and as a mean over the duration of the priming exercise (p = 0.0166, 0.0024 and 0.0024 for WU<sub>100%</sub>, WU<sub>150%</sub> and WU<sub>all-out</sub>, respectively). WU<sub>150%</sub> was significantly greater than WU<sub>100%</sub> (p = 0.0124) for  $\dot{VO}_2$  post-priming exercise. WU<sub>100%</sub> displayed a reduction in  $\dot{VO}_2$  post-priming compared to WU<sub>all-out</sub> that approached significance (p = 0.052). Peak values attained during the WU<sub>100%</sub> priming

exercise were significantly reduced compared to  $WU_{150\%}$  (p = 0.0007) and  $WU_{all-out}$  (p = 0.016).



*Figure 4-5:* Oxygen uptake (VO<sub>2</sub>) at different stages from rest to following the 3km-TT in relation to priming exercise condition

<sup>a</sup> Significantly different from  $WU_{con}$  (p < 0.05)

<sup>b</sup> Significantly different from  $WU_{100\%}$  (p < 0.05)

<sup>c</sup> Significantly different from both WU<sub>con</sub> and WU<sub>100%</sub> (p < 0.05)

<sup>d</sup> Significantly different from both  $WU_{100\%}$  and  $WU_{150\%}$  (p < 0.05)

Following the five minutes of passive rest seated on the ergometer,  $WU_{150\%}$  VO<sub>2</sub> (averaged over the final two minutes) was significantly elevated (p = 0.0275) compared to  $WU_{100\%}$ .  $WU_{all-out}$  was significantly elevated compared to both  $WU_{100\%}$  (p = 0.0002) and  $WU_{150\%}$  (p = 0.0249), but not compared to  $WU_{con}$  (p = 0.4083).

The  $\dot{V}O_2$  post-3km-TT (30 second average) was significantly different between WU<sub>100%</sub> and WU<sub>con</sub> (p = 0.0207). No significant differences were observed for 3km-TT mean or peak  $\dot{V}O_2$ . Change in the mean (±90% CL) for  $\dot{V}O_{2peak}$  during the 3km-TT relative to WU<sub>con</sub> equated to -0.4 (± 5.5), -0.9 (±2.9) and -2.0% (±3.1) for WU<sub>100%</sub>, WU<sub>150%</sub> and WU<sub>all-out</sub>, respectively (*Table 4-5*). The chances of negative outcome associated with these confidence limits were 42, 47 and 73% for WU<sub>100%</sub>, WU<sub>150%</sub> and WU<sub>all-out</sub>, respectively, with all displaying a qualitative inference of "unclear", indicating that it

could not be concluded with any certainty that the results were due to a true change in  $T_{\rm C}$ .

Condition	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
Change in mean (%)	-0.4	-0.9	-2.0
<b>SD</b> of change (%)	9.3	4.9	5.2
90% confidence limits (%)	5.6 - 5.1	-3.7 - 2.0	-4.9 - 1.0
<i>p</i> value	0.903	0.5851	0.2499
<b>Practical inference<sup>‡</sup></b>	Unclear	Unclear	Unclear

*Table 4-5:* Log transformed mean changes in VO<sub>2peak</sub> during the 3km-TT from control (WU<sub>con</sub>) between conditions and chances that the true difference in the changes is substantial

<sup>‡</sup> Based on a smallest worthwhile beneficial or harmful change in performance of 1%

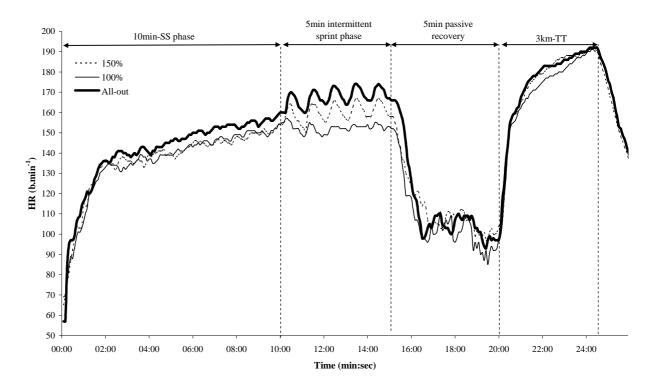
#### Heart rate (HR)

A typical HR response for one participant to the three intermittent sprint priming conditions can be observed in *Figure 4-6*. Qualitatively, it can be observed that each of the prescribed intermittent sprint priming strategies ( $WU_{100\%}$ ,  $WU_{150\%}$  and  $WU_{all-out}$ ) follow the same pattern, but with greater HR values during each successive priming phase ( $WU_{100\%} < WU_{150\%} < WU_{all-out}$ ). During the five minute recovery phase and the 3km-TT, HR values were similar between  $WU_{100\%}$ ,  $WU_{150\%}$  and  $WU_{all-out}$ .

There were no significant differences (p > 0.05) between WU<sub>100%</sub>, WU<sub>150%</sub> and WU<sub>all-out</sub> for resting HR, or for HR following (or averaged over) the 10 minute steady-state portion of the priming exercise (*Figure 4-7*). Immediately following the priming exercise, HR for WU<sub>con</sub> was significantly reduced compared to all other conditions (p = 0.0101, 0.0054 and 0.0069 for WU<sub>100%</sub>, WU<sub>150%</sub> and WU<sub>all-out</sub>, respectively). When HR data was presented as a mean over the duration of the whole priming exercise, both WU<sub>100%</sub> (p = 0.0387) and WU<sub>150%</sub> (p = 0.0046) were significantly greater than WU<sub>con</sub>. However, WU<sub>100%</sub> and WU<sub>150</sub> (p = 0.2145) were not significantly different from each other (p = 0.5306) or WU<sub>all-out</sub> (p = 0.4771 and 0.5825, WU<sub>100%</sub> and WU<sub>150%</sub>, respectively), nor was WU<sub>con</sub> different from WU<sub>all-out</sub> (p = 0.2145). The log-transformed means associated with these significant changes resulted in increases that were "very likely" (WU<sub>100%</sub>) and "almost certainly" (WU<sub>150%</sub>) substantial true changes due to the effects of the condition (% change: 10.5 and 11.5%; 90% CL: 2.7 – 18.9 and 6.0 – 17.3; WU<sub>100%</sub> and WU<sub>150%</sub>, respectively with smallest worthwhile change of  $\pm 1\%$ ).

The peak HR attained during priming exercise and the  $HR_{mean}$  in the last two min of recovery for the  $WU_{100\%}$  condition was significantly reduced in comparison to the

WU<sub>150%</sub> (p = 0.0281) and WU<sub>all-out</sub> (p = 0.0125) conditions. WU<sub>100%</sub> was the only significantly different HR value pre-3km-TT, being significantly less than WU<sub>150%</sub> (p = 0.0179) and WU<sub>all-out</sub> (p = 0.0088). Unlike the HR<sub>mean</sub> of the priming exercise, no true changes in HR<sub>peak</sub> attributable to priming condition were apparent from the 3km-TT, as observable in *Table 4-6*.

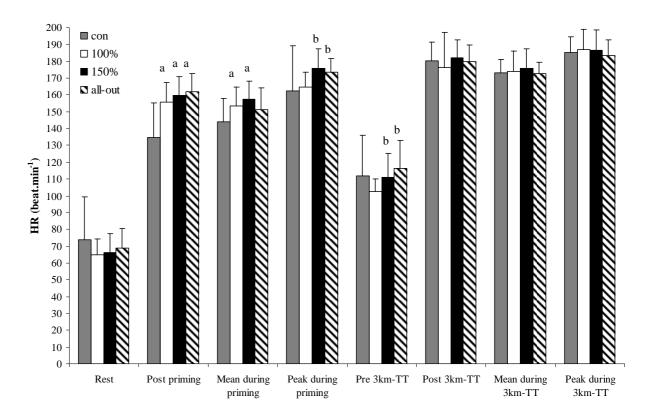


*Figure 4-6:* Typical heart rate (HR) response for one participant from rest to following the 3km-TT for each intermittent-sprint priming condition

Table 4-6: Log transformed mean changes in HR <sub>peak</sub> during the 3km-TT from control (WU <sub>con</sub> ) between	
conditions and chances that the true difference in the changes is substantial	

Condition	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
Change in mean (%)	0.7	-0.1	-1.0
<b>SD of change (%)</b>	1.5	2.2	2.6
90% confidence limits (%)	0.3 – 1.6	-1.4 - 1.3	-2.4 - 0.5
<i>p</i> value	0.2227	0.9125	0.2578
Practical inference <sup>‡</sup>	Unclear	Unclear	Unclear

<sup>‡</sup> Based on a smallest worthwhile beneficial or harmful change in performance of 1%



*Figure 4-7:* Heart rate (HR) at different stages from rest to following the 3km-TT in relation to priming exercise condition <sup>a</sup> Significantly different from  $WU_{con}$  (p < 0.05) <sup>b</sup> Significantly different from  $WU_{100\%}$  (p < 0.05)

# Oxygen uptake ( $\dot{V}O2$ ) kinetics

Despite a trend for reduced  $\tau$  and MRT after WU<sub>100%</sub>, WU<sub>150%</sub> and WU<sub>all-out</sub> (compared to WU<sub>con</sub>), there were no statistical differences (*Table 4-7*). However, the percent change in mean (and 90% CL) suggests a "likely" chance of true difference in the negative direction (i.e. a beneficial reduction).

The O<sub>2</sub> deficit for WU<sub>150%</sub> was significantly reduced in comparison to WU<sub>con</sub> (p = 0.0249). The 90%CL for this change were -30.1 - -6.5, and a qualitative inference of "very likely" change in the negative direction (i.e. a beneficial reduction; *Table 4-8*).

*Table 4-7:* VO<sub>2</sub> kinetics parameters during the 3km-TT in relation to priming exercise condition (Mean ± SD)

	WU <sub>con</sub>	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
Tau (s)	$15.9\pm5.1$	$13.4\pm4.5$	$12.5\pm4.5$	$13.0\pm3.4$
MRT (s)	$19.3 \pm 4.2$	$18.3\pm6.1$	$16.5 \pm 2.7$	$17.2\pm2.2$
$O_2$ Def (L)	$73.3 \pm 18.6$	$66.5\pm23.3$	$59.4 \pm 15.6$ <sup>a</sup>	$60.7\pm9.3$
<sup>a</sup> C:: C: 1: C	C NULL (	0.05)		

<sup>a</sup> Significantly different from  $WU_{con}$  (p < 0.05)

Condition	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
		Tau	
Change in mean (%)	-16.8	-22.9	-17.1
SD of change (%)	32.9	57.2	38.2
90% confidence	-31.2 - 0.7	-44.7 - 7.4	-33.6 - 3.4
limits (%)			
<i>p</i> value	0.1108	0.1839	0.1549
Practical inference <sup>‡</sup>	Likely beneficial	Likely beneficial	Likely beneficial
		Mean Response Time	
Change in mean (%)	-8.2	-13.7	-9.4
SD of change (%)	36.7	21.9	24.9
90% confidence	-25.8 - 13.6	-24.02.0	-21.6 - 4.7
limits (%)			
<i>p</i> value	0.4801	0.0624	0.2421
Practical inference <sup>‡</sup>	Possibly beneficial	Very likely beneficial	Likely beneficial
		Oxygen Deficit	
Change in mean (%)	-12.0	-19.2	-15.3
SD of change (%)	38.8	25.1	30.4
90% confidence	-29.7 - 10.2	-30.16.5	-29.0 - 0.9
limits (%)			
p value	0.3254	0.0249	0.1168
Practical inference <sup>‡</sup>	Likely beneficial	Very likely beneficial	Likely beneficial

*Table 4-8:* Log transformed mean changes in VO<sub>2</sub> kinetics variables from control (WU<sub>con</sub>) between conditions and chances that the true difference in the changes is substantial

<sup>‡</sup> Based on a smallest worthwhile beneficial or harmful change in performance of 1%

# **Chapter Five: Discussion**

Previous studies have established that a high intensity intermittent sprint priming strategy was more effective than continuous priming exercise in improving short duration (~2min) kayak performance (Bishop et al., 2001; Bishop et al., 2003). It is currently unclear what the optimal intermittent intensity is for short duration cycle performance i.e. as intermittent priming strategies are more effective than continuous, would such high intensity sprint efforts (~200%  $\dot{V}O_{2peak}$ ) be required, or would a lower intensity be just as, or more, effective? Thus, the aim of the present study was to determine the effect of three different intermittent priming ('warm up') strategies (varying in intensity) on laboratory-based 3km-TT cycle performance in moderately-well trained male endurance cyclists. Using the participants preferred or self-chosen 'warm up' as the control condition also allowed us to determine which priming strategies were better (or worse) than how the participants would normally prepare for an event of similar nature.

The main finding of this study suggests that 3km-TT performance (time and  $W_{mean}$ ) is dependent on the intensity of the priming strategy employed. Overall, it was observed that  $WU_{con}$  (self-chosen) was the most effective priming strategy in preparing the participants for performance in the 3km-TT. The lowest ( $WU_{100\%}$ ) and medium ( $WU_{150\%}$ ) intermittent intensity priming strategies were similar to  $WU_{con}$ , and each other, in terms of subsequent performance. In contrast, the highest intensity priming condition ( $WU_{all-out}$ ) was clearly the least effective in preparing the participants for subsequent performance. This condition resulted in significantly reduced  $W_{mean}$  (and increased time) compared to all other priming conditions.

# 1. Performance Measures

On average,  $WU_{con}$  was the priming condition that exhibited the best performance (highest  $W_{mean}$  and fastest time), although it should be acknowledged that its improvement compared to  $WU_{100\%}$  and  $WU_{150\%}$  was not significant (*Table 4-4*). All three of these conditions resulted in 3km-TT performances that were significantly better than  $WU_{all-out}$ . Interestingly, the current study showed that a very high intensity preparation was detrimental to 3km-TT cycle ergometer performance. Clearly, the small SD of percent

change in  $W_{mean}$  for  $WU_{all-out}$  in relation to  $WU_{con}$  (4.7%), in addition to the negative range of the 90%CL (-8.3 - -3.1), provides strong evidence that the WUall-out priming condition would 'almost certainly' be harmful to 3km-TT performance (Table 4-4). This observation is in contrast to the previous findings of Bishop et al. (2003), where priming exercise using a similar intermittent protocol and relative intensity to  $WU_{all-out}$  (~200% ~ $\dot{V}O_{2peak}$ ) improved 2min-TT kayak ergometer performance in relation to a continuous priming exercise at 50% $\Delta$  (~65% VO<sub>2peak</sub>). Possible reasons for the inconsistency with the study of Bishop et al. (2003) may include the greater duration of the TT in the current study (>4min for 3km cycle TT vs. 2min kayak TT) and the greater muscle mass involved in cycling compared to kayaking. This would potentially result in greater fatiguing effect of the priming exercise, potentially due to a more extreme shift in the blood/muscle acidosis and/or energy cost of exercise. Evidence of this may be observed in the differences in [BLa] following the most intense priming exercise conditions in the current study (8.31  $\pm$ 1.93 mmol·L<sup>-1</sup>) and that of Bishop et al. (2003) (~3-4 mmol·L<sup>-1</sup>). With  $WU_{con}$ ,  $WU_{100\%}$  and WU<sub>150%</sub>, the elevated [BLa] is likely to have improved performance, compared to if there was no priming exercise, by protecting against fatigue caused by the shift in muscle  $K^+$ (Nielsen et al., 2001). However, too great a shift in muscle  $K^+$  that would accompany the large increase in [BLa] in WU<sub>all-out</sub> likely caused an inhibition of muscle contractile function (Bishop et al., 2001; Cairns, 2006), thus in part explaining the reduced performance. Numerous previous studies have demonstrated that a degree of acidemia is required to accelerate the VO<sub>2</sub> kinetic response (e.g. Billat, Bocquet et al., 2000; Burnley, Doust, & Jones, 2002; Gerbino et al., 1996). However, if this acidemia is excessive, regardless of any speeding of the kinetic response, muscle performance is likely to be impaired. It has yet to be established what the optimal [BLa] is for priming purposes, and debate surrounds the common assertion that [BLa] is a performance inhibitor (Cairns, 2006; Gladden, 2000; Nielsen et al., 2001; Westerblad et al., 2002).

Parts of the results from the present study are consistent with some of those previously reported by Hajoglou et al. (2005). For example, there was no difference in power output between the two active priming conditions when the power output in each 500m segment was considered. However, both active priming conditions significantly improved compared to the control condition up to 2000m. In the current study, there was approximately 7%

difference in W<sub>mean</sub> for the first two 500m segments between the two conditions that exhibited the greatest difference in whole trial W<sub>mean</sub> (WU<sub>con</sub> and WU<sub>all-out</sub>), however given the variability these were not significant. This provides some indication that both conditions were not able to generate similar amounts of force in the early stages of the trial, but not to the same level of confidence as reported by Hajoglou et al. (2005). Surprisingly however in contrast to Hajoglou et al. (2005), the detrimental effects of WU<sub>all-out</sub> were most evident in the second half of the 3km-TT, especially the final 500m where participants were unable to increase their power output for a 'strong finish' in comparison to the other priming conditions (Table 4-3; Figure 4-1). This particular finding disagrees with that reported by Hajoglou et al. (2005), who noted that the greatest benefit of both the easy and hard priming exercises when compared to the control was in the early stages of the 3km-TT, and that after 3-4 minutes of heavy exercise, the response pattern is was very similar whether prior exercise was performed or not. To summarise, both Hajoglou et al. (2005) and the current study reported no difference in power output at each 500m with all but one condition which performed markedly worse, but Hajoglou et al. (2005) reported the performance improvements early in the 3km-TT, whereas the current study demonstrated improved performance in the latter stages.

In light of the inconsistencies between the two studies, there are a number of potential explanations. Firstly, large individual variations were evident in the  $W_{peak-TT}$  in the present study, which due to the pacing strategy of this study occurred in the first 500m, and the ability to generate high forces early in the 3km-TT would have affected the  $W_{mean}$  in those early stages. This would result in large SD's, thus reducing the ability of the *t*-test to detect significant differences in  $W_{mean}$ . This explanation is in agreement with Bishop et al. (2001), who stated that the large individual variations in  $W_{mean}$  between trials reduced the power of the ANOVA in detecting significant findings. Secondly, Hajoglou et al. (2005) did not make use of a pacing strategy, whereas the current study did. This may have affected the anaerobic dependence at both the early and latter stages of the 3km-TT. Indeed, both the anaerobic contribution during 4km-TT (Hettinga, De Koning, Broersen, van Geffen, & Foster, 2006) and O<sub>2</sub> deficit (Foster et al., as cited in Craig et al., 1995) have been shown to be dependant on pacing strategy, and Hajoglou et al. (2005) acknowledged the possibility of the differing pacing strategies between conditions affecting the different  $\dot{V}O_2$  kinetics.

In the study of Hettinga et al. (2006), different pacing strategies in the individual pursuit (4km-TT) were investigated with respect to energy system contribution at various stages and resulting power output. The strategy that was most similar to the one used in the current study is that with an increasing power profile for the first 2000m, and this was reported to result in decreased power attributable to anaerobic energy sources in the second half compared to the first. This is in contrast to the even paced strategy, which is most similar to that used by Hajoglou et al. (2005). The even paced strategy was reported by Hettinga et al. (2006) to result in increased power attributable to anaerobic energy sources in the second half compared to the first; thus potentially explaining Hajoglou et al. (2005) reporting that early the stages were more deterministic of overall 3km-TT performance, in comparison to latter stages in current study. However, as stated above, W<sub>mean</sub> in the first 500m was reduced in WU<sub>all-out</sub> compared to WU<sub>100%</sub> and WU<sub>150%</sub>, but not (significantly) reduced compared to  $WU_{con}$  (due to the large variance in all conditions for  $W_{peak}$  in the first 15s of the 3km-TT). Additionally, W<sub>mean - 1/2 way</sub> was also reduced in WU<sub>all-out</sub> compared to all other conditions, indicating that first half performance was in fact reduced somewhat following the most intense priming condition. VO<sub>2peak</sub> during the 3km-TT was not different between priming conditions, which was consistent with Hajoglou et al. (2005), and in part explain why they stated that the response pattern is similar after three to four minutes, regardless of priming exercise condition.

## 2. Physiological responses to different priming intensities

Several studies have investigated the effect of different priming strategies on physiological responses. These have involved different intensity, duration and types (continuous versus intermittent, passive versus active). The current study measured four major physiological responses to determine whether they were associated with (or responsible for) increased/decreased performance.

### **VO**<sub>2</sub> Kinetics

In a previous priming study, Hajoglou et al. (2005) stated that the observed speeding of the  $\dot{V}O_2$  response after a priming exercise in their study appeared to be an important factor in

the role of priming exercise in improving 3km-TT cycling performance. It is conceivable that if, due to the prior exercise, the aerobic system becomes more 'primed', then more O<sub>2</sub> will be utilised at the onset of exercise, placing less reliance on anaerobic energy sources, and thus delaying the onset of fatigue during the high intensity maximal effort (Bishop, 2003b; Edwards, Challis, Chapman, Claxton, & Fysh, 2001; Nummela & Rusko, 1995). Indeed, previous work has shown that prior exercise (even low intensity) can remove some of the inertia in mitochondrial ATP production at the onset of a subsequent bout of heavy exercise (Campbell-O'Sullivan et al., 2002). However, despite these physiological possibilities, and a trend for a greater  $\tau$  and MRT in  $WU_{con}$  in the present study, no significant differences in either kinetic parameter between conditions were observed suggesting that differing intensity prior exercise had minimal effect on a exercise bout performed in the very heavy-intensity domain. It is worthy of note however the qualitative inferences observed with regards to the  $\dot{V}O_2$  kinetic parameters (*Table 4-8*). These qualitative inferences included "Possibly beneficial", "Likely beneficial" and "Very likely beneficial" changes in  $\tau$ , MRT and O<sub>2</sub> deficit. However, this did not translate to beneficial changes in performance, but instead non-significant (WU100% and WU150%) and significant (WU<sub>all-out</sub>) decreases in  $W_{mean}$ , providing some evidence that  $\dot{V}O_2$  kinetics are not determinant on performance in a task of such high intensity (103.2% W<sub>peak</sub>, range 90.7 -120.2%).

It should be noted that previous work involving prior exercise and subsequent square-wave cycling transitions has demonstrated that accelerated  $\dot{V}O_2$  kinetics, as evidenced by reduced MRT or  $\tau$ , is not always obvious (Wilkerson et al., 2004) regardless of an apparently reduced slow component (Burnley, Doust, Ball et al., 2002) or significantly increased HR in the higher intensities (Jones et al., 2003; Koga et al., 1997). Therefore it would seem that the concept of accelerating the  $\dot{V}O_2$  kinetic response through modified priming strategies may not always be possible, and may be dependent on a number of factors, such as intensity, duration and timing between priming and subsequent exercise. It is interesting to note that with two of these studies however (Jones et al., 2003; Wilkerson et al., 2004) that despite the absence of an accelerated  $\dot{V}O_2$  kinetic response, performance was still enhanced. This is consistent with the current study, whereby there were no differences in MRT or  $\tau$  between WU<sub>all-out</sub> and all other conditions that had significantly different power

outputs, but there was an accelerated response in WU<sub>150%</sub> where there was *no* performance enhancement. Methodologically, the failure to identify difference in  $\tau$  (and MRT) could have been due to the number of transitions performed. In  $\dot{VO}_2$  kinetic studies, it is usually necessary to have subjects complete multiple transitions (n = 2-6) prior to kinetic exponential modelling in order to reduce breath-by-breath noise. However, due to supramaximal performance nature of this study and the four different conditions, it placed large physical demand on the athletes, was time consuming and interfered with their training. Therefore, it was not appropriate to have athletes perform repeated trials for each condition. Hence, breath-by-breath variability could have had some influence on the determined kinetic parameters.

Whilst, Hajoglou et al. (2005) suggested the speeding of  $\dot{V}O_2$  kinetics as a key variable in improving performance, it should be acknowledged that their control condition consisted of maximal exercise (3km-TT) in the absence of any priming exercise. Whilst both experimental conditions (hard and easy priming strategies) did involve exercise, there was no difference in either performance or MRT between them. It seems therefore, that this 'speeding' of the  $\dot{V}O_2$  kinetics is only a significant factor when comparing exercise that utilised priming exercise (perhaps regardless of intensity) versus one that did not (nonexercise control). When one considers that 1) 'warm-up' is standard practice for any physical exercise (especially one of such high intensity: ~110% W<sub>peak</sub>); 2) it is unlikely that an athlete would compete with no prior exercise, and 3) the absence of priming has been shown to detrimental to performance (Hajoglou et al., 2005; Jones et al., 2003; Stewart & Sleivert, 1998; Wilkerson et al., 2004), the use of a non-exercise control condition for sports performance research is surprising, and this true control condition would appear somewhat redundant. The use of the athletes self-chosen strategy (as the current best practice, and experimental control) and subsequent efforts to refine or improve the performance by modifying the priming strategy appears to be more appropriate when working with competitive athletes.

Despite neither  $\tau$  nor MRT presenting any significant findings that appeared to relate to performance differences, differences in O<sub>2</sub> deficit were somewhat more obvious. Interestingly, although it resulted in a non-significant reduction in W<sub>mean</sub> (-1.5%, *p* = 0.57;

log transformed data) and  $\dot{VO}_{2peak}$  (-0.9%, p = 0.59) compared to  $WU_{con}$ ,  $WU_{150\%}$  displayed a significant reduction in O<sub>2</sub> deficit (-19.2%, p = 0.025), with data that gave a qualitative inference of a 'very likely beneficial' true change in the mean (Table 4-8). In the current study,  $O_2$  deficit was calculated by multiplying the MRT by the amplitude of change (A';  $\dot{V}O_{2-\text{new steady-state}}$  -  $\dot{V}O_{2-\text{baseline}}$ ). Therefore, as the reduction in baseline  $\dot{V}O_2$  for  $WU_{150\%}$ from WU<sub>con</sub> in the two minutes prior to the 3km-TT was not significant, 'noise' in the baseline  $\dot{V}O_2$  values multiplied by 'noise' in the peak steady-state  $\dot{V}O_2$  values may have resulted in an A' that was of great enough magnitude to significantly influence the resulting O<sub>2</sub> deficit value. This is an especially plausible explanation when one considers that the reduction in MRT for WU150% from WUcon approached significance and displayed a favourable practical inference (-13.7%, p = 0.062, 'very likely beneficial'). This significant change in amplitude, without an accompanying decrease in MRT, is supported by the findings of Wilkerson et al. (2004). Wilkerson et al. (2004) described an increased asymptotic "gain" of the primary  $\dot{V}O_2$  response following a more intense priming condition in the absence of any change in the primary  $\tau$ . This indicates that it is possible for a new peak steady-state  $\dot{V}O_2$  to be attained, while the *rate* of change remains constant.

### *VO<sub>2peak</sub> attained*

If priming exercise stimulates the aerobic system and recruits/activates all muscle fibres, then it may be conceivable to expect a higher end-exercise  $\dot{V}O_2$  at the end of a trial than that measured at the end of a prolonged incremental test to exhaustion. Indeed, a higher  $\dot{V}O_{2peak}$  following a priming intervention has been documented previously in 'square-wave' protocols of submaximal intensities (Burnley, Doust, & Jones, 2002; Koppo & Bouckaert, 2000; Koppo et al., 2003; Wilkerson et al., 2004). In the present study however (103.2%  $W_{peak}$ ), no condition displayed  $\dot{V}O_{2peak}$  values that were significantly different from the initial incremental assessment (*Table 3-1*), suggesting that none of the priming strategies stimulated additional O<sub>2</sub> utilisation, at least to the extent detectable with current methods of measurement (breath-by-breath analysis). This finding is in agreement with a number of previous studies (Bangsbo et al., 2001; Billat, Bocquet et al., 2000; Bishop et al., 2001; Bishop et al., 2003; Judelson et al., 2004; Koppo et al., 2002).

### *Core temperature* $(T_C)$

It has been proposed that the positive effects of a 'warm-up' are due primarily to the increase in T<sub>C</sub> (Bishop, 2003a, 2003b; Mohr et al., 2004). However, it should be noted that these papers did not directly demonstrate the effects of increased  $T_C$  or  $T_M$  on performance per se, but rather that some increase in temperature accompanied active 'warm-ups'. To investigate the effects of either  $T_C \mbox{ or } T_M$  independently on the  $\dot{V}O_2$  kinetic response, some studies have utilised passive warming (hot water perfused pants, warm/hot water baths, heat packs in combination with heat creams and rubs) and reported no speeding of the  $VO_2$ kinetic response (Burnley, Doust, & Jones, 2002; Koga et al., 1997; Koppo et al., 2002), and reduced [BLa] compared to active 'warm-up' (Gray et al., 2002). In the current study, there were no significant differences in  $T_{\rm C}$  amongst conditions either post-priming, pre- or post 3km-TT for WU<sub>con</sub>, WU<sub>100%</sub> and WU<sub>150%</sub>. However, for the highest intensity (WU<sub>all</sub>-<sub>out</sub>) T<sub>C</sub> was elevated (1.2%, p = 0.03; log transformed data) compared to WU<sub>con</sub> postpriming, and compared to  $WU_{100\%}$  pre- (1.2%, p = 0.05) and post-3km-TT (1.0%, p = 0.02). Both WU<sub>con</sub> and WU<sub>100%</sub> could be considered the 'easier' intensities. These results, which are consistent with those observed for [BLa], support the belief that within the range observed in this study, T<sub>C</sub> will not affect 3km-TT performance. However, when the increase in T<sub>C</sub> is too great, as observed for WU<sub>all-out</sub>, its interaction with other physiological factors (e.g. VO<sub>2</sub> (through an increased Q<sub>10</sub> effect), HR (change in cardiac output/stroke volume)) is likely to result in impaired performance. This is supported by the findings of a previous study on priming and anaerobic running performance (Stewart & Sleivert, 1998) which reported that priming at 60% and 70%  $\dot{V}O_{2peak}$  resulted in increases in T<sub>C</sub> and T<sub>lim</sub> that were not significantly different from each other and both greater than the no priming However, priming at 80%  $\dot{V}O_{2peak}$  resulted in an increase in T<sub>C</sub> that was control. significantly greater again, and that this was accompanied by a significant reduction in T<sub>lim</sub>. To summarise, the results from Stewart et al. (1998) and the current study highlight potential positive effects of some increase in  $T_{\rm C}$ . However, if it is increased by too much, it is likely to be accompanied by reduced performance in high intensity exercise, as observed in the present study, whilst acknowledging the above limitations. It must also be taken into consideration in the current study that a TT of this duration (245.5 - 290 s) is unlikely to produce  $T_C$  values that are of a level dangerous to the athlete (i.e. ~40°). However, it may have elevated enough to cause some shift in the physiological state of the participants, resulting in diminished performance ( $WU_{all-out}$ ).

### Heart Rate (HR)

The HR response during each priming strategy (Figure 4-7) is somewhat interesting when the mean values associated with each condition are considered. Of the three intermittent priming conditions, WU<sub>all-out</sub> strangely displayed the lowest mean HR for the priming phase, a value that, in contrast to  $WU_{100\%}$  and  $WU_{150\%}$ , was not significantly elevated compared to WU<sub>con</sub>. Additionally, HR<sub>peak</sub> for the priming phase was less than WU<sub>150%</sub> (non-significantly however). This is potentially due to electrical interference from the ergometer disrupting the signal to the Polar watch, or simply due to individual variations in HR between visits. However, both WU150% and WUall-out were significantly elevated compared to WU<sub>100%</sub>, but surprisingly not compared to WU<sub>con</sub> prior to the 3km-TT. As with  $\dot{V}O_2$ , no difference was observed between conditions for peak and mean values during the 3km-TT. As  $WU_{con}$  and  $WU_{150\%}$  were elevated compared to  $WU_{100\%}$  and  $WU_{con}$ , but not different from each other, in conjunction with the resulting power outputs it would seem reasonable to conclude that HR prior to 3km-TT within a range similar to that observed in the current study will not be a determining factor in performance, whereas the HR immediately following the priming exercise may have some bearing on performance. This is supported by Koppo et al. (2003) who reported an increased HR during the second 'square-wave' bout and by Stewart et al. (1998) who also reported that too great an intensity priming exercise results in too great a HR response and increase in body temperature for succeeding high intensity anaerobic running, whereas no prior exercise does not physiologically prepare the athlete sufficiently for the high demand exercise. It was however in contrast to other studies (Bishop et al., 2001; Burnley, Doust, Ball et al., 2002; Koppo et al., 2003) which reported that HR<sub>peak</sub> can in fact be altered by different priming strategies, and with Bearden et al. (2001) who stated that HR and VO<sub>2</sub> do not always have the same response to priming exercise, suggesting O<sub>2</sub> delivery and O<sub>2</sub> utilisation differ. However, the HR analysis performed by Bearden et al. (2001) was much more in-depth than that of the current study, including the application of kinetic models for the examination of  $\tau$ , MRT and  $\delta$  of the HR response. Additionally, in contrast to the current study, it has also be previously reported that an increase in HR<sub>peak</sub> following heavy

priming exercise accompanies the significantly increased  $T_{lim}$  at 110% and 120%  $VO_{2peak}$  (Jones et al., 2003). However, as with many other priming studies and in contrast to the current study, the study of Jones et al. (2003) compared heavy priming with a control condition where no priming exercise at all was performed.

#### Central Governor Model

The 'Central Governor model (CGM) of fatigue provides another possibility in the explanation of the results displayed in this study. This particular model has been proposed by a number of authors, along with different mechanisms put forward by each author to explain responses to exercise using this model.

Briefly, the CGM proposes that, subconsciously, the athlete centrally controls physical efforts based on physiological responses to exercise, and its associated feedback based on a whole host of peripheral information (Weir, Beck, Cramer, & Housh, 2006). Discomfort caused by an increase in acidosis (as represented in the current investigation by the elevated [BLa] in WU<sub>all-out</sub>) contributes to the conscious decision to terminate exercise, or unconscious decision to decrease maximal power output (Noakes, St Clair Gibson, & Lambert, 2004) or alter the pacing strategy implemented (Weir et al., 2006). WU<sub>all-out</sub> displayed not only significantly decreased W<sub>peak</sub> compared to all other conditions, but also the subjects needed to reduce their overall power output in order to complete the 3k-TT (i.e. decreased  $W_{mean}$ ). It is reported that homeostasis in the organ systems is maintained at exhaustion, providing evidence of CNS regulation via the number of motor units recruited during exercise, thereby setting the total metabolic demand (Noakes et al., 2004). Similarly, evidence of homeostasis being maintained via mechanisms of the CGM has been shown by an absence of any significant difference in heat storage between exercise conditions, despite differences in power output and subjective RPE (Tucker, Marle, Lambert, & Noakes, 2007).

It is also suggested that some disruption of homeostasis is required to complete the task, and/or stimulate adaptations to training (Weir et al., 2006). Thus, homeostasis is not maintained, but rather "terminal metabolic crisis" is prevented (St Clair Gibson & Noakes, 2004; Weir et al., 2006). Agreeably, evidence suggests that while the rise in core temperature during exercise in the heat does not limit performance *per se*, the core

temperature increases at an accelerated rate which would allow the organism to anticipate the point of termination and avoid that avoid metabolic catastrophe (Marino, 2004). This again would be in agreement with Tucker et al. (2007), as well as helping to explain the decreased performance coupled with the increased  $T_C$  for the  $WU_{all-out}$  condition in the current study. Subjects during  $WU_{all-out}$  exhibited significantly increased  $T_C$  compared to  $WU_{con}$  and  $WU_{100\%}$  at different stages of the priming and trial, and had it not been for such large variability, it is likely this increased  $T_C$  would have been significant with all over conditions at all measured points following the priming and 3km-TT. When considering the CGM, it is plausible that following the strenuous priming of  $WU_{all-out}$ , the increased  $T_C$ stimulated the CNS to decrease power output, thereby avoiding a change in homeostasis of the magnitude to cause terminal metabolic crisis (Marino, 2004; St Clair Gibson & Noakes, 2004; Weir et al., 2006).

Feedback control results in continuous adjustments in pace, power output and metabolic activity over the entire task, influencing the overall pacing strategy, and may also be influenced by knowledge of an endpoint (Albertus, Tucker, St Clair Gibson et al., 2005; St Clair Gibson, Lambert, Rauch et al., 2006). Again, this is perhaps best displayed in the current study by the most intense priming condition, WU<sub>all-out</sub>. The accelerated metabolic response of this condition, as evidenced post-priming, would have provided feedback to the CNS to decrease W<sub>mean</sub> in order to avoid catastrophic failure, whilst also allowing the subjects to complete the required task (with its known endpoint of three kilometres). As the subjects were likely to have worked too hard in the WU<sub>all-out</sub> condition, they would potentially have been subconsciously limiting themselves before the task began, in addition to the constant feedback throughout. When considering the CGM as a theory to explain pacing strategies and effort, it therefore seems acceptable for the WU<sub>con</sub> condition to perform best, given that, in a distance the subjects have performed many times before (including two familiarisation trials), they would best be able to subconsciously regulate their physiological responses in a pre-event routine of which they are most familiar in comparison to one of both a highly intense and foreign nature.

### 3. Limitations

Participants in the present study were advised to 'warm-up as you normally would before an event of similar distance' for the self-chosen (WU<sub>con</sub>) trial. However, whilst the duration of the priming phase of WU<sub>con</sub> (self-chosen) was similar to each of the prescribed priming conditions ( $15:33 \pm 4:43$  vs. 15:00 min) some athletes reported afterward that their priming was slightly shorter than they would typically perform on race day in the field (at the track). This could have been due to the participants not wanting to spend extended periods of time in the lab, and also because many track cyclists employ 'primary' and 'secondary' warmups, whereby they may perform priming exercise up to an hour before the event, and then a shorter second priming exercise much closer to their event. Clearly, replicating actual raceday conditions and preparation strategies in the laboratory is a challenge for researchers.

A number of factors may have affected the  $\dot{VO}_2$  kinetic responses observed in the current study. Firstly, it is possible that, as mentioned above, the 'all-out' start pacing strategy in the current study may have helped reduce variation in  $W_{mean}$ , but it may have also affected the  $\dot{VO}_2$  kinetic response (Hettinga et al., 2006). This can be supported by the significant findings with regards to  $\dot{VO}_2$  kinetics reported by Hajoglou et al. (2005), who did not enforce a pacing strategy, but whose participants appeared to adopt a fairly even pacing strategy themselves. Although, it must also be noted that Hajoglou et al. (2005) only reported significant differences between the 'no-warm-up' control and active priming strategies. It is also possible that the gas analysis employed in the current study may have had some error of measurement associated with it that made such sensitive measures (such as  $\tau$  at ~100% W<sub>peak</sub>) difficult to detect accurately, especially given that the single trial performed per participant per condition is likely to have introduced additional noise. While this apparatus has proven reliability and validity (Larsson et al., 2004), its potential error in measuring sensitive mechanisms such as this cannot be ignored and may have been improved with repeated trials had this been possible.

It must be acknowledged the technical limitations involved with measuring  $T_c$ , be it the possibility of convective cooling with tympanic in the current study (Hansen et al., 1993), location of the pill in the intestinal tract when using intestinal pill system (Edwards & Clark, 2006; Kolka, Quigley, Blanchard, Toyota, & Stephenson, 1993), as well as the

different sites of measurement (i.e.  $T_C$  with tympanic or rectal,  $T_C$  versus  $T_M$ ) presenting different findings. However, it has been proven previously that tympanic is indeed a valid and reliable measure of  $T_C$  when strict procedures are followed, such as minimising airflow. This was achieved in the current study through the cotton wool placed in the ear, the exclusion of any fans and a constant laboratory temperature. Additionally, it has been previously shown that  $T_M$  does not directly influence the  $\dot{V}O_2$  kinetics *per se*, thus providing further justification for measuring  $T_C$  instead. Additionally, while the change in  $T_C$  amongst conditions were not different, the potential for a change in  $T_M$  (which was not measured) cannot be ignored, nor can its potential effect on performance.

### 4. Practical Application

In terms of athletes integrating the current (and previous) findings into their precompetitive routine, a number of recommendations may be given with regards to priming exercise which will provide optimal preparation for 3km-TT cycling performance. Firstly, although the range of  $T_C$  changes observed in the current study was negligible, athletes must be aware to not increase  $T_C$  (nor  $T_M$  and [BLa]) by too great a magnitude. Too great an intensity of priming exercise is likely to result in substantial increases in both  $T_C$  and acidosis that are likely to inhibit performance, potentially through an increased  $O_2$  cost of exercise and reduced muscle contractile function (Bishop, 2003a; Bishop et al., 2001).  $T_C$ did not present results that were significant between conditions or determinant of performance. Therefore, is  $T_C$  really so important during priming? Are we actually 'warming up' the body? Both current and previous data suggests not; rather, other physiological (and psychological) 'priming' factors may be more important. The  $\dot{V}O_2$ kinetics did not appear to be an important determinant of performance, nor are they likely to change in such high intensity exercise; be it self-paced or all-out paced.

The current study would suggest that athletes are in fact able to self-select their most effective priming strategy. However, considering the non-significant difference in performance that  $WU_{con}$  had compared with  $WU_{100\%}$  and  $WU_{150\%}$ , it is possible that other athletes may benefit from a prescribed priming strategy, of which the intermittent high-intensity nature of  $WU_{100\%}$  and  $WU_{150\%}$  is suitable for 3km-TT cycling performance, and is in agreement with previous work (Mandengue, Seck, Bishop et al., 2005). It should be

acknowledged that the 'optimal' intensity and duration of priming exercise also is likely to be individual, with some responding positively to higher intensity priming, while others a lesser intensity. Such differences may depend on the physical and mental attributes the athlete possesses, and perhaps what style of rider they are. For example, an athlete who has a larger build and is able to generate high forces, and has a more aggressive approach to riding, may favour the all-out start. This type of athlete may benefit from a preparation that has greater emphasis on priming the anaerobic energy pathways. In contrast, a rider of slighter build who adopts an even paced racing strategy may benefit more from placing greater emphasis on priming the aerobic energy pathways. Indeed, it has been previously documented that two cyclists who differed by only 0.2s in a 4000m trial (4min:18.8s versus 4min:19.0s) had quite different physiology; one relied heavily on his well developed aerobic capacity power at lactate threshold, while the other was capable of considerable anaerobic metabolism (Schumacher & Mueller, 2002).

It is also important to consider the psychological benefits of 'warming-up' and the mental routine that athletes often go through in preparation for a race. Athletes competing in track cycling quite often adopt longer during priming exercise than that which was prescribed in the current study, and may not have the same 'mental alertness' or feelings of preparedness that they would get from their own self-chosen 'warm-up'. Additionally, because athletes have been led to believe that they need long duration priming exercise, they may not believe/accept that one of short duration and high intensity will adequately prepare them; thus inhibiting performance from a mental perspective. It is expected that, even had the shorter duration priming strategy been by far the most effective, it would still be likely difficult to convince athletes to abandon their long duration priming exercise, being what they have done for so long. Clearly, an educational approach alongside practicing different priming strategies, and their subsequent effect on performance in the field, is required.

Further research to determine the effect that the prescribed priming strategies in this study have on shorter distances, such as the 'kilo' (1km-TT) and 'sprint' events, both in the laboratory and on the track would be worthwhile exploring since the apparent 'fatiguing-effect' of  $WU_{all-out}$  may not be present in an event of shorter distance. Indeed, very high intensity priming for short events (~60s) may be desirable to optimise performance, as observed by Bishop et al. (2003) for 2min kayak performance. However, this is currently

unknown for cycling and other sports such as running and swimming. Additionally, future research may investigate the effects of priming and different pacing strategies (i.e. shorter duration 'all-out' sprint at the start of the 3km-TT) would have on the 3km-TT, especially since variation in  $W_{mean}$  for the early stages of the 3km-TT in the current study were observed.

### 5. Conclusion

In conclusion, the results from this study showed that 3km-TT cycling performance was significantly inhibited by a very intense priming strategy. It appears that athletes are able to self-select their most effective priming strategy, although it did not significantly improve performance compared to the lowest and moderate intensity intermittent priming strategies. The physiological responses to the WU<sub>all-out</sub> priming strategy that appeared to be detrimental to performance were the markedly increased [BLa],  $\dot{VO}_2$  and (potentially) HR (unclear due to disrupted recordings), as recorded immediately following the priming exercise. The priming strategies investigated had no effect on phase II  $\dot{VO}_2$  kinetic parameters or  $T_C$ . In an attempt to improve athletic performances, further work utilising the priming strategies from the current study with events of shorter duration is required to further determine if high-intensity priming strategies positively affect performance in other track cycling events.

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## Appendices

Appendix 1-1: Values for [BLa] and T<sub>C</sub> during the priming exercise and 3km-TT in relation to priming exercise condition (Mean  $\pm$  SD)

	WU <sub>con</sub>	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
Time-point				
		Blood Lactate Conc	entration (mmol·L <sup>-1</sup> )	
Rest	$0.96 \pm 0.30$	$0.93\pm0.32$	$1.02 \pm 0.41$	$0.98\pm0.37$
Post-priming	$3.87 \pm 1.47$	$3.19 \pm 1.51$	$6.17 \pm 2.36$ <sup>c</sup>	$8.31 \pm 1.93$ <sup>d</sup>
Pre-3km-TT	$3.09 \pm 1.63$	$2.14 \pm 1.10$	$5.27 \pm 1.72$ <sup>c</sup>	$7.35 \pm 1.92$ <sup>d</sup>
Post-3km-TT	$10.18\pm2.60$	$9.68\pm3.42$	$10.64 \pm 4.14$	$10.55\pm3.18$
		Core Temp	erature (°C)	
Rest	$36.03 \pm 0.46$	36.13 ± 0.79	$36.30 \pm 0.49$ <sup>a</sup>	$36.17\pm0.34$
Post-priming	$37.30 \pm 0.59$	$37.40\pm0.50$	$37.44 \pm 0.52$	$37.75 \pm 0.40^{a}$
Pre-3km-TT	$36.93 \pm 0.64$	$36.95\pm0.65$	$37.05 \pm 0.51$	$37.21 \pm 0.38$ <sup>b</sup>
Post-3km-TT	$37.41 \pm 0.75$	$37.39 \pm 0.59$	$37.67 \pm 0.67$	$37.78 \pm 0.39$ <sup>b</sup>
Significantly different fi	rom WU <sub>con</sub> $(p < 0.05)$			
Significantly different fi	rom WU <sub>100%</sub> ( $p < 0.05$	5)		
Significantly different fi	rom both WU <sub>con</sub> and V	$WU_{100\%}$ (p < 0.05)		
Significantly different f				
Significantly different i		(p < 0.05)		

	WU <sub>con</sub>	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
Time-point				
		Oxygen Upt	ake (L•min <sup>-1</sup> )	
Rest	$0.56\pm0.08$	$0.60\pm0.09$	$0.58\pm0.12$	$0.64\pm0.14$
Post 10min SS	-	$3.87 \pm 0.39$	$3.79\pm0.45$	$3.83\pm0.42$
Mean for 10min SS	-	$3.65\pm0.33$	$3.61\pm0.34$	$3.63\pm0.35$
Post-priming	$1.93\pm0.43$	$2.68\pm0.32$ $^a$	$2.97\pm0.41~^{\rm c}$	$3.01 \pm 0.39^{a}$
Mean during priming	$3.31\pm0.35$	$3.74\pm0.34~^a$	$3.83 \pm 0.34$ <sup>a</sup>	$3.84\pm0.34~^a$
Peak during priming	$4.18\pm0.29$	$4.07\pm0.39$	$4.35\pm0.41~^{b}$	$4.38\pm0.35~^{b}$
Pre-3km-TT	$0.96\pm0.75$	$0.76\pm0.11$	$0.86\pm0.15~^{b}$	$0.94 \pm 0.12$ <sup>d</sup>
Post-3km-TT	$3.94\pm0.40$	$3.53\pm0.57$ $^{a}$	$3.52\pm0.75$	$3.67\pm0.41$
Mean during 3km-TT	$4.51\pm0.46$	$4.38\pm0.45$	$4.50\pm0.55$	$4.44\pm0.47$
Peak during 3km-TT	$4.86\pm0.51$	$4.84\pm0.50$	$4.82\pm0.54$	$4.76\pm0.50$
		Heart Rat	e ( <b>b·min</b> <sup>-1</sup> )	
Rest	$74 \pm 26$	$65 \pm 10$	$66 \pm 11$	$69 \pm 12$
Post 10min SS	-	$160 \pm 7$	$162 \pm 10$	$161 \pm 5$
Mean for 10min SS	-	$150 \pm 11$	$151 \pm 11$	$143 \pm 14$
Post-priming	$135 \pm 21$	$156\pm12$ <sup>a</sup>	$160 \pm 11$ <sup>a</sup>	$162 \pm 10^{a}$
Mean during priming	$144 \pm 14$	$154 \pm 11$ <sup>a</sup>	$157\pm11$ <sup>a</sup>	$151 \pm 13$
Peak during priming	$163 \pm 27$	$165 \pm 9$	$176\pm11$ <sup>b</sup>	$173\pm8$ <sup>b</sup>
Pre-3km-TT	$112 \pm 24$	$102 \pm 8$	$111 \pm 14^{b}$	$116\pm16$ <sup>b</sup>
Post-3km-TT	$180 \pm 11$	$176 \pm 21$	$182 \pm 10$	$180 \pm 10$
Mean during 3km-TT	$173\pm8$	$174 \pm 12$	$176 \pm 11$	$173 \pm 7$
Peak during 3km-TT	$185\pm9$	$187 \pm 12$	$187 \pm 12$	$184 \pm 9$

Appendix 1-2: Values for VO <sub>2</sub> and HR during the priming exercise and 3km-TT in relation to priming
exercise condition (Mean $\pm$ SD)

<sup>a</sup> Significantly different from WU<sub>con</sub> (p < 0.05) <sup>b</sup> Significantly different from WU<sub>100%</sub> (p < 0.05) <sup>c</sup> Significantly different from both WU<sub>con</sub> and WU<sub>100%</sub> (p < 0.05) <sup>d</sup> Significantly different from both WU<sub>100%</sub> and WU<sub>150%</sub> (p < 0.05)

Condition	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
		D4	
Change in mean (%)	8.2	Rest 3.2	12.4
SD of change (%)	14.0	20.0	12.4
90% confidence limits (%)	-0.2 - 17.3	-8.9 - 16.8	0.4 - 25.8
<i>p</i> value	0.1090	0.6510	0.0896
Practical inference <sup>‡</sup>	Likely positive	Possibly positive	Likely positive
		Post-priming	
Change in mean (%)	41.2	56.2	58.1
SD of change (%)	20.7	26.9	28.0
90% confidence limits (%)	25.2 - 59.2	33.6 - 82.6	34.4 - 86.0
<i>p</i> value	0.0005	0.0005	0.0006
Practical inference <sup>‡</sup>	Almost certainly positive	Almost certainly positive	Almost certainly positiv
		Mean during priming	
Change in mean $(0/)$	13.2	15.9	16.3
Change in mean (%)			
SD of change (%)	13.4	11.2	11.4
90% confidence limits (%)	4.8 - 22.3	8.6 - 23.7	8.8 - 24.2
<i>p</i> value	0.0166	0.0024	0.0024
Practical inference <sup>‡</sup>	Very likely positive	Almost certainly positive	Almost certainly positiv
		Peak during priming	
Change in mean (%)	-3.0	3.8	4.5
SD of change (%)	8.3	9.4	6.3
90% confidence limits (%)	7.6 - 1.8	-1.7 - 9.6	0.7 - 8.4
<i>p</i> value	0.2748	0.2436	0.0557
Practical inference <sup>‡</sup>	Likely negative	Likely positive	Likely positive
		Pre-3km-TT	
Change in mean (%)	-9.0	3.1	13.4
SD of change (%)	49.5	50.9	46.0
90% confidence limits (%)	-31.7 - 21.2	23.2 - 38.4	-13.1 - 48.1
<i>p</i> value	0.5601	0.8544	0.4083
1			
<b>Practical inference<sup>‡</sup></b>	Unclear	Unclear	Likely positive
		Post-3km-TT	
Change in mean (%)	-11.0	-12.4	-6.8
SD of change (%)	13.2	22.7	10.2
90% confidence limits (%)	-17.53.9	-23.20.1	-12.21.2
<i>p</i> value	0.0207	0.0975	0.0555
Practical inference <sup>‡</sup>	Very likely negative	Likely negative	Very likely negative
		Mean during 3km-TT	
Change in mean (%)	-2.8	-0.5	-1.5
SD of change (%)	7.6	6.5	5.5
90% confidence limits (%)	7.0 - 1.6	4.2 - 3.3	4.6 - 1.7
<i>p</i> value	0.2690	0.8127	0.4026
Practical inference <sup>‡</sup>	Likely negative	Unclear	Possibly negative
Change in mean (%)	-0.4	<b>Peak during 3km-TT</b> -0.9	-2.0
SD of change (%)	9.3	4.9	5.2
90% confidence limits (%)	5.6 - 5.1	-3.7 - 2.0	-4.9 - 1.0
	0.9030	0.5851	4.9 - 1.0 0.2499
<i>p</i> value Practical inference <sup>‡</sup>			Possibly negative
r racucal interence"	Unclear	Unclear	Possibly negative

# *Appendix 1-3:* Log transformed mean changes in VO<sub>2</sub> from control (WU<sub>con</sub>) between conditions and chances that the true difference in the changes is substantial

<sup>‡</sup>Based on a smallest worthwhile beneficial or harmful change in performance of 1%

Condition	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
		Rest	
Change in mean (%)	-1.8	-0.1	4.2
SD of change (%)	12.1	9.1	11.4
90% confidence limits (%)	-8.4 - 5.4	-5.6 - 5.7	-2.5 - 11.3
<i>p</i> value	0.6529	0.9739	0.2836
Practical inference <sup>‡</sup>	Unclear	Unclear	Likely positive
		Post-priming	
Change in mean (%)	16.2	20.3	22.0
SD of change (%)	14.6	14.7	16.5
90% confidence limits (%)	6.8 - 26.5	9.9 - 31.8	10.1 - 35.1
<i>p</i> value	0.0101	0.0054	0.0069
Practical inference <sup>‡</sup>	Almost certainly positive	Almost certainly positive	Almost certainly positiv
	10.5	Mean during priming	( <b>0</b> )
Change in mean (%)	10.5	11.5	6.2
SD of change (%)	10.0	7.5	13.5
90% confidence limits (%)	2.7 - 18.9	6.0 - 17.3	2.3 - 15.5
<i>p</i> value	0.0387	0.0046	0.2145
Practical inference <sup>‡</sup>	Very likely positive	Almost certainly positive	Likely positive
		Peak during priming	
Change in mean (%)	-1.3	3.5	2.5
SD of change (%)	7.4	6.1	6.5
90% confidence limits (%)	6.1 - 3.8	0.7 - 7.8	1.3 - 6.5
<i>p</i> value	0.6417	0.1554	0.2556
<i>p</i> value Practical inference <sup>‡</sup>	Unclear		
Practical interence	Unclear	Likely positive	Likely positive
		Pre-3km-TT	
Change in mean (%)	1.1	5.8	10.6
SD of change (%)	11.8	12.2	15.5
90% confidence limits (%)	7.3 - 10.2	-1.9 - 14.1	0.4 - 21.7
<i>p</i> value	0.8173	0.2058	0.0875
Practical inference <sup>‡</sup>	Possibly positive	Likely positive	Likely positive
I factical interence	r ossibily positive	Likely positive	Likely positive
		Post-3km-TT	
Change in mean (%)	-2.9	-0.2	-1.5
SD of change (%)	10.2	1.4	3.0
90% confidence limits (%)	-9.3 - 4.0	-1.1 - 0.6	-3.3 - 0.3
<i>p</i> value	0.4476	0.6270	0.1562
Practical inference <sup>‡</sup>	Possibly negative	Likely trivial	Possibly negative
Change in mean (0/)	0.1	Mean during 3km-TT 1.3	-0.2
Change in mean (%)		1.5	0.2 3.9
SD of change (%)	3.4		
90% confidence limits (%)	2.1 - 2.4	0.1 - 2.4	2.6 - 2.3
<i>p</i> value	0.9134	0.0758	0.9034
Practical inference <sup>‡</sup>	Unclear	Possibly positive	Unclear
		Peak during 3km-TT	
Change in mean (%)	0.7	-0.1	-1.0
SD of change (%)	1.5	2.2	2.6
90% confidence limits (%)	-0.3 - 1.6	-1.4 - 1.3	-2.4 - 0.5
	0.3 - 1.6 0.2227	0.9125	2.4 - 0.5 0.2578
<i>p</i> value			
Practical inference <sup>‡</sup>	Likely trivial	Likely trivial	Unclear

# *Appendix 1-4:* Log transformed mean changes in HR from control (WU<sub>con</sub>) between conditions and chances that the true difference in the changes is substantial

<sup>‡</sup>Based on a smallest worthwhile beneficial or harmful change in performance of 1%

Condition	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
		_	
		Rest	
Change in mean (%)	4.6	4.0	0.4
SD of change (%)	38.2	36.5	41.9
90% confidence limits (%)	23.5 - 19.1	15.9 - 28.5	21.2 - 28.0
<i>p</i> value	0.7071	0.7445	0.9754
Practical inference <sup>‡</sup>	Unclear	Unclear	Unclear
		Post-priming	
Change in mean (%)	-22.9	60.2	125.5
SD of change (%)	72.5	54.2	50.4
90% confidence limits (%)	49.3 - 17.4	17.0 - 119.3	68.4 - 202.1
<i>p</i> value	0.2863	0.0225	0.0006
Practical inference <sup>‡</sup>	Likely positive	Very likely positive	Almost certainly positiv
		Pre-3km-TT	
Change in mean (%)	21.7	85.8	158.1
SD of change (%)	63.1	71.7	60.5
90% confidence limits (%)	47.0 - 15.8	19.1 - 189.7	77.4 - 275.4
<i>p</i> value	0.2792	0.0320	0.0015
Practical inference <sup>‡</sup>	Likely negative	Very likely positive	Almost certainly positive
		Post-3km-TT	
Change in mean (%)	-6.1	-3.0	7.3
SD of change (%)	21.5	56.8	22.5
90% confidence limits (%)	17.8 - 7.3	31.8 - 38.0	6.6 - 23.4
<i>p</i> value	0.4067	0.8781	0.3731
Practical inference <sup>‡</sup>	Likely negative	Unclear	Likely positive

*Appendix 1-5:* Log transformed mean changes in [BLa] from control (WU<sub>con</sub>) between conditions and chances that the true difference in the changes is substantial

\* Based on a smallest worthwhile beneficial or harmful change in performance of 1%

Condition	WU <sub>100%</sub>	WU <sub>150%</sub>	WU <sub>all-out</sub>
		Rest	
Change in mean (%)	0.3	0.7	0.4
SD of change (%)	1.8	1.0	1.0
90% confidence limits (%)	0.8 - 1.3	0.2 - 1.3	-0.2 - 1.0
<i>p</i> value	0.6566	0.0423	0.2346
Practical inference <sup>‡</sup>	Likely trivial	Likely trivial	Very likely trivial
		Post-priming	
Change in mean (%)	0.3	0.4	1.2
SD of change (%)	2.2	2.3	1.5
90% confidence limits (%)	-1.0 - 1.5	1.0 - 1.7	0.3 - 2.1
<i>p</i> value	0.6996	0.6187	0.0347
Practical inference <sup>‡</sup>	Likely trivial	Possibly trivial	Possibly negative
		Pre-3km-TT	
Change in mean (%)	0.1	0.3	0.9
SD of change (%)	2.1	2.4	1.3
90% confidence limits (%)	1.2 - 1.3	1.1 - 1.8	0.1 - 1.8
<i>p</i> value	0.9392	0.6809	0.0642
Practical inference <sup>‡</sup>	Likely trivial	Possibly trivial	Possibly trivial
		Post-3km-TT	
Change in mean (%)	0.0	0.7	1.0
SD of change (%)	1.9	2.5	1.8
90% confidence limits (%)	1.1 - 1.0	-0.7 - 2.2	0.0 - 2.0
<i>p</i> value	0.9388	0.3993	0.1083
Practical inference <sup>‡</sup>	Likely trivial	Possibly trivial	Possibly trivial

<i>Appendix 1-6:</i> Log transformed mean changes in $T_C$ from control (WU <sub>con</sub> ) between conditions and chances
that the true difference in the changes is substantial

Appendix 2-1: Participant information sheet

**Participant Information Sheet** 



#### **Date Information Sheet Produced:**

27<sup>th</sup> March 2006

#### **Project Title**

The effects of different intermittent 'warm-up' strategies on 3km cycling performance.

#### Invitation

As an 18-40 year old male cyclist, you are invited to participate in a study investigating the effects of different warm-up strategies on 3km cycling performance. This study is being undertaken as part of a Masters of Health Science qualification. Participation is completely voluntary and you may withdraw at any stage without giving a reason or being disadvantaged.

#### What is the purpose of this research?

The aim of this thesis is to explore the effects of different warm-up strategies with the intention of determining the optimum warm-up strategies for maximal cycling events for improved athletic performance. Warming-up is an accepted practice in the preparation for any sporting or exercise participation or competition. However, many coaches, conditioners and athletes themselves often have no scientific basis on the intensity chosen to perform the warm-up, commonly just performing that which they have always done, or those before them have done. It is now being recognised by coaches and conditioners that the warm-up required before performances involving high intensity exercise may be more important than it was once considered. This in turn has generated scientific interest on how to effectively utilise the time prior to a performance.

#### How are people chosen to be asked to be part of this research?

Athletes for this study will be recruited by advertising (posters, emails in club newsletter) the study at cycling clubs and training venues throughout Auckland. In addition, participants from previous similar studies will be invited to partake. The investigator will also visit cycling clubs and speak to athletes and coaches to recruit participants.

#### What happens in this research?

Participation in this study involves repeated (6) visits to the laboratory to determine the optimal warm up intensity. It is estimated that each testing session will take approximately 40-60 minutes from the time that you arrive at the lab to when the tests are finished. It will take six sessions per subject (one preliminary aerobic assessment trial, one familiarisation trial and four performance trials).

Mores specifically, you will be required to compete the following:

1) In a preliminary assessment, you will perform a standard continuous incremental exercise test to voluntary failure to determine your aerobic capacity (VO<sub>2peak</sub>). The test will involve a ten minute warm-up (self-selected), followed by an exercise test to exhaustion which will start easy and get progressively harder. During visit one, you will also be familiarised with the time-trial assessment being performed in the subsequent visits.

2) You will perform 4 different warm up routines (your own self-chosen warm-up, high intensity intermittent warm-ups performed at 100% and 150% of the power you achieved during visit one, as well as "all-out" intervals, during which you will perform sprint intervals with maximum effort), on separate days, followed by a three kilometre performance trial. During each of the warm-ups and performance trials, heart rate (HR), oxygen uptake (VO<sub>2</sub>), body core temperature

(tympanic thermometer in the ear) and blood lactate responses will be measured to see how they influence performance.

#### What are the discomforts and risks?

You may experience muscular discomfort during and/or after testing and training. This is of similar magnitude to a hard training session which you regularly expose yourself to.

#### How will these discomforts and risks be alleviated?

These effects of muscular discomfort are unlikely to be greater than you would experience following a normal training session, assessment or competition and you will therefore be familiar with it. All steps will be taken to ensure these effects are minimised by appropriate warm-ups and rest periods being prescribed during and between testing and training sessions.

Maximal testing will be supervised by accredited sport scientists and carried out by the student who is familiar with lab-based assessment of cyclists. The supervisor and the student both hold current Fist Aid certificates.

#### What are the benefits?

Participants in this study will receive free physiological assessments of their aerobic capacity, which are essential in the optimal development of an athlete's athletic progress. In addition to this, subjects will also be given direct feedback as to the optimal warm-up strategy for them individually to improve race performance, and coaches will be given feedback as to how to design the most effective warm-up for cyclists competing in events of similar nature.

#### What compensation is available for injury or negligence?

Compensation is available through the Accident Compensation Corporation within its normal limitations.

#### How will my privacy be protected?

No individual participant will be identifiable in the final report as all data will be grouped, with only means displayed. All individual data will be stored on a password protected computer and hard copies in a locked room. Only the researcher and advisors will have access to and view this data. Coaches and physiologists that work with you may be given your assessment data with the your permission

#### What are the costs of participating in this research?

The only cost of you is your time, as outlined above (six sessions, 30-60min/session).

#### What opportunity do I have to consider this invitation?

You have two weeks to consider this invitation and give a response as to whether you want to participate or not.

#### How do I agree to participate in this research?

Contact the principle investigator (Jordan McIntyre, contact details below) to join the study and receive the necessary consent forms and information.

#### Will I receive feedback on the results of this research?

You will be given individual feedback on your aerobic assessment and it's comparison to the group means and with norms for your exercise population. Individualised feedback as well as group feedback will be provided to you on which warm-up condition suit you best and which was shown to be the most effective among all participants. Feedback will be given in written report format and you will be given the opportunity to seek further verbal feedback from the researchers.

This study will be published in scientific journals and presented at national or international exercise science conferences.

#### What do I do if I have concerns about this research?

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor, *Dr Andrew Kilding (see below)* 

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Madeline Banda, *madeline.banda* @*aut.ac.nz*, 921 9999 ext 8044.

#### Who do I contact for further information about this research?

Researcher Contact Details: Jordan McIntyre Institute of Sport and Recreation Research New Zealand Auckland University of Technology Division of Sport and Recreation Private Bag 92006

Ph: 921 9999 x 7119 Mobile: 021 110 7440 Email: jordan.mcintyre@aut.ac.nz

Project Supervisor Contact Details: Dr Andrew Kilding Institute of Sport and Recreation Research New Zealand Auckland University of Technology Division of Sport and Recreation Private Bag 92006

Ph: 921 9999 x 7056 Email: <u>andrew.kilding@aut.ac.nz</u>

Approved by the Auckland University of Technology Ethics Committee on 10 April 2006, AUTEC Reference number Kilding06/68. Appendix 2-2: Participant consent form

**Consent to Participation in Research** 



Title of Project:	The effects of different intermittent 'warm-up' strategies on 3km cycling performance.
Project Supervisor:	Dr Andrew Kilding
Researcher:	Jordan McIntyre
	d understood the information provided about this research project neet dated 24/04/2006)
I have had an o	opportunity to ask questions and to have them answered.
	at I may withdraw myself or any information that I have provided for this ime prior to completion of data collection, without being disadvantaged in
I agree to take	part in this research.
I wish to receiv	e a copy of the report from the research: $tick one$ : Yes O No O
Participant signature:	
Participant name:	
Participant Contact Det	ails (if appropriate):
Date:	

## Approved by the Auckland University of Technology Ethics Committee on 10 April 2006, AUTEC Reference number Kilding06/68

Note: The Participant should retain a copy of this form.



## **MEMORANDUM**

To:	Andrew Kilding
From:	Madeline Banda Executive Secretary, AUTEC
Date:	10 May 2006
Subject:	Ethics Application Number 06/68 The effects of different intermittent 'warm up'
-	strategies on 3km cycling performance.

#### Dear Andrew

Thank you for providing written evidence as requested. I am pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC) at their meeting on 10 April 2006. Your ethics application is now approved for a period of three years until 10 May 2009. I advise that as part of the ethics approval process, you are required to submit to AUTEC the following:

- A brief annual progress report indicating compliance with the ethical approval given using form EA2, which is available online through <u>http://www.aut.ac.nz/research/ethics</u>, including a request for extension of the approval if the project will not be completed by the above expiry date;
- A brief report on the status of the project using form EA3, which is available online through <u>http://www.aut.ac.nz/research/ethics</u>. This report is to be submitted either when the approval expires on 10 May 2009 or on completion of the project, whichever comes sooner;

You are reminded that, as applicant, you are responsible for ensuring that any research undertaken under this approval is carried out within the parameters approved for your application. Any change to the research outside the parameters of this approval must be submitted to AUTEC for approval before that change is implemented.

Please note that AUTEC grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

To enable us to provide you with efficient service, we ask that you use the application number and study title in all written and verbal correspondence with us. Should you have any further enquiries regarding this matter, you are welcome to contact Charles Grinter, Ethics Coordinator, by email at <u>charles.grinter@aut.ac.nz</u> or by telephone on 921 9999 at extension 8860.

On behalf of the Committee and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

Madeline Banda Executive Secretary Auckland University of Technology Ethics Committee Cc: Jordan McIntyre jordan.mcintyre@aut.ac.nz