FATIGUE DURING HIGH-INTENSITY EXERCISE: RELATIONSHIP TO THE CRITICAL POWER CONCEPT

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I certify that all material in this thesis which is not my own work has been identified and that no material has previously been submitted and approved for the award of a degree by this or any other University.

Weerapong Chidnok

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Abstract

The hyperbolic power-duration relationship for high-intensity exercise is defined by two parameters: an asymptote (critical power; CP) reflecting the highest sustainable rate of oxidative metabolism, and a curvature constant (W′), which indicates a fixed amount of work that can be completed above CP ($W_{>CP}$). According to the CP model of bioenergetics, constant work rate exercise above CP depletes the capacity-limited W′ with fatigue occurring when W′ is completely expended. The complete depletion of W′ has been reported to occur when $\dot{V}_{O2max}$ is attained and a critical degree of muscle metabolic perturbation (decline of finite anaerobic substrates and accumulation of fatigue-related metabolites) is reached. However, while the CP model is effective at predicting metabolic perturbation and the tolerable duration of severe-intensity constant work rate (CWR) exercise, it is unclear if metabolic perturbation and exercise performance can be explained by the CP model when different methods of work rate imposition are applied. Therefore, the purpose of this thesis was to: 1) investigate the efficacy of the CP concept to predict performance in exercise tests using different work rate forcing functions; and 2) explore whether the physiological bases for W′ are consistent across different methods of work rate imposition. In study 1, compared to severe-intensity CWR exercise, the tolerable duration of intermittent severe-intensity exercise with heavy- (S-H) moderate- (S-M) and light-intensity (S-L) 'recovery' intervals was increased by 47%, 100% and 219%, respectively. $W_{>CP}$ (W′) was significantly greater by 46%, 98%, and 220% for S-H, S-M and S-L, respectively, when compared to S-CWR, and the slopes for the increases in $\dot{V}_{O2}$ and iEMG were progressively lowered as the recovery work rate was reduced. In study 2, both the $\dot{V}_{O2max}$ and $W_{>CP}$ were similar across incremental cycling protocols that imposed a fixed ramp rate and cadence (4.33 ± 0.60 $L \cdot min^{-1}$; 14.8 ± 9.2 kJ), a fixed ramp rate with cadence self-selected by the subjects (4.31 ± 0.62 $L \cdot min^{-1}$; 15.0 ± 9.9 kJ) and a step

incremental test where subjects were instructed to select power output according to prescribed increments in ratings of perceived exertion (4.36 ± 0.59 L·min^{-1}; 13.0 ± 8.4 kJ). In study 3, the $\dot{V}_{O2max}$ and $W_{>CP}$ were also not different across a 3 min all-out cycling test (4.10 ± 0.79 L·min^{-1}; 16.5 ± 4.0 kJ), cycling at a constant work rate predicted to lead to exhaustion in 3 min until the limit of tolerance (4.20 ± 0.77 L·min^{-1}; 16.6 ± 7.4 kJ) and a self-paced 3 min work-trial (4.14 ± 0.75 L·min^{-1}; 15.3 ± 5.6 kJ). In study 4, after completing severe-intensity exercise (>CP) to exhaustion, muscle homeostasis ([PCr], pH, [ADP] and [P_i]) returned towards baseline and subjects were able to exercise for at least 10 min at a heavy-intensity work rate (<CP); however, when the work rate was lowered but remained in the severe-intensity domain (>CP), muscle metabolites ([PCr], pH, [ADP] and [P_i]) did not recover and exercise tolerance was severely limited (39 ± 31 s). Finally in study 5, during severe-intensity intermittent knee extension exercise, the tolerable duration of exercise was 304 ± 68 s when 18 s recovery was allowed and was increased by ~69% and ~179% when the intermittent recovery periods were extended to 30 s and 48 s, respectively. The increased exercise tolerance with longer recovery periods occurred in concert with increased $W_{>CP}$ (3.8 ± 1.0 kJ, 5.6 ± 1.8 kJ and 7.9 ± 3.1 kJ for the intermittent protocols with 18, 30 and 48 s of recovery, respectively) and a delayed attainment of critical intramuscular metabolite concentrations ([PCr], pH, [ADP] and [P_i]). Therefore, the results of this thesis demonstrate that fatigue during various high-intensity exercise protocols is influenced by the capacity to complete work above the CP (W′) and that W′ depletion is linked to the attainment of $\dot{V}_{O2max}$ and the attainment of critical levels of intramuscular [PCr], pH, [ADP] and [P_i]. These findings suggest that the CP model can be adapted to predict the degree of metabolic perturbation and exercise performance across a range of exercise settings in humans.

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Symbols and abbreviation

[ ]	concentration
Δ	difference
%Δ	% difference between GET and $\dot{V}_{O2max}$
^{31}P-MRS	^{31}phosphorous nuclear magnetic resonance spectroscopy
ADP	adenosine diphosphate
ATP	adenosine triphosphate
Ca^{2+}	calcium
CI	confidence interval (e.g., 95% CI; CI_{95})
CO_2	carbon dioxide
CP	critical power (i.e., asymptote of the power/time hyperbola)
EMG	electromyogram
ET	endurance training
GET	gas exchange threshold
H^+	hydrogen ion/proton
HR	heart rate
iEMG	integrated electromyogram (μV·s)
K^+	potassium ion
MVC	maximal voluntary contraction
O_2	oxygen
P	power output
PCr	phosphocreatine (or creatine phosphate)
P_i	inorganic phosphate
T_{lim} / T_e	limit of tolerance/ time-to-exhaustion
$\dot{V}_{CO2}$	carbon dioxide output

$\dot{V}_E$	pulmonary ventilation (expired)
$\dot{V}_{O2}$	pulmonary oxygen uptake
$\dot{V}_{O2max}$	maximum oxygen uptake
$\dot{V}_{O2peak}$	peak oxygen uptake
W	watt
W′	curvature constant of the hyperbolic power-duration relationship
WR	work rate

Declaration

The material contained within this thesis is original work conducted and written by the author. The following communications and publications are a direct consequence of this work.

Publications

Chidnok W, DiMenna FJ, Bailey SJ, Vanhatalo A, Morton RH, Wilkerson DP, Jones AM. Exercise tolerance in intermittent cycling: application of the critical power concept. *Med Sci Sports Exerc.* 2012; 44: 966-76.

Chidnok W, Dimenna FJ, Bailey SJ, Burnley M, Wilkerson DP, Vanhatalo A, Jones AM. $\dot{V}_{O2max}$ is not altered by self-pacing during incremental exercise. *Eur J Appl Physiol*. 2013; 113: 529-539.

Chidnok W, Dimenna FJ, Bailey SJ, Burnley M, Wilkerson DP, Vanhatalo A, Jones AM. Effects of pacing strategy on work done above critical power during high-intensity exercise. *Med Sci Sports Exerc.* 2013; [Epub ahead of print].

Chidnok W, Fulford J, Bailey SJ, DiMenna FJ, Skiba PF, Vanhatalo A, Jones AM. Muscle metabolic determinants of exercise tolerance following exhaustion: relationship to the 'critical power'. *J Appl Physiol,* 2013; [Epub ahead of print].

Conference communications

Chidnok W, DiMenna FJ, Bailey SJ, Vanhatalo A, Wilkerson DP, Jones AM (2011). Effect of 'recovery' intensity on oxygen uptake and exercise tolerance during severe intermittent cycling (Abstract). *BASES Annual Student Conference, University of Chester, UK.*

Chidnok W, DiMenna FJ, Bailey SJ, Vanhatalo A, Wilkerson DP, Jones AM (2012). Maximal oxygen uptake is not different during incremental exercise tests where work rate is self-selected or experimentally imposed (Abstract). *BASES Student Conference, University of East London, UK.*

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Other publications

Dimenna FJ, Bailey SJ, Vanhatalo A, **Chidnok W**, Jones AM. Elevated baseline $\dot{V}_{O_2}$ per se does not slow O_2 uptake kinetics during work-to-work exercise transitions. *Journal of Applied Physiology*, 2010; 109(4): 1148-54.

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Chapter 1 Introduction

Human locomotion is driven by the contraction of the skeletal muscles. Skeletal muscle contraction is an active mechanical process and, as such, the repeated muscle contractions that occur during exercise depend on a continuous energy supply. The release of chemical energy from the hydrolysis of intramuscular adenosine triphosphate (ATP) is coupled to the mechanical process of skeletal muscle contraction; however, intramuscular ATP stores are finite and rapidly depleted as the muscles contract. If the rate of muscle ATP turnover fails to meet the energetic demands of muscle contraction, muscle contractions become prohibited and exercise is terminated. Fortunately, skeletal muscles possess a number of diverse ATP resynthesis pathways that are able to sustain muscle contractions during exercise.

In general, ATP can be resynthesised through metabolic pathways that operate independent of oxygen (O_2), which are collectively referred to as anaerobic metabolic pathways, and through a metabolic pathway that is dependent on O_2, termed oxidative metabolism. The principal anaerobic metabolic pathways include the ATP-PCr system and anaerobic glycolysis. Following the onset of exercise there is a rapid decline in muscle phosphocreatine (PCr) owing to the catalytic activity of the creatine kinase (CK) enzyme. Chemical energy liberated during PCr hydrolysis is used to resynthesise ATP with a 1:1 stoichiometry. The anaerobic catabolism of glucose also proceeds rapidly after exercise commences with 2 molecules of ATP resynthesised from each molecule of glucose that undergoes anaerobic glycolysis. However, while PCr hydrolysis and anaerobic glycolysis can resynthesise ATP very rapidly, these anaerobic metabolic pathways are limited in capacity since they rely on finite metabolic substrates (PCr and glycogen). Significant dependence on the ATP-PCr system and anaerobic glycolysis

also results in accumulation of metabolic by-products, adenosine di-phosphate (ADP), inorganic phosphate (P_i), and hydrogen ions (H^+), which perturb the intramuscular milieu and have been purported to interfere with the process of muscle contraction. Therefore, ATP turnover through anaerobic metabolic pathways is capacity-limited and not sustainable.

Oxidative resynthesis of ATP can utilise both carbohydrate and fat as macronutrient substrates. In comparison to the anaerobic catabolism of glucose, which promotes the resynthesis of two ATP molecules, glucose oxidation yields 38 molecules of ATP. Accordingly, glucose oxidation is a more efficient use of this finite energy reserve. Moreover, since fat oxidation has a greater ATP yield compared to carbohydrate oxidation, the capacity for ATP resynthesis is greater through aerobic than anaerobic metabolism. Another advantage of oxidative metabolism is that its metabolic by-products, carbon dioxide (CO_2) and water (H_2O), are easily regulated through ventilation and osmosis, respectively, such that intramuscular homeostasis is preserved. Therefore, ATP turnover through aerobic metabolism is not capacity-limited and can be sustained for a prolonged duration. When the aerobic and anaerobic energy systems are considered together, a greater proportional energy contribution from aerobic metabolism, and by extension a lower energy contribution from anaerobic metabolism at a given work rate, is likely to improve skeletal muscle fatigue resistance and endurance exercise performance.

The critical power (CP) model of human bioenergetics proposes that two endogenous energy supply components (CP and W′) interact to dictate the limit of tolerance during high-intensity exercise (Jones et al., 2010; Monod and Scherrer, 1965; Poole et al., 1988). CP is given by the asymptote of the hyperbolic relationship between power

output (P) and the T_{lim} during high-intensity exercise, while the curvature constant of the power-duration hyperbola (W′) represents a fixed amount of work that can be performed above CP (Jones et al., 2010; Monod and Scherrer, 1965; Moritani et al., 1981) (Figure 1). The extent to which a steady state in oxygen uptake ($\dot{V}O_2$) can be achieved, and thus the extent to which the ATP demand of exercise can be principally met through oxidative energy turnover, is dictated by the position of the work rate relative to the CP. When exercising below CP, $\dot{V}O_2$ attains a steady state such that the reliance on the anaerobic energy reserves is low and muscle homeostasis is maintained, whereas exercising above CP prevents the attainment of a $\dot{V}O_2$ steady state leading to continued increase in ATP turnover through anaerobic metabolism, and perturbation of the intramuscular milieu, until T_{lim} is attained (Jones et al., 2008; Poole et al., 1988; Wagner, 2000). Therefore, the CP of a muscle (or muscular group) in both dynamic work and isometric exercise is considered to represent the highest sustainable rate of oxidative metabolism (Hill and Smith, 1993; Monod and Scherrer, 1965; Moritani et al., 1981).

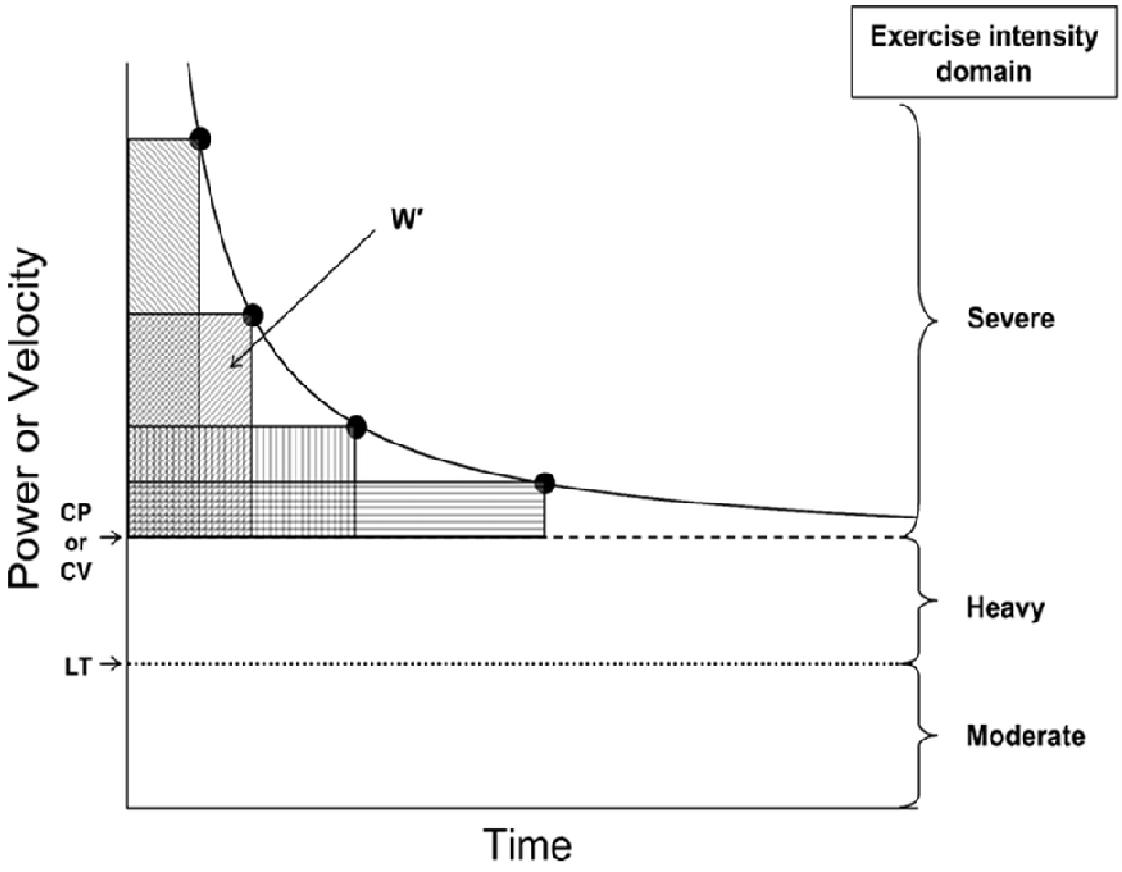

Figure 1

The three exercise intensity domains illustrating moderate-, heavy- and severe-intensity exercise. The moderate exercise intensity domain includes all work rates below the lactate threshold (LT) or gas exchange threshold (GET), the heavy exercise intensity domain comprises those work rates performed between the GET and CP, and the severe exercise intensity domain is given by all work rates performed above the CP that elicit the $\dot{V}_{O2max}$. For severe-intensity exercise, the hyperbolic power-time (P-*t*) relationship is described by two parameters: the CP, which is the asymptote for power (during cycling exercise) or velocity (during running exercise) which theoretically represents the highest sustainable work rate; and the curvature constant (W′), which indicates the maximum amount of work (in kJ for cycling or distance for running) that can be completed above CP (Burnley and Jones, 2007; Jones et al., 2010).

The W′ appears to be linked to ATP turnover through PCr hydrolysis and anaerobic glycolysis, and the attendant accumulation of intra-muscular (H^+, ADP, and P_i) and extra-muscular (potassium [K^+]) metabolites (Fukuba et al., 2003; Jones et al., 2008; Monod and Scherrer, 1965; Moritani et al., 1981; Poole et al., 1988; Vanhatalo et al., 2010). According to the CP model, if an individual exercises at a power output that is less than or equal to CP, energetic demands can be met principally by aerobic means such that exercise can continue for a considerable period of time (at least 1 hour). When power output exceeds CP, however, the rate of aerobic energy supply is insufficient to meet the metabolic demand and the resultant shortfall must be satisfied using the capacity-limited W′. Importantly, the development of the $\dot{V}_{O_2}$ slow component (a delayed-onset continued rise in $\dot{V}_{O_2}$ during exercise completed above the gas-exchange threshold) and the depletion of the W′ occur concomitantly such that the depletion of a finite capacity for $W_{>CP}$ (W′) and the achievement of a reproducible maximal $\dot{V}_{O2}$ ($\dot{V}_{O2max}$) occur consistently when exhaustion ensues during supra-CP exercise protocols (Burnley and Jones, 2007; Ferguson et al., 2007; Jones et al., 2010; Murgatroyd et al., 2011; Vanhatalo et al., 2011; Figure 2). These events appear to coincide with the attainment of some critical level of high-energy phosphate depletion and/or metabolite accumulation (Jones et al., 2008; Poole, et al., 1988; Wagner, 2000). Therefore, the tolerable duration of exercise above the CP appears to depend upon the interaction between the W′, the $\dot{V}_{O_2}$ slow component and the $\dot{V}_{O_2max}$ (Burnley and Jones, 2007). Accordingly, interventions which delay the attainment of the $\dot{V}_{O_2max}$, by reducing the trajectory of the $\dot{V}_{O_2}$ slow component, would be expected to improve exercise tolerance by reducing the consumption of the finite anaerobic energy reserves, the accumulation of fatigue-inducing metabolites and therefore, the depletion of W′ (Burnley and Jones, 2007; Jones and Burnley, 2009). Alternatively, interventions which

increase the $\dot{V}_{O2max}$ might increase W′ and thus the capacity to perform work above the CP and exercise tolerance.

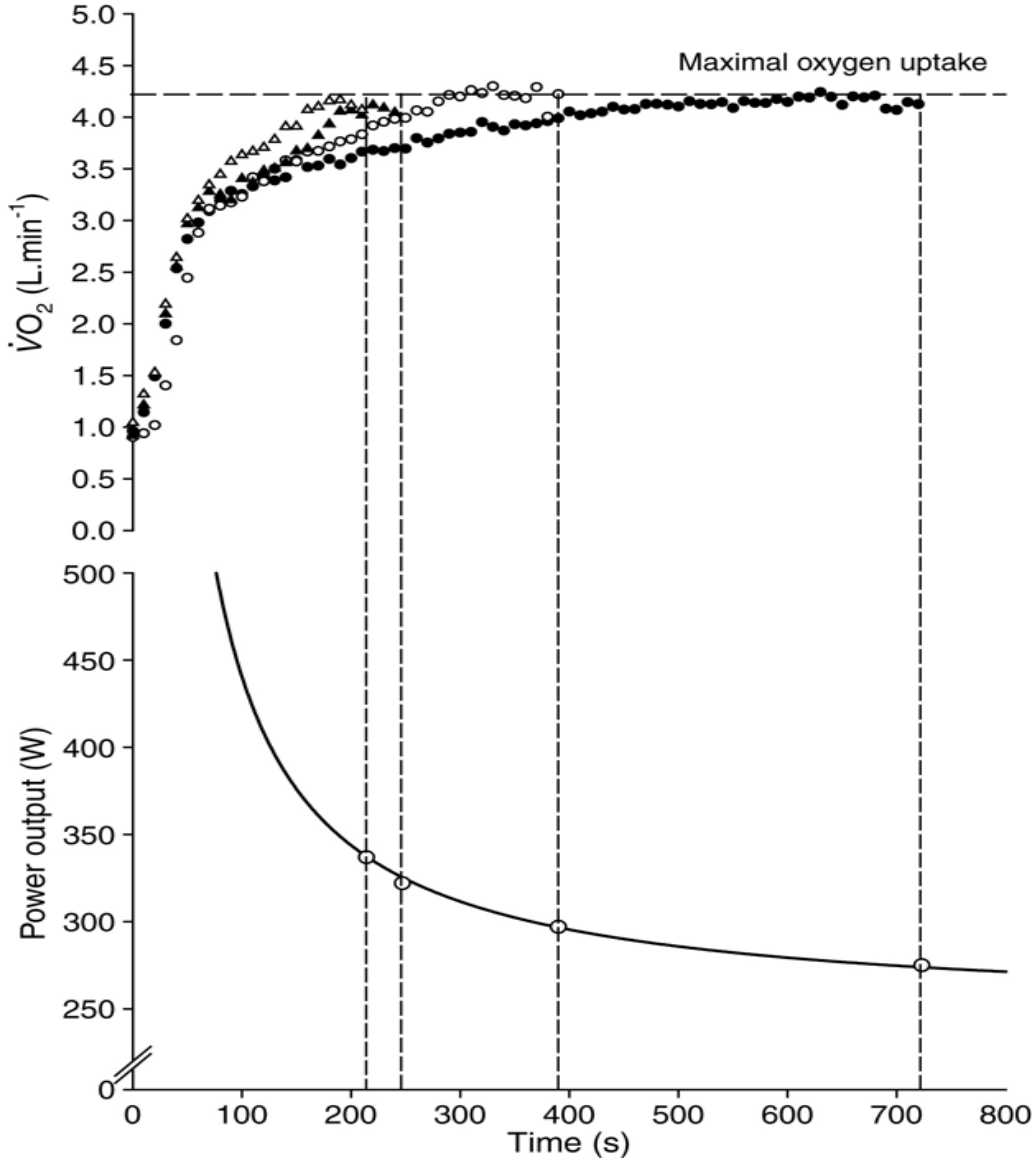

Figure 2

Derivation of the power-duration relationship from severe-intensity exercise bouts. The upper panel depicts the $\dot{V}_{O2}$ response profiles at different work rates between 85-100% $\dot{V}_{O2max}$, with the end-point $\dot{V}_{O2}$ being equal to $\dot{V}_{O2max}$ in each case. The lower panel depicts the power outputs plotted against time to exhaustion to show the hyperbolic character of the power-duration relationship (Burnley and Jones, 2007). Note that the attainment of the $\dot{V}_{O2max}$ temporally coincides with the depletion of the W′ and the termination of exercise.

Most of our understanding of the CP concept and its mechanistic bases is based on studies employing constant work rate (CWR) exercise. The extent to which the CP model is effective at predicting metabolic perturbation (decline of finite anaerobic substrates and accumulation of fatigue-related metabolites) and exercise performance when different work rate forcing functions (incremental, intermittent and self-paced exercise) are imposed is less clear. There are conflicting findings as to whether CP and W′ estimated from intermittent exercise are similar (and therefore have the same physiological equivalents) to those measured during continuous exercise (Buchheit et al., 2008; Kachouri et al., 1996; Morton and Billat, 2004). Moreover, recent data suggest that the $\dot{V}_{O_{2max}}$ can be increased during a self-paced incremental exercise test (Mauger and Sculthorpe, 2012), an effect which would be expected to increase the amount of work performed above the CP ($W_{>CP}$) and the tolerable duration of exercise. Understanding whether the CP model is applicable during intermittent and self-paced exercise is important since human locomotion during tasks of daily living and sporting competitions is rarely performed at a constant work rate. Therefore the purpose of this thesis was to test whether the CP model of human bioenergetics is also valid during exercise where work rate is not held constant. The thesis was also designed to explore the mechanistic bases of the CP model when applied to different work rate forcing functions. A premise of the thesis is that, during high-intensity exercise, the W′ is closely related to the fatigue process, irrespective of the work rate forcing function or degree of self-pacing.

Chapter 2 Review of Literature

The tolerable duration of exercise appears to be linked to the metabolic response profile that is exhibited during exercise. The metabolic responses during CWR exercise are highly predictable depending upon the exercise-intensity domain within which an individual is exercising with important implications for exercise tolerance (Burnley and Jones, 2007; Jones and Poole, 2005; Jones et al., 2008; Poole et al., 1988; Whipp and Wasserman, 1972; Wilkerson et al., 2004).

Exercise intensity domains

Moderate-intensity exercise domain

During moderate-intensity exercise, which comprises all work rates below the gas exchange threshold (GET), pulmonary $\dot{V}O_2$ rises in a mono-exponential fashion to attain a steady state within 2-3 minutes (Whipp and Wasserman, 1972). Consequently, there is no significant elevation in blood [lactate] and as such moderate exercise can be tolerated for several hours, with fatigue likely being mediated by glycogen depletion, muscle damage, increased core temperature and central fatigue (González-Alonso et al., 1997; St Clair Gibson et al., 2001).

Heavy-intensity exercise domain

The heavy intensity domain comprises all work rates above GET but below CP. It is also important to note that the maximal lactate steady state (MLSS), defined as the highest work rate where the increase in blood [lactate] is less than 1 mM between 10 and 30 minutes of exercise (Beneke et al., 2000; Jones and Doust, 1998; Snyder et al., 1994), is also a measure of the boundary between the heavy and severe exercise intensity domains (Pringle and Jones, 2002; Smith and Jones, 2001) The fundamental $\dot{V}O_2$ response is supplemented by an additional $\dot{V}O_2$ slow component at work rates above

the GET. This $\dot{V}O_2$ slow component emerges ~ 100-180 s into the exercise bout and is accompanied by an elevated blood [lactate]. In this intensity domain, the $\dot{V}O_2$ slow component and blood [lactate] will eventually stabilise at submaximal values (Whipp and Wasserman, 1972) and the tolerable duration of exercise is in the range of 20 min - 3 hours.

Severe-intensity exercise domain

Exercise performed above the CP/MLSS is classified as severe-intensity exercise. Here, both $\dot{V}O_2$ and blood [lactate] increase inexorably until the peak values are attained (Åstrand and Saltin, 1961; Wasserman and Whipp, 1975; Poole et al., 1988; Gaesser and Poole, 1996). In this exercise intensity domain, exercise tolerance can be predicted based on the hyperbolic relationship between power output and time to exhaustion (Monod and Scherrer, 1965; Poole et al., 1988; Hill et al., 2002). As a result, the tolerable duration of severe exercise is < 20 minutes (Poole et al., 1988).

Extreme-intensity exercise

Whereas severe exercise leads to fatigue owing to the attainment of the $\dot{V}O_{2max}$, extreme-intensity exercise is characterised by fatigue ensuing prior to the attainment of the $\dot{V}O_{2max}$ (Hill et al., 2002). The tolerable duration of exercise within this intensity domain is typically restricted to <140 s and blood [lactate] may be lower than that observed immediately post severe exercise consequent to the reduced exercise duration (Hill et al., 2002).

The focus of this thesis was to explore the physiological bases for severe-intensity exercise tolerance and performance across a variety of exercise tests and how this relates to the CP model of human bioenergetics. This review of literature aims to

outline: 1) the evolution of the CP concept; 2) the components of the CP model of human bioenergetics and current understanding of the physiological bases for its constituents; 3) the role of the CP model in predicting the tolerable duration of severe-intensity CWR exercise; and 4) recent adaptations of the CP model that might permit its application in different exercise contexts.

The critical power concept: Historical development

In 1925, A.V. Hill characterised the relationship between velocity and world record times for various athletic events (Hill, 1925). There has been significant interest in the relationship between work/power/velocity and time since these seminal observations by Hill (1925) and an overview of key developments that have shaped our understanding of these relationships is summarised below.

Work-time model

Monod and Scherrer (1965) reported that when a series of exhaustive CWR tests were performed in an isolated muscle group, there was a linear relationship between work done (W) and the time to exhaustion (T_{lim}). The slope of the relationship was termed CP, which the authors defined as and the "the maximum rate (of work) that it can keep up for a very long time without fatigue", and a ‘fixed energetic reserve’ was indicated by the y-intercept of the W-T_{lim} relationship (Monod and Scherrer, 1965). Today, CP is considered as the highest sustainable rate of aerobic metabolism (Hill, 1993; Monod and Scherrer, 1965; Moritani et al., 1981) and the ‘fixed energetic reserve’ is termed the W′ (Fukuba et al., 2003; Gaesser et al., 1995; Poole et al., 1988; Smith and Hill, 1993). Many investigations have provided evidence that this work-time model accurately describes experimental data in cycling (Jenkins and Quigley, 1990; Moritani et al.,

1981; Poole et al., 1990) and in running (Hughson et al, 1984; Smith and Jones, 2001). The work-time model in its modern form is given by:

$$W = CP \times T + W' \qquad \textit{Equation 1}$$

Importantly, *Equation 1* can be used to accurately calculate the best possible performance time for a given quantity of work after the work-time relationship has been established and the CP and W′ parameters have been derived.

Power-time model

Given that work is a function of time and power output (W = P x T), Moritani et al. (1981) reasoned that time to exhaustion for any constant power output greater than CP could be calculated by rearranging the work-time model with T as the dependent variable. This has been confirmed in several studies that have reported an excellent fit of experimental data from whole body exercise to a hyperbolic power-time relationship (Gaesser et al., 1995; Hill, 2004; Hill et al., 2002; Hill and Smith, 1999). The power-time function is written as:

$$T = W' / (P - CP) \qquad \textit{Equation 2}$$

Appreciation of the power-time model is improved if the axes are reversed so that the time continuum is set along the x-axis (Figure 1).

1/time model

The hyperbolic power-time function can be linearised by setting the power output as the dependent variable:

$$P = W'/T + CP \quad \text{or} \quad P = W'(1/T) + CP \qquad \textit{Equation 3}$$

The above function is known as the 1/time model, where the y-intercept represents CP and the slope of the linear regression indicates the W′. This linearised form of the power-time relationship has also been shown to provide an accurate fit to experimental data in several experimental studies (Coats et al., 2003; Miura et al. 2000; 1999; Poole et al., 1988; Pringle and Jones, 2002).

The 3 min all-out test

It is well documented that the power-time curves are constructed using data obtained from four or more independent high-intensity constant power exercise bouts for which the tolerable duration is 2-15 min (Poole et al., 1988). This conventional approach to the CP and W′ is experimentally demanding owing to the completion of 4-5 exhaustive exercise bouts. Recently, it has been proposed that the CP and W′ can be estimated using a single all-out protocol where the W′ is expended as a function of time until W′ = 0 and the highest power output that can be sustained at the end of the test approximates CP. The all-out test power profile is reliable (Burnley, 2006), the variables show close agreement with the estimates from the conventional protocol (Vanhatalo, 2007; Vanhatalo, 2008) and the test can be used to accurately predict exercise tolerance (Vanhatalo, 2011). The CP derived from the all-out test is also sensitive to a change after high intensity interval training (Vanhatalo, 2008).

Therefore, CP and W′ can be determined from a variety of different exercise testing protocols.

Critical power as a physiological and performance threshold

In a landmark study, Poole et al. (1988) examined pulmonary gas exchange and the blood [lactate] response during CWR cycling exercise at CP and 5% above CP. The authors found that $\dot{V}_{O_2}$ and blood [lactate] response profiles stabilised at submaximal values and exercise could be tolerated for at least 24 min without fatigue during exercise at CP. However, for exercise just 5% above CP, $\dot{V}_{O_2}$ rose inexorably to $\dot{V}_{O_{2max}}$ and blood [lactate] increased progressively until the subject was unable to continue the exercise task (Poole et al., 1988). More recently, Jones et al. (2008) showed using ^{31}P-magnetic resonance spectroscopy (^{31}P-MRS) that when CWR exercise was performed slightly above CP, intramuscular [PCr] and pH continued to decrease, and [P_i] continued to increase, until the T_{lim} was reached. During exercise performed just below the CP, however, stable values for [PCr], pH and [P_i] were attained within 3 min of the start of exercise and exercise could be tolerated for at least 20 min without fatigue (Jones et al., 2008). Taken together, these findings demonstrate that the CP separates a range of work rates that elicit the $\dot{V}_{O_{2max}}$, critical intramuscular metabolite concentrations, and reduced exercise tolerance (severe-intensity exercise) from a range of work rates that elicit stable and submaximal values for $\dot{V}_{O_2}$ and intramuscular metabolites and can be well tolerated (heavy-intensity exercise) (Poole et al., 1988; Jones et al., 2008; Jones et al., 2010).

The critical power concept: A two parameter model

A.V. Hill and Otto Meyerhof were the first to demonstrate that both aerobic and anaerobic energy sources were utilised to fuel high-intensity skeletal muscle contractions, and they were awarded the Nobel Prize for Physiology and Medicine in 1922 based on this work. The CP concept extends the pioneering work of Hill and Meyerhoff by describing how aerobic and anaerobic energy sources interact to

determine the tolerable duration of exercise. Indeed, the CP concept describes an endogenous bioenergetics supply system that can be defined by a two-parameter model; i.e., an aerobic component that is rate- but not capacity-limited (CP), and a supplementary component linked to anaerobic metabolism that remains constant regardless of the rate at which it is expended (W′) (Jones et al., 2010). The CP model proposes that when an external power output exceeds CP, the rate of aerobic energy supply is insufficient to meet the metabolic demand and the resultant shortfall must be satisfied using the capacity-limited W′ with exhaustion occurring when W′ is completely expended. While it is accepted that the CP is determined by oxidative metabolism, the mechanistic bases for the W′ (a fixed amount of work that can be completed above CP) are not entirely understood (Jones et al., 2010). Evidence to support the dependence of the CP on oxidative metabolism is provided by observations that CP is lowered when exercising in hypoxia (Moritani et al., 1981), and increased when exercising in hyperoxia (Vanhatalo et al., 2010). The W′ parameter has traditionally been considered to represent a fixed anaerobic energy reserve (Monod and Scherrer, 1965; Moritani et al., 1981) and observations that W′ can be lowered with glycogen depletion (Miura et al., 2000) and might be increased with creatine supplementation (Miura et al., 1999; Smith et al., 1998) support this notion. However it has recently been argued that, since PCr hydrolysis (Rossiter et al., 2001, 2002) and the accumulation of anaerobic metabolic by-products (ADP, P_i) have an important role in signalling oxidative phosophorylation (Chance and Williams, 1955; Balaban, 1992; Brown, 1992; Bose et al., 2003), and since O_2 bound to muscle myoglobin and haemoglobin in venous blood are also considered to contribute towards W′ (Miura et al., 1999, 2000; Monod and Scherrer, 1965; Moritani et al., 1981), classifying the CP and W′ as discrete aerobic and anaerobic processes is an oversimplification (Jones et al., 2010). Therefore, while W′ appears to be linked to ATP turnover through PCr

hydrolysis and anaerobic glycolysis, and the attendant accumulation of intra- (H^+, ADP and P_i) and extra-muscular (potassium [K^+]) metabolites (Fukuba et al., 2003; Jones et al., 2008; Monod and Scherrer, 1965; Moritani et al., 1981; Poole et al., 1988; Vanhatalo et al., 2010), further research is required to define the mechanistic bases for the W′.

Factors influencing the critical power model of bioenergetics

$\dot{V}O_2$ slow component

During exercise performed above the GET, the $\dot{V}O_2$ response during exercise is complicated by the emergence of the $\dot{V}O_2$ slow component. It has been established that the $\dot{V}O_2$ slow component is of delayed onset (Barstow and Molé, 1991; Paterson and Whipp, 1991) and results in the $\dot{V}O_2$ steady-state being delayed (heavy-intensity exercise) or unattainable (severe-intensity exercise) (Whipp and Wasserman, 1972; Barstow and Molé, 1991; Paterson and Whipp, 1991). Since the development of the $\dot{V}O_2$ slow component and the depletion of W′ have been reported to occur in synchrony during severe-intensity exercise (Burnley and Jones, 2007; Ferguson et al., 2007), the response characteristics of the $\dot{V}O_2$ slow component is likely to have important implications for the tolerable duration of severe-intensity exercise. It has been shown that approximately 86% of the pulmonary $\dot{V}O_2$ slow component can be ascribed to processes intrinsic to the contracting musculature (Poole et al., 1991). The $\dot{V}O_2$ slow component has been linked to alterations in muscle fibre recruitment since it is negatively correlated with the % of type I muscle fibres (Barstow et al., 1996), it can be associated with changes in EMG (Shinohara and Moritani, 1996; Saunders et al., 2000; Borrani et al., 2001; Perry et al., 2001; Burnley et al., 2002; Osborne and Schneider, 2006; DiMenna et al., 2008; Layec et al., 2009; DiMenna et al., 2010) and it is observed during exercise which recruits type II muscle fibres but not during exercise that only

recruits type I muscle fibres (Krustrup et al., 2004). Moreover, the $\dot{V}_{O_2}$ slow component is accompanied by an increase in ATP turnover rate (Bangsbo et al., 2001, reflective of a progressive lowering of muscle contractile efficiency as exercise proceeds) and greater rates of glycogen utilisation (Krustrup et al., 2004), PCr utilisation (Rossiter et al., 2001, 2002) and muscle metabolite accumulation (Bailey et al., 2010; Vanhatalo et al., 2010). Therefore the $\dot{V}_{O_2}$ slow component is accompanied by muscle metabolic perturbation and these parameters appear to reflect the rate of W′ utilisation and, by extension, the capacity to complete severe-intensity exercise. In the severe-intensity exercise domain the $\dot{V}_{O_2}$ slow component is set on a trajectory that results in the attainment of the $\dot{V}_{O_{2max}}$ (Whipp and Wasserman, 1972; Barstow and Molé, 1991; Paterson and Whipp, 1991) and it has been suggested that the attainment of the $\dot{V}_{O_{2max}}$ and the complete depletion of the W′ are temporally concomitant (Burnley and Jones, 2007; Ferguson et al., 2007). Accordingly, interventions that blunt the development of the $\dot{V}_{O_2}$ slow component and/or increase the $\dot{V}_{O_{2max}}$ would be expected to improve severe-intensity exercise tolerance by delaying W′ depletion.

Maximal Oxygen Uptake

In 1923, Hill and Lupton studied subjects running at increasing speeds on an outdoor track and observed that oxygen intake rose steadily as speed was increased, attaining a maximum "beyond which no bodily effort can drive it" (Hill, 1923; Hill, 1924). This 'maximal oxygen uptake' ($\dot{V}_{O_{2max}}$) has since been defined as the highest $\dot{V}_{O_2}$ (i.e., rate of O_2 consumption), averaged over a 15- to 30 s period, which is attainable for a given form of exercise, as evidenced by a failure of $\dot{V}_{O_2}$ to increase further despite an increase in power output (Wasserman, 1994). Historically, $\dot{V}_{O_{2max}}$ has been considered to reflect the limits of the human cardio-respiratory system to transport and utilize O_2 and it has,

therefore, become a cornerstone of experimental, clinical and applied exercise physiology (Wasserman, 1994).

It is sufficient to say that the central process which determines $\dot{V}_{O2max}$ is dependent upon pulmonary, cardiovascular and haematological functional capacities. According to Fick's formula, $\dot{V}_{O2}$ is determined by two processes: central (oxygen transport) and peripheral (tissue oxygen extraction):

$$\dot{V}_{O2} = \dot{Q} \times C(a\text{-}\bar{v})O_2 \qquad \textit{Equation 4}$$

Systemic oxygen uptake can be expressed mathematically as the product of cardiac output ($\dot{Q}$) multiplied by arteriovenous oxygen concentration difference ($C(a\text{-}v)O_2$; *Equation 4*). At maximal exercise, the $\dot{V}_{O2}$ will reach its highest level ($\dot{V}_{O2max}$) which is governed by maximal cardiac output and maximal arterio-venous oxygen concentration difference (Wasserman, 1994):

$$\dot{V}_{O2max} = \dot{Q}_{max} \times C(a\text{-}\bar{v})O_{2max} \qquad \textit{Equation 5}$$

It is generally considered that $\dot{Q}$ and CaO_2 are the main factors determining $\dot{V}_{O2max}$ (Saltin and Strange, 1992; Wagner 2000; Gonzalez-Alonso and Calbet, 2003), but see Spurway et al. (2012) for other considerations.

The exercise protocol used to measure $\dot{V}_{O2max}$ has developed from a discontinuous series of bouts at different constant power outputs (Hill, 1923; Taylor, 1955) to single incremental or ramp exercise tests (Davis, 1982, Whipp, 1981). These latter tests sometimes include a 'verification phase' in which a power output which is higher than

the peak power output attained during the incremental test is subsequently sustained for as long as possible (Midgley, 2007; Rossiter, 2006). Evidence has been presented that, for a given mode of exercise, $\dot{V}_{O2max}$ is reproducible across different maximal-effort testing protocols (Bogaard, 1996; Davis, 1982; Takaishi, 1992; Zhang, 1991). Intriguingly, however, Mauger and Sculthorpe (2012) have recently reported that self-paced incremental cycling using 'clamps' of ratings of perceived exertion (RPE), i.e., five two-min stages during which subjects incremented the power output according to a prescribed RPE, resulted in the attainment of higher peak $\dot{V}_{O2}$ compared to a traditional $\dot{V}_{O2max}$ protocol in which power output increments were applied in a strictly linear fashion (40 ± 10 vs. 37 ± 8 $ml \cdot kg^{-1} \cdot min^{-1}$, respectively). The authors suggested that allowing subjects the ability to regulate power output in an anticipatory manner enabled them to reduce the influence of afferent signaling on the perception of discomfort and thereby avoid termination of the test before a 'true' $\dot{V}_{O2max}$ could be attained (Mauger and Sculthorpe, 2012). The authors also suggested that this greater level of subject autonomy in selecting power output and pacing the maximal effort might allow for increased or 'more efficient' muscle fiber recruitment; however, they did not assess muscle activation using, for example, electromyography (EMG) and so were unable to confirm this possibility. The authors observed a ~15% increase in peak power output during the self-paced test compared to the conventional incremental test and speculated that the protocol allowed for this higher peak power output (and the increased $\dot{V}_{O2}$ that it may require) to be achieved at the same level of discomfort.

It is well known that the peak $\dot{V}_{O2}$ attained during incremental exercise may be influenced by the total duration of the test. It has been suggested that incremental tests lasting longer than 12-14 min may result in lower peak $\dot{V}_{O2}$ values (Astorino, 2004; Yoon, 2007). The reason for this discrepancy is unclear, but might be linked to an

inability to reach the same absolute power output during longer tests and also to differences in fatigue mechanisms (Astorino, 2004; Jones, 2010; Yoon, 2007). Interestingly, in the study by Mauger and Sculthorpe (2012), the mean test duration in the conventional incremental test was ~30 % longer (i.e., ~13 min) than the self-paced test (i.e. 10 min). It is possible, therefore, that test duration contributed to the higher peak $\dot{V}_{O2}$ measured in the self-paced test compared to the conventional test (Mauger and Sculthorpe, 2012). However, if the peak $\dot{V}_{O2}$ can indeed be increased in a self-paced incremental test compared to a duration-matched ramp incremental test this would be expected to increase the W′ (Burnley and Jones, 2007; Ferguson et al., 2007).

Pacing strategy

It has been shown that variations in pacing strategy can influence exercise performance during high intensity exercise. Indeed, exercise performance is enhanced with an all-out or positive pacing strategy (Bailey et al., 2011; Bishop, 2002; De Koning, 1999; Foster et al., 1993; Jones et al., 2007), but not with a negative pacing strategy (Bailey et al., 2011; Jones et al., 2007). Importantly, the studies of Jones et al. (2007) and Bailey et al. (2011) used the CP model to predict a work rate at which the subjects would be expected to complete a fixed amount of work over a 3 min period. Given that the fast-start pacing strategy used in these studies resulted in more work being completed than predicted by the CP model, this suggests that pacing strategy might influence the CP model of bioenergetics and thus performance in severe-intensity exercise tasks. The study by Bailey et al. (2011) also showed that the fast-start strategy allowed $\dot{V}_{O2max}$ to be attained while the even-start and slow-start conditions did not. Given the relationship between $\dot{V}_{O2max}$ and W′, this suggests that the capacity to complete supra-CP work might be greater with a fast-start pacing strategy. During competition, exercise is self-paced and this is an important aspect of athletic performance (Foster,

1993) which allows the athlete to alter the power output responses during exercise when physiological homeostasis is challenged (Lander et al., 2009; St Clair Gibson et al., 2006). Pacing strategies vary according to event duration and an athlete's level of experience, but are believed to reflect an attempt to optimize performance without incurring premature or intolerable challenges to homeostasis (St Clair Gibson, 2006). Interestingly, there is evidence to suggest that self pacing reduces the metabolic stress associated with a given performance (Lander et al., 2009). For example, it has been shown that allowing subjects to self pace at a fixed rating of perceived exertion (RPE) enabled them to complete a 5000 m rowing bout in the same amount of time, but with less physiological perturbation (reductions in blood lactate, core temperature and iEMG), compared to enforced-pace CWR exercise where the same mean power output was maintained (Lander et al., 2009). This suggests that, once the ability to self-pace has been denied, the metabolic challenge would more rapidly lead to the attainment of maximal values (Lander et al., 2009). If correct, the amount of work performed above the CP and the peak $\dot{V}_{O2}$ might be enhanced during self-paced exercise relative to CWR exercise.

Intermittent exercise

It has been suggested that once W′ has been exhausted and the T_{lim} attained during supra-CP exercise, the work rate must be reduced below the CP in order for W′ to be reconstituted and for exercise to be continued (Coats et al., 2003). Coats and co-workers (2003) asked subjects to complete severe intensity CWR to T_{lim} (attained in around 6 min), and then immediately reduced the work rate to 80% GET, 90% CP, or 110% CP; the subjects then attempted to complete 20 minutes of exercise. It was reported that all six subjects completed the 20 min target time at 80% GET, only two subjects completed the 20 min target time at 90% CP (mean ± SD exercise time: 577 ± 306 s), while none of the subjects completed the 20 min target time at 110% CP (mean

± SD exercise time: 30 ± 12 s). The authors interpreted these findings to suggest that the W′ recovers in an intensity-dependent manner following supra-CP exercise with important implications for exercise tolerance (Coats, 2003). The physiological profiles during intermittent exercise have been much less well examined than constant work rate exercise. It is possible that the tolerable duration of intermittent exercise is a function of the exercise intensity and thus the potential for W′ reconstitution in the recovery interval. Similarly, enhancing the duration of the recovery interval separating intermittent exercise bouts would be predicted to extend the tolerable duration of intermittent exercise by enhancing W′ resynthesis between the work intervals. However, there are conflicting findings as to whether CP and W′ estimated from intermittent exercise are similar (and therefore have the same physiological equivalents) to those measured during continuous exercise (Buchheit et al., 2008; Kachouri et al., 1996; Morton and Billat, 2004). Further research is required to assess how manipulating the exercise intensity and recovery duration of the 'recovery' periods during intermittent exercise impacts on W′ resynthesis, exercise tolerance, and the metabolic bases for any changes in these variables.

Application of the critical power model during different work rate forcing functions

The majority of studies have employed the CP model as a tool to successfully predict the tolerable duration of severe-intensity CWR exercise. To what extent the CP model is effective in predicting the tolerable duration of exercise when a different pattern of work rate imposition is applied is less clear. It has been proposed that the CP model can be used to predict the time to exhaustion in ramp exercise using the equation developed by Morton (Morton, 2011; for review):

$$t = CP / S + \text{sqrt}(2W'/S) \quad \textit{Equation 6}$$

Where t is the desired time-to-exhaustion on a ramp incremental exercise test and S is the ramp slope (Watts/s).

In addition, Morton and Billat (2004) have adapted the two-parameter CP model for intermittent exercise by including four independent variables (P and t during both work and recovery phases) during intermittent exercise (*Equation 7*). This suggests that the CP model might also be appropriate for predicting the tolerable duration of intermittent exercise.

$$t = n (t_w + t_r) + [W' - n \{(P_w - CP) t_w - (CP - P_r) t_r\}] / (P_w - CP) \quad \textit{Equation 7}$$

Where t is total endurance time, n is the number of completed work/recovery cycles, t_w and t_r are the durations and P_w and P_r are the power outputs of the work and recovery intervals, respectively.

The recent development of these models is important since investigations can now be conducted to determine whether the CP model is also applicable during exercise tests employing methods of work rate imposition other than CWR exercise. These models have received limited empirical research attention to date.

Summary

The tolerable duration of severe-intensity CWR exercise is a hyperbolic function of the administered work rate. The asymptote of this rectangular hyperbola is the CP and the curvature constant is W′, the latter representing a fixed amount of work that can be completed above CP during severe-intensity CWR exercise. In the severe-intensity exercise domain (work rate >CP) W′ is depleted in synchrony with the development of the $\dot{V}_{O2}$ slow component, the depletion of the finite anaerobic reserves (PCr and glycogen) and the accumulation of muscle metabolites (H^+ and P_i). Complete depletion of W′ coincides with the attainment of $\dot{V}_{O2max}$, critically low levels of anaerobic energy reserves, critically high level of fatigue-inducing muscle metabolites and exhaustion during severe-intensity CWR exercise (Jones et al., 2008; Poole et al., 1988). While the CP model is effective at predicting the degree of metabolic perturbation and the tolerable duration of severe-intensity CWR exercise, its effectiveness to predict the physiological responses and tolerable duration of intermittent exercise and incremental exercise, and exercise performance during self-paced exercise, is less clear. It has been reported that the W′ recovers in an intensity-dependent manner following supra-CP exercise (Coats, 2003). However, the intramuscular bases for this intensity-dependent W′ recovery have yet to be investigated. The intramuscular bases for the recovery of W′ during intermittent exercise and the effects of different recovery durations on W′ and exercise tolerance is also unknown.

Aims

The overall purpose of this thesis was to assess the accuracy with which the CP concept could predict the tolerable duration of intermittent and incremental exercise, and exercise performance during self-paced exercise, in healthy adult humans. This series of investigations applied the CP concept to a variety of exercise protocols that employed

different work rate forcing functions to test the effectiveness of the CP model to predict performance and metabolic perturbation, and whether the CP model has application in tests that better reflect patterns of work rate distribution during sporting competitions. Where possible, these investigations also attempted to elucidate the mechanistic bases for alterations in physiological variables through use of techniques such as iEMG and ^{31}P-MRS. The overarching hypothesis is that 'exhaustion' attained during any type of high-intensity exercise comes about via the depletion of W′.

The specific aims of this thesis are as follows:

1) To investigate the effectiveness of the CP model to explain the physiological and performance responses during intermittent high-intensity cycling exercise.

2) To compare the peak $\dot{V}_{O2}$ attained and $W_{>CP}$ completed during self-paced incremental cycling test at fixed rating of perceived exertion (RPE) to the $\dot{V}_{O2max}$ and $W_{>CP}$ completed in a duration-matched enforced-pace incremental cycling test.

3) To determine whether $W_{>CP}$ and peak $\dot{V}o_2$ are similar during exhaustive enforced-pace CWR exercise, self-paced cycling (SPT), enforced-pace incremental exercise (INC) and all-out sprint cycling.

4) To investigate the mechanistic bases for the changes in exercise tolerance and the reconstitution of the W′ immediately following exhaustive severe-intensity exercise during exercise bouts performed at different recovery intensities using ^{31}P-MRS.

5) To determine the responses of intramuscular phosphate-linked metabolites (PCr, P_i and ADP) and pH during intermittent severe-intensity exercise bouts performed with different recovery durations were assessed using ^{31}P-MRS.

Hypotheses

This thesis will address the following hypotheses:

1) During exhaustive intermittent exercise in which severe exercise was interspersed with recovery periods in different exercise intensity domains (severe, heavy, moderate and light), the $W_{>CP}$, and therefore the T_{lim}, would be greatest for light, intermediate for moderate, and least for heavy recovery condition, while no W′ recovery would occur in severe recovery condition. It was also hypothesized that changes in the overall rate of W′ depletion during these intermittent exercise protocols would be reflected in changes to the rates at which $\dot{V}_{O_2}$ and iEMG increased to the limit of tolerance.

2) The highest $\dot{V}_{O_2}$ attained and $W_{>CP}$ would be enhanced during self-paced incremental exercise relative to fixed ramp rate incremental exercise tests continued to the limit of tolerance.

3) Subjects would achieve the same peak $\dot{V}_{O_2}$ and complete the same amount of work in excess of CP at the point at which exercise was terminated in the CWR, INC and SPT tests. It was also hypothesized that EP and WEP from the 3 min all-out test could be used to accurately predict T_{lim} for CWR and INC, and performance during SPT.

4) Recovery exercise <CP would be sustained for an appreciable duration without significant fatigue development after exhaustive exercise and muscle [PCr] and pH

would be recovered significantly following exhaustion. Moreover, exercise tolerance would be severely limited during recovery exercise >CP as a consequence of an inability to recover [PCr] and pH.

5) During exhaustive intermittent exercise in which severe exercise was interspersed with passive recovery periods of different durations (18 s, 30 s, and 48 s), the muscle metabolite reconstitution and the total work done above CP, and therefore the T_{lim}, would be greatest for long, intermediate for moderate, and least for short recovery durations.

Chapter 3 General Methods

General Experimental Procedures

The five experimental Chapters (Chapters 4-8) that comprise this thesis required 272 exercise tests to be conducted. All of the exercise tests were conducted in an air conditioned exercise physiology laboratory at sea level with an ambient temperature of 21°C. The procedures employed in each of these experimental Chapters were approved by the University of Exeter Research Ethics Committee prior to the commencement of data collection.

Subjects

The subjects who volunteered to participate in these investigations were recruited from the student and staff University community. Subjects were non-smokers who were free from disease and were not currently using dietary supplements. The subjects were all recreationally active at the point of recruitment and were familiar with the experimental procedures used in the study. Subjects were instructed to report to the laboratory in a rested state at least 3 hours postprandial, having completed no strenuous exercise within the previous 24 hours. Subjects were also instructed to avoid alcohol and caffeine for 24 and 6 hours, preceding each exercise test, respectively. Each subject underwent testing at the same time of day (±2 hours) and all subjects were familiarised with the mode(s) of exercise and experimental procedures prior to the initiation of the experimental testing.

Informed Consent

Before agreeing to participate in these investigations, subjects were given an information sheet that provided a detailed description of the experimental procedures they would be subjected to. The potential risks and benefits of participating in each of

these investigations was also clarified in the information sheet and subjects were informed that, while their anonymity would be preserved and their data safely stored, the data of the group of subjects investigated may be published in academic journals or presented at national/international conferences. It was also made clear to the participants that they were free to withdraw from the investigation at any point with no disadvantage to themselves. Any additional questions or concerns the subjects had were answered and, provided the subjects were clear and happy with all aspects of the study, they gave their written informed consent to participate.

Health and Safety

All testing procedures adhered to the health and safety guidelines established by the Sport and Health Sciences Department at the University of Exeter and great care was taken to ensure that the laboratory provided a clean and safe environment that was appropriate for exercise testing of human subjects. Ergometers, trolleys and work surfaces were cleaned using dilute Virkon disinfectant and all respiratory apparatus was similarly disinfected according to manufacturers' recommendations. Experimenters wore disposable latex gloves during blood sampling and all sharps and biohazard materials were disposed of appropriately. A proper 'cool-down' was provided upon completion of the requisite exercise challenge.

Measurement Procedures

Descriptive Data

For all investigations, each subject's stature and mass were measured and these along with age were recorded prior to the initiation of testing. In all experiments that employed cycle ergometry, the peak power output and $\dot{V}O_{2max}$ as well as the power

output and $\dot{V}_{O_2}$ at the GET were also determined during the preliminary exercise testing session (as described below).

Cycle Ergometry

In Chapters 4-6 cycle ergometry was the exercise modality employed to investigate the physiological and performance variables of interest. All these cycle tests were performed on an electronically-braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) which can administer work rate in various functions. The ergometer functions that were used in the series of experiments that comprise this thesis include the step, proportional and linear work rate forcing functions. The step function allows work rate to be increased or decreased, rapidly (1,000 $W \cdot s^{-1}$), from one constant work rate to another in a stepwise manner for a predetermined duration. This work rate forcing function was employed during all the step exercise tests of various exercise intensities. The proportional function allows work rate to increase linearly as a function of time and this work rate forcing function was employed during the ramp incremental exercise tests. Both the step and proportional work rate functions administer the external power output independent of pedal cadence by instantaneously adjusting flywheel resistance via electrical braking. The linear work rate function, on the other hand, is a cadence dependent method of work rate imposition and is given by the following equation:

$$\text{Linear factor} = \text{Power output} \div \text{Cadence}^2 \qquad \textit{Equation 8}$$

In this mode the ergometer imposes a fixed work rate such that the attainment of a particular cadence will elicit a known power output. This work rate forcing function was employed during the all-out sprint exercise tests described in Chapter 4-6. The

ergometer was calibrated regularly by a laboratory technician in accordance with the manufacturer's guidelines.

Computrainer cycle ergometry

In chapters 5 and 6, a self-paced test (SPT) was performed on a Computrainer cycle ergometry system (RacerMate Computrainer, Seattle, Washington, USA), which provides reliable and valid measurements of power output during self-paced cycling (Davison et al., 2009). During the SPT, a computer screen displaying the Computrainer's software program was placed in front of the subject. This allowed the subject to watch a computer-projected simulation of themselves and the distance they had covered as they cycled.

Single-legged knee-extension ergometer

The exercise tests described in Chapter 7-8 were conducted in the prone position, with subjects secured to the ergometer bed via Velcro straps at the thigh, buttocks, lower back and middle back to minimise extraneous movement. The ergometer consisted of a nylon frame secured on top of the bed close to the subject's feet and a base unit placed at the distal end of the bed. The subject's right foot was connected to a rope running along the top of the frame to the base unit, on which a mounted pulley system permitted brass weight plates to be lifted and lowered. Exercise was performed at the rate of 40 contractions·min^{-1}, with the subject lifting and lowering the weight over a distance of ~0.22 m in accordance with a visual cue presented on a monitor and an audible cue timed to the bottom of the down stroke. A shaft encoder (type BDK-06, Baumer Electrics, Swindon, UK) was fitted within the pulley system to record the distance travelled by the load, alongside a non-magnetic load cell (type F250, Novatech

Measurements, St Leonards-On-Sea, UK) to record applied forces, which were then used to calculated the work rate.

Pulmonary Gas Exchange

In Chapters 4-6, pulmonary gas exchange and ventilation were measured breath-by-breath during all laboratory exercise tests. This analysis was performed using a metabolic cart system that comprised of a bidirectional "TripleV" digital transducer and differential paramagnetic (O_2) and infrared absorption (CO_2) analysers (Jaeger Oxycon Pro, Hoechberg, Germany). Irrespective of the gas analysis system utilised, the gas analysers were calibrated before each test with gases of known concentration and the volume sensor was calibrated using a 3-liter syringe (Hans Rudolph, Kansas City, MO). During all tests subjects wore a nose clip and breathed through a low-dead-space, low-resistance mouthpiece that was connected securely to the transducer. Gas was sampled continuously via a capillary line and $\dot{V}_{O_2}$, carbon dioxide output ($\dot{V}_{CO_2}$) and minute ventilation ($\dot{V}_E$) were displayed breath-by-breath on-line following correction of the delay between the volume and concentration signals for each breath. Following the completion of each test, raw breath-by-breath gas exchange and ventilation data were exported for later analysis.

Heart Rate

During all exercise tests except for those conducted within the magnetic resonance scanner, heart rate (HR) was measured every 5-s average using short-range radiotelemetry (Polar S610, Polar Electro Oy, Kempele, Finland). After all tests, raw HR data were exported for later analysis.

Electromyography

During the exercise tests conducted in Chapters 4-6, the surface electromyography (EMG) of the *m. vastus lateralis* of the right leg, was measured to assess the gross neuromuscular activity and infer muscle activation during exercise. For these measurements, the leg was initially shaved and cleaned with alcohol around the belly of the muscle and graphite snap electrodes (Unilect 40713, Unomedical, Stonehouse, Great Britain) were adhered to the prepared area in a bipolar arrangement (interelectrode distance, 40 mm) with ground electrodes positioned on nearby tissue. Elastic bandages were used to secure electrodes and wires in place and pen marks were made around electrodes to enable precise placement reproduction on subsequent tests. The EMG signal activity was recorded using a ME3000PB Muscle Tester (Mega Electronics Ltd., Kuopio, Finland) at a sampling frequency of 1,000 Hz. The bipolar signal was amplified (amplifier input impedance > 1 MΩ), and data were collected online in raw form and stored on a personal computer using MegaWin software (Mega Electronics). The raw electromyographic data were subsequently exported as an ASCII file and digitally filtered using Labview 8.2 (National Instruments, Newbury, UK). Initially, the signals were filtered with a 20 Hz high-pass, second-order Butterworth filter to remove contamination from movement artifacts. The signal was then rectified and low-pass filtered at a frequency of 500 Hz to produce a linear envelope. $iEMG_{max}$ was defined as the highest 1-s value. Mean iEMG was calculated for 10-s intervals throughout both the baseline and exercise periods and these values were normalized to the $iEMG_{max}$ that preceded the bout. Baseline iEMG was defined as the mean iEMG during the 90-s preceding the onset of exercise and end-exercise iEMG was defined as the mean iEMG during the final 10-s of exercise.

Blood Lactate Concentration

During all exercise tests for each experimental condition in Chapters 4-6, a fingertip blood sample was obtained to determine the whole blood [lactate]. Prior to drawing the initial sample for the exercise bout, the sampling site was cleaned thoroughly with alcohol and a disposable safety lancet (Safety-Lanzette, Sarstedt) was used to puncture the skin. For all samples that were subsequently drawn from this puncture, initial drops of blood were wiped away and ~ 20-25 μL of free-flowing arterialised blood was collected into a heparinised microvette (Microvette CB 300, Sarstedt) and analysed using an automated blood lactate analyser (YSI 1500, Yellow Springs Instruments, Yellow Springs, OH, United States). The analyser was calibrated regularly by a laboratory technician in accordance with the manufacturer's guidelines.

Maximal Voluntary Contraction (MVC)

For the assessment in Chapter 4, subjects sat on an isokinetic dynamometer (Biodex Isokinetic, Biodex Medical Ltd., UK) with their hips, thighs and torso strapped securely to the seat and their right lower leg strapped securely to the movement arm. The seat was positioned such that the subject's hip angle was 100° and the movement arm was set such that their knee angle was fixed at 120° (anatomical joint angle; i.e., full extension = 180°). From this position, subjects performed three maximal isometric knee extension contractions of 5 s duration separated by 60 s recovery. The contraction that elicited the highest peak torque was considered the maximal voluntary contraction (MVC). The $iEMG_{max}$ for each bout was considered as the highest 1-s value attained during the preceding MVC and iEMG data collected during the subsequent cycling bout were normalized to this value.

Exercise Tolerance

In Chapters 4-6 exercise tolerance was assessed by the time to the limit of tolerance (T_{lim}) during cycle exercise. During these tests T_{lim} was defined as the point at which the participant's cadence (a preferred value between 80-90 rev·min^{-1} that was to be held constant during the exercise) dropped by more than 10 rpm despite strong verbal encouragement to continue. Exercise tolerance was determined during one-legged knee- extension exercise in Chapters 7 and 8, T_{lim} was taken as the time at which the participants were no longer able to keep pace with the required contraction frequency (40 repetitions·min^{-1}). Again strong verbal encouragement was provided throughout the test.

^{31}Phosphorous Magnetic Resonance Spectroscopy

Intramuscular metabolic responses to exercise were measured *in vivo* using ^{31}Phosphorous Magnetic Resonance Spectroscopy (^{31}P-MRS) in Chapters 7 and 8. During these tests, subjects were positioned in the prone body position within the bore of a 1.5-T superconducting MR scanner. Prior to and during exercise, data were acquired every 1.5 s, with a spectral width of 1500 Hz, and 1000 data points. Phase cycling with four phase cycles was employed, leading to a spectrum being acquired every 6 s. The subsequent spectra were quantified via peak fitting, assuming prior knowledge, using the jMRUI (version 3) software package employing the AMARES fitting algorithm. Spectra were fitted assuming the presence of the following peaks: P_i, phosphodiester, PCr, α-ATP (two peaks, amplitude ratio 1:1), γ -ATP (two peaks, amplitude ratio 1:1) and β-ATP (three peaks, amplitude ratio 1:2:1). In all cases, relative amplitudes recorded during exercise were corrected for partial saturation by obtaining a baseline spectrum before exercise with long repetition time (TR = 20 s) in which the relative unsaturated peak amplitudes could be determined. Intracellular pH

was calculated using the chemical shift of the P_i spectral peak relative to the PCr peak (Taylor et al., 1983).

Testing Procedures

Information on the testing procedures applied is provided in the specific chapters.

Exercise Tolerance in Intermittent Cycling: Application of the Critical Power Concept

WEERAPONG CHIDNOK[1], FRED J. DIMENNA[1], STEPHEN J. BAILEY[1], ANNI VANHATALO[1], R. HUGH MORTON[2], DARYL P. WILKERSON[1], and ANDREW M. JONES[1]

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ABSTRACT

CHIDNOK, W., F. J. DIMENNA, S. J. BAILEY, A. VANHATALO, R. H. MORTON, D. P. WILKERSON, and A. M. JONES. Exercise Tolerance in Intermittent Cycling: Application of the Critical Power Concept. *Med. Sci. Sports Exerc.*, Vol. 44, No. 5, pp. 966–976, 2012. **Purpose:** This study tested the relevance of the critical power (CP) model for explaining exercise tolerance during intermittent high-intensity exercise with different recovery intensities. **Methods:** After estimation of CP and W' from a 3-min all-out test, seven male subjects completed, in randomized order, a cycle test to exhaustion at a severe-intensity constant-work-rate (S-CWR) and four cycle tests to exhaustion using different intermittent ("work–recovery") protocols (i.e., severe–severe (S–S), severe–heavy (S–H), severe–moderate (S–M), and severe–light (S–L)). **Results:** The tolerable duration of exercise in S-CWR was 384 ± 48 s, and this was increased by 47%, 100%, and 219% for S–H, S–M, and S–L, respectively (all $P < 0.05$). Consistent with this, compared with S-CWR (22.9 ± 7.4 kJ), the work done above the CP was significantly greater by 46%, 98%, and 220% for S–H, S–M, and S–L, respectively (all $P < 0.05$). The slope of the relationship between $\dot{V}O_2$ and time was significantly reduced for S–H, S–M, and S–L (0.09 ± 0.02, 0.09 ± 0.01, and 0.07 ± 0.02 L·min^{-2}, respectively) compared with S-CWR (0.16 ± 0.03 L·min^{-2}, $P < 0.05$). In addition, the slope of the relationship between integrated EMG and time showed a systematic decline for S–H, S–M, and S–L compared with S-CWR ($P < 0.05$). **Conclusions:** These results indicate that, when recovery intervals during intermittent exercise are performed below the CP, exercise tolerance is improved in proportion to the reconstitution of the finite W'. The enhanced exercise tolerance with the lower-intensity recovery intervals was associated with a blunted increase in both $\dot{V}O_2$ and integrated EMG with time.
Key Words: CRITICAL POWER, W', $\dot{V}O_2$ KINETICS, INTERVAL TRAINING, FATIGUE

The physiological responses to constant-work-rate (CWR) exercise are dictated by the intensity domain in which an individual is exercising (16,30,41,42). During moderate-intensity exercise (i.e., exercise performed below the lactate or gas exchange threshold, GET), a steady-state $\dot{V}O_2$ is typically achieved within 2–3 min. During exercise above the GET, however, development of the so-called $\dot{V}O_2$ "slow component" results in the attainment of a delayed and elevated steady state (heavy-intensity exercise) (40,41) or a continued rise in $\dot{V}O_2$ until $\dot{V}O_{2peak}$ is achieved, with the latter portending the limit of tolerance (T_{lim}), that is, severe-intensity exercise (14,30,42). Blood [lactate] (where square brackets denote concentration) responses also discriminate exercise intensity domains: for moderate exercise, blood [lactate] remains close to resting values; for heavy exercise, an elevated but stable blood [lactate] is maintained; and for severe exercise, blood [lactate] rises inexorably until the limit of tolerance is reached (30,42). Finally, there is evidence that Type II fiber recruitment (23) and neuromuscular activity, as reflected by the integrated EMG (iEMG [8,38]), increase with time during severe exercise in concert with the development of the $\dot{V}O_2$ slow component (15).

The asymptote of the hyperbolic relationship between power output (P) and the limit of tolerance during high-intensity exercise (e.g., the so-called "critical power" during cycling; CP) marks the boundary between the heavy and severe domains and, therefore, represents an important physiological threshold that approximates the so-called maximal lactate steady state (31,32). The CP is considered to represent the highest sustainable rate of oxidative metabolism, whereas the curvature constant of the P–T_{lim} hyperbola (W') represents a fixed amount of work that can be performed above CP (19,25,26). The CP concept, therefore, describes an endogenous bioenergetic supply system that can be defined by a two-parameter model, namely, an oxidative component that is rate but not capacity-limited (CP) and a supplementary component that remains constant regardless of the rate at which it is expended (W') (19). Consistently low values of muscle phosphocreatine concentration ([PCr]) and pH

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have been reported at the limit of tolerance during CWR exercise > CP (18,36). Theoretically, once the W' has been exhausted and the limit of tolerance attained, a reduction in work rate to below the CP is required (to enable a reconstitution of the W') if exercise is to be continued. The CP and W' can be estimated using a single all-out protocol where the W' is expended as a function of time until $W' = 0$, at which point the highest power output that can be elicited by maximal effort equals CP. The all-out test power profile is reliable (4), and the derived parameters show close agreement with the estimates from the conventional protocol (34,35) and can be used to accurately predict exercise tolerance (1,34).

Whereas the two-parameter CP model and associated physiological responses have been well described for severe CWR exercise, the profiles during intermittent exercise (i.e., severe "work" intervals separated by intervals of active or passive "recovery") have been much less well examined. This is perhaps surprising given the widespread use of interval training by athletes and its potential applicability in the clinical setting (9,13,24). Morton and Billat (27) adapted the two-parameter CP model for intermittent exercise by including four independent variables (P and t during both work and recovery phases; i.e., P_w, t_w, P_r, and t_r, respectively) and found good fits to data collected during intermittent exercise. This suggests that the CP model is also appropriate for this form of exercise. However, there are conflicting findings as to whether CP and W' estimated from intermittent exercise are similar (and therefore have the same physiological equivalents) to those measured during continuous exercise (3,20,27).

The purpose of this investigation was to apply the CP model to better understand the physiological responses to intermittent exercise. Specifically, we hypothesized that, during exhaustive intermittent exercise in which severe exercise was interspersed with recovery periods in different exercise intensity domains (i.e., severe, heavy, moderate and light (i.e., 20 W)), the total work done above CP, and therefore the limit of tolerance, would be greatest for light, intermediate for moderate, and least for heavy recovery condition, whereas no W' recovery would occur in the severe recovery condition. We also hypothesized that changes in the overall rate of W' depletion during these intermittent exercise protocols would be reflected in changes to the rates at which $\dot{V}O_2$ and iEMG increased to the limit of tolerance.

METHODS

Subjects. Seven male subjects (mean ± SD: age = 26 ± 5 yr, height = 1.79 ± 0.06 m, body mass = 81 ± 6 kg) volunteered and gave written informed consent to participate in this study, which had been approved by the University of Exeter Research Ethics Committee. The subjects were all recreationally active and were familiar with the experimental procedures used in the study. On test days, subjects were instructed to report to the laboratory in a rested state, having completed no strenuous exercise within the previous 24 h and having abstained from food, alcohol, and caffeine for the preceding 3 h. Testing was conducted at the same time of day (±2 h) for each subject, and laboratory visits were separated by at least 48 h.

Experimental overview. All testing was completed at sea level in an air-conditioned laboratory at a temperature of 21°C. The subjects visited the laboratory on eight occasions during a 3-wk period to perform exercise tests on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). On the first visit, subjects completed a ramp incremental exercise test to determine $\dot{V}O_{2peak}$ and GET. During the second visit, subjects completed a familiarization 3-min all-out test, which was not used in data analysis. This test was repeated during the third laboratory visit and was used to estimate end-test power and work done above end-test power, which have been shown to closely approximate CP and W', respectively (34,35). After these preliminary tests, subjects completed five cycling tests to exhaustion: on one occasion, the subjects completed a sustained severe-intensity exercise bout at CWR, and on the other occasions, subjects completed intermittent exercise during which 60 s of severe exercise was interspersed with 30 s of severe, heavy, moderate, or light exercise. These tests were presented to the subjects in randomized order. Before these tests, subjects' maximum iEMG ($iEMG_{max}$) of the right musculus vastus lateralis was determined. Strong verbal encouragement was provided during all of the tests that were required for this study.

Exercise tests. The ramp incremental exercise test consisted of 3 min of pedaling at 0 W, followed by a continuous ramped increase in work rate of 30 W·min^{-1} until the subject was unable to continue. The subjects cycled at a cadence of 80 rpm, and tests were terminated on volitional fatigue or when the required pedal rate could no longer be maintained (i.e., fell by >10 rpm). Peak power output (P_{peak}) was recorded, and $\dot{V}O_2$ data were averaged into 10-s bins. $\dot{V}O_{2peak}$ was defined as the highest 30-s rolling mean value recorded. The GET was determined from 10-s bin serial means from a cluster of measures including 1) the first disproportionate increase in carbon dioxide output ($\dot{V}CO_2$) from visual inspection of individual plots of $\dot{V}CO_2$ versus $\dot{V}O_2$, 2) an increase in $\dot{V}_E/\dot{V}O_2$ ($\dot{V}_E$, expiratory ventilation) with no increase in $\dot{V}_E/\dot{V}CO_2$, and 3) an increase in end-tidal O_2 tension with no fall in end-tidal CO_2 tension (39). The power output corresponding to the metabolic rate at which GET occurred (P_{GET}) was estimated with account taken of the mean response time of the $\dot{V}O_2$ response, which was assumed to approximate 40 s.

The 3-min all-out test began with 3 min of "unloaded" (i.e., 0 W) baseline pedaling at 80 rpm, followed by a 3-min all-out effort (4). Subjects were asked to accelerate their cadence to ~110–120 rpm during the last 5 s of the baseline period. The resistance on the pedals during the 3-min all-out effort was set using the linear mode of the Lode

Excalibur Sport ergometer such that the subject would attain the power output halfway between P_{GET} and P_{peak} (see above) on reaching their preferred cadence (linear factor = power/preferred cadence^2). The preferred cadence was 80 rpm for all subjects in this study. To prevent pacing, subjects were not informed of the elapsed time. To ensure an all-out effort, subjects were instructed to attain their peak power output as quickly as possible from the start of the test and to maintain their cadence as high as possible at all times throughout. The $\dot{V}O_{2peak}$ during the 3-min all-out test was defined as the highest 15-s serial mean value recorded during the bout. The CP was estimated as the mean power output during the final 30 s of the test (EP) and the W' was estimated as the power–time integral above EP using commercially available software (Microsoft Excel, 2007; Microsoft Corp., Redmond, WA).

The power output that would be predicted to lead to exhaustion in 6 min (P_6) was then calculated from the following equation:

$$P_6 = (W'/t_{desired}) + CP \quad [1]$$

where P is the power output, $t_{desired}$ is the desired time-to-exhaustion (i.e., 360 s), CP is the critical power (estimated as EP in the all-out test), and W' is the finite work capacity > CP in joules (estimated as work done above EP).

Subjects returned to the laboratory on five occasions to perform the CWR and intermittent exercise tests (Fig. 1). Subjects cycled at a cadence of 80 rpm during all tests. The severe CWR bout (S-CWR) began with 3 min of 0-W baseline cycling after which work rate was abruptly increased to P_6. Subjects were instructed to continue for as long as possible, and the test was terminated when cadence fell by >10 rpm. For the intermittent exercise bouts, subjects completed four different exercise protocols in which 60 s of exercise at a severe work rate was separated by 30 s of exercise at lower ("recovery") work rates (Fig. 1). In each case, the test began with the same baseline cycling period as S-CWR and was continued until cadence fell by >10 rpm. All intermittent exercise tests included "work" intervals performed at a severe work rate of P_{6-} (i.e., P_6 + 50% Δ_2, where $\Delta_2 = P_6 - CP$). However, the work rates during the "recovery" intervals were varied for the four conditions as subjects cycled at either a lower severe work rate of P_{6-} (i.e., P_6 − 50% Δ_2), a heavy-intensity work rate halfway between P_{GET} and CP (i.e., at P_{GET} + 50% Δ_1, where Δ_1 = the range of work rates between P_{GET} and CP), a moderate-intensity work rate (90% P_{GET}), or a light work rate of 20 W. These four intermittent exercise bouts were designated as S–S, S–H, S–M, and S–L, respectively. Subjects were not informed of the work rates, the elapsed time, the expected time-to-exhaustion, or their performance in any of the tests until the entire study had been completed.

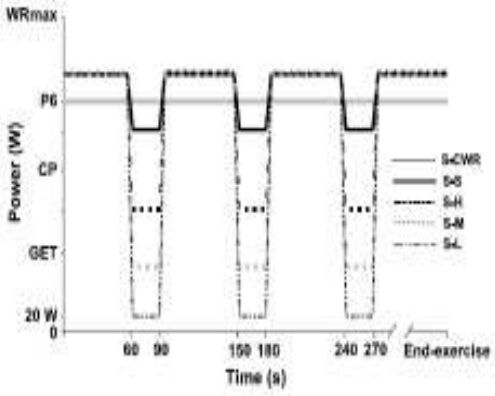

FIGURE 1—Schematic of the experimental protocol. Subjects completed S-CWR exercise bout and four intermittent protocols in which 60 s of severe exercise was interspersed with 30 s of exercise at a lower work rate in different exercise intensity domains (severe, heavy, moderate, and light). See text for further details.

Before the CWR and intermittent exercise tests, the right leg was shaved and cleaned with alcohol around the belly of the musculus vastus lateralis, and graphite snap electrodes (Unilect, Unomedical Ltd., Deeside, UK) were adhered to the prepared area in a bipolar arrangement (interelectrode distance = 40 mm). A ground electrode was positioned on the musculus rectus femoris equidistant from the active electrodes. To secure electrodes and wires in place and to minimize movement during cycling, an elastic bandage was wrapped around the subject's leg. Pen marks were made around the electrodes to enable reproduction of the placement in subsequent tests. For the assessment of maximal voluntary contraction (MVC) and associated neuromuscular activity (EMG), subjects sat on an isokinetic dynamometer (Biodex Isokinetic; Biodex Medical Ltd., Shirley, New York) with their hips, thighs, and torso strapped securely to the seat and their right lower leg strapped securely to the movement arm. The seat was positioned such that the subject's hip angle was 100°, and the movement arm was set such that their knee angle was fixed at 120° (anatomical joint angle; i.e., full extension = 180°). From this position, subjects performed three maximal isometric knee extension contractions of 5 s in duration separated by 60 s of recovery. The contraction that elicited the highest peak torque was considered the MVC. Once this testing was completed, 15 min of rest was allowed before beginning the exhaustive cycling bouts.

Measurements. During all cycling tests, pulmonary gas exchange was measured breath-by-breath with subjects wearing a nose clip and breathing through a low dead space, low-resistance mouthpiece, and an impeller turbine assembly (Jaeger Triple V; Erich Jaeger GmbH, Hoechberg, Germany). The inspired and expired gas volumes and gas concentration signals were continuously sampled at 100 Hz, the latter using paramagnetic (O_2) and infrared (CO_2) analyzers (Jaeger Oxycon Pro; Erich Jaeger GmbH) via a capillary line connected to the mouthpiece. These analyzers were calibrated before each test with gases of known concentration, and the turbine volume transducer was calibrated using a 3-L syringe (Hans Rudolph, Inc., Kansas City, MO). The volume and concentration signals were time aligned by accounting for the delay in capillary gas transit and analyzer rise time

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relative to the volume signal. Oxygen uptake, $\dot{V}CO_2$, and $\dot{V}_E$ were displayed breath-by-breath. HR was measured every 5 s during all tests using short-range radiotelemetry (Polar S610; Polar Electro Oy, Kempele, Finland).

During the CWR and intermittent exercise tests, EMG activity was recorded using a ME3000PB Muscle Tester (Mega Electronics Ltd., Kuopio, Finland) at a sampling frequency of 1000 Hz. The bipolar signal was amplified (amplifier input impedance >1 MΩ), and data were collected online in raw form and stored on a personal computer using MegaWin software (Mega Electronics). The raw EMG data were subsequently exported as an ASCII file and digitally filtered using LabView 8.2 (National Instruments, Newbury, UK). Initially, the signals were filtered with a 20-Hz high-pass, second-order Butterworth filter to remove contamination from movement artifacts. A linear envelope was then produced via rectification (transformation of all signals to absolute values by converting negative values to positive values) and low pass (500 Hz) filtering to smooth high-frequency peaks (e.g., signals in the external environment detected by the electrodes). This process improves the ability to detect relevant aspects of the signal.

During the CWR and intermittent exercise tests, a blood sample from a fingertip was collected into a capillary tube during the last 20 s of baseline cycling and within 20 s of the termination of exercise. These samples were immediately analyzed to determine blood [lactate] (YSI 1500; Yellow Springs Instruments, Yellow Springs, OH).

Data analysis procedures. The total work done ($W = PT_{lim}$) before the limit of tolerance and the average power output during each of the exercise tests were calculated. Subsequently, mathematically equivalent linear transformations of the hyperbolic P–T_{lim} relationship were used to determine two sets of CP and W' estimates according to the following equations (i.e., the W–T_{lim} model and $1/T_{lim}$ model, respectively):

$$W = (CPT_{lim}) + W' \quad [2]$$

$$P = W'(1/T_{lim}) + CP \quad [3]$$

where P indicates the mean power output during the entire trial. These parameters were also calculated using the version of the two-parameter model that has been modified for intermittent exercise (27):

$$t = n(t_w + t_r) + [W' - n\{(P_w - CP)t_w - (CP - P_r)t_r\}]/(P_w - CP) \quad [4]$$

where t is total endurance time, n is the number of completed work-recovery cycles, t_w and t_r are the durations, and P_w and P_r the power outputs of the work and recovery intervals, respectively. P and t over all of the repeated work-recovery cycles were cumulated, and then the CP and W' parameters were derived using nonlinear regression.

To assess the work done above CP ($W = PT_{lim}$; expressed in kilojoules) during the CWR and intermittent exercise bouts, we used the following equations for S-CWR (equation 5a), S–S (equation 5b), and S–H, S–M, and S–L (equation 5c):

$$W_{>CP} = [(P_o T_{lim}) - (CP\,T_{lim})]/1000 \quad [5a]$$

$$W_{>CP} = \left[\left((P_{o+} t_w) + (P_{o-} t_r)\right) - (CP\,T_{lim})\right]/1000 \quad [5b]$$

$$W_{>CP} = [(P_{o+} t_w) - (CP\,t_w)]/1000 \quad [5c]$$

where T_{lim} is the time-to-exhaustion, t_w is the cumulative time spent during intermittent work intervals, and t_r is the cumulative time spent during intermittent recovery intervals.

The breath-by-breath $\dot{V}O_2$ data collected during the CWR and intermittent exercise tests were initially examined to exclude errant breaths caused by coughing, swallowing, sighing, etc., and those values lying >4 SD from the local mean (five-point rolling mean) were removed. The remaining data were subsequently linearly interpolated to provide second-by-second values. $\dot{V}O_{2baseline}$ was defined as the mean $\dot{V}O_2$ measured during the final 90 s of cycling before the onset of exercise, whereas the end-exercise $\dot{V}O_2$ was defined as the mean $\dot{V}O_2$ measured during the final 15 s of exercise. To estimate the total O_2 consumed (L), we used the second-by-second $\dot{V}O_2$ data to calculate the $\dot{V}O_2$–time integral above baseline for each experimental condition up to 180 s, 300 s (the longest time that all subjects were able to reach for all conditions), and the end of exercise. Baseline and end-exercise HR were defined as the mean HR measured during the final 15 s of baseline cycling and the final 15 s of each exercise bout, respectively.

Because of the intermittent nature of the exercise tests used in this study, we did not attempt to formally characterize $\dot{V}O_2$ kinetics via conventional modeling procedures. However, we did obtain a broad characterization of "delayed-onset" response dynamics by calculating the slope of the $\dot{V}O_2$ response using linear regression analysis. The first 60 s of data were deleted to remove the influence of the fundamental response phase and, thereafter, $\dot{V}O_2$ values at 90-s intervals (i.e., the average $\dot{V}O_2$ during the 15 s before completion of each work cycle during intermittent exercise) were determined up to the last completed cycle (and to end-exercise for S-CWR) and fit using the following equation:

$$\dot{V}O_2 = ax + b \quad [6]$$

where x is the time, a is the slope, and b is the y-intercept.

The average iEMG was calculated for 1-s intervals during the MVC and for 10-s intervals throughout both the baseline and exercise periods of the CWR and intermittent exercise tests. The $iEMG_{max}$ in the MVC was considered as the highest 1-s value, and iEMG data collected during the subsequent cycling bout were normalized to this value. The baseline iEMG was defined as the average iEMG during the 90 s preceding the onset of exercise and the end-exercise iEMG

was defined as the average iEMG during the final 10 s of exercise. We then calculated the slope of the iEMG response during exercise using a similar procedure to that already described for calculating the $\dot{V}O_2$ slope (see above). However, iEMG was averaged during the final 10 s of each work interval and the first work interval was not excluded from the fitting window.

Statistical analysis. A one-way repeated-measures ANOVA was used to determine the effects on the relevant physiological and performance variables elicited by the work rate functions for the CWR and intermittent exercise tests and the comparison of $\dot{V}O_2$ peak between all exercise tests. Where the analysis revealed a significant difference, individual paired *t*-tests were used with LSD to determine the origin of such effects. All data are presented as mean ± SD. Statistical significance was accepted when $P < 0.05$.

RESULTS

The $\dot{V}O_{2peak}$ measured during the ramp incremental test was 4.10 ± 0.78 L·min^{-1}. P_{peak} and P_{GET} were 371 ± 62 and 105 ± 15 W, respectively. The CP and W' estimated from the 3-min all-out test were 241 ± 59 W and 21.1 ± 4.7 kJ, respectively. The $\dot{V}O_{2peak}$ measured during the 3-min all-out test was 4.06 ± 0.89 L·min^{-1}, which was not significantly different from the $\dot{V}O_{2peak}$ measured during the ramp incremental test ($P > 0.05$). The CWR exercise bout (i.e., P_6) was completed at 300 ± 60 W, and the work interval for the intermittent exercise bouts (i.e., P_{6+}) was 329 ± 61 W. The work rates applied during the recovery intervals were 270 ± 59, 173 ± 35, 95 ± 13, and 20 ± 0 W for S–S, S–H, S–M, and S–L, respectively. The mean work rates were therefore 309 ± 60, 277 ± 52, 251 ± 44, and 226 ± 41 W for S–S, S–H, S–M, and S–L, respectively. The mean work rates for S–S and S–H were significantly higher than the CP ($P < 0.05$), whereas the mean work rates for S–M and S–L were not significantly different from the CP.

Table 1 presents physiological variables and T_{lim} values during the CWR and intermittent severe cycling bouts. T_{lim} was significantly different in all cases. Specifically, T_{lim} was 384 ± 48 s for S-CWR, was lower by ~15% for S–S, and was higher by ~47% for S–H, by ~100% for S–M, and by ~219% for S–L. The W'_{CP} was not significantly different between S-CWR and S–S (~23 kJ) or from the subjects' W' estimated from the 3-min all-out test ($P > 0.05$). However, the W'_{CP} became progressively greater as the recovery work rate was reduced from S–H to S–M to S–L (Table 1). The end-exercise $\dot{V}O_2$ from the CWR and intermittent exercise tests were not significantly different from one another or from the $\dot{V}O_{2peak}$ values measured during the ramp incremental test or the 3-min all-out test, with the exception that end-exercise $\dot{V}O_2$ was significantly lower for S–L compared with S-CWR (Table 1). The group mean $\dot{V}O_2$ response profiles are depicted in Figure 2.

A significant reduction in the $\dot{V}O_2$ response slope was present for S–H, S–M, and S–L compared with both S-CWR and S–S. There was no significant difference for total O_2 consumed during the first 180 or 300 s of exercise for S–S compared with S-CWR; however, the values for S–H, S–M, and S–L became progressively smaller in both cases (Table 1). A similar pattern was present when O_2 consumed was calculated to end-exercise.

Blood [lactate] and iEMG measurements are also presented in Table 1, and group mean iEMG response profiles are depicted in Figure 3. There was no significant difference in baseline or end-exercise blood [lactate] or baseline iEMG between conditions. End-exercise iEMG was significantly less for S–M compared with S-CWR, S–S, and S–H and for S–L compared with all other conditions; there was no significant difference in end-exercise iEMG between S-CWR, S–S, and S–H. The iEMG slopes showed a progressive decline for S–H, S–M, and S–L (Table 1).

The estimates of CP and W' derived from the work–time model, the 1/time model, and the intermittent model along with the estimates from the 3-min all-out test are provided

TABLE 1. Selected physiological variables and limit of tolerance during CWR and intermittent severe-intensity cycling.

	S-CWR	S–S	S–H	S–M	S–L
Baseline HR (beats·min^{-1})	85 ± 14	87 ± 13	88 ± 9	87 ± 12	85 ± 10
End-exercise HR (beats·min^{-1})	182 ± 10	178 ± 8	181 ± 8	177 ± 9	175 ± 12
Baseline $\dot{V}O_2$ (L·min^{-1})	1.08 ± 0.13	1.06 ± 0.16	1.09 ± 0.12	1.06 ± 0.13	1.09 ± 0.14
End-exercise $\dot{V}O_2$ (L·min^{-1})	4.25 ± 0.86	4.18 ± 0.78	4.10 ± 0.59	4.04 ± 0.60	3.89 ± 0.77*
Slope $\dot{V}O_2$ (L·min^{-2})	0.16 ± 0.03	0.19 ± 0.11	0.09 ± 0.02**	0.09 ± 0.01**	0.07 ± 0.02**
Total $\dot{V}O_2$ over 180 s (L)	9.4 ± 1.8	9.5 ± 1.8	9.1 ± 1.5***	8.9 ± 1.5****	8.4 ± 1.6*****
Total $\dot{V}O_2$ over 300 s (L)	17.6 ± 3.4	17.6 ± 3.5	16.5 ± 2.7**	15.7 ± 2.5****	14.8 ± 2.7*****
Total $\dot{V}O_2$ end (L)	23.9 ± 6.5	19.7 ± 5.9*	33.9 ± 2.3**	46.1 ± 23.9****	71.8 ± 39.8*****
Baseline blood [lactate] (mM)	0.9 ± 0.2	0.9 ± 0.1	0.9 ± 0.2	0.9 ± 0.2	1.1 ± 0.3
End-exercise blood [lactate] (mM)	8.4 ± 1.4	9.2 ± 1.8	9.5 ± 2.3	9.0 ± 1.4	8.9 ± 1.3
Baseline iEMG (% $iEMG_{max}$)	8 ± 4	8 ± 4	8 ± 4	8 ± 4	8 ± 3
End-exercise iEMG (% $iEMG_{max}$)	44 ± 11	40 ± 11	39 ± 8	34 ± 10****	34 ± 10****
Slope iEMG (% $iEMG_{max}$ per minute)	2.1 ± 1.0	1.8 ± 1.4	0.8 ± 0.6*	0.2 ± 0.4*	0.1 ± 0.2****
T_{lim} (s)	384 ± 48	323 ± 29*	557 ± 90**	759 ± 243****	1224 ± 497*****
W'_{CP} (kJ)	22.9 ± 7.4	22.8 ± 5.0	33.4 ± 8.2**	45.3 ± 16.3****	72.3 ± 27.2*****

* Significantly different from S-CWR ($P < 0.05$).
** Significantly different from S-CWR and S–S ($P < 0.05$).
*** Significantly different from S–S ($P < 0.05$).
**** Significantly different from S-CWR, S–S, and S–H ($P < 0.05$).
***** Significantly different from S-CWR, S–S, S–H, and S–M ($P < 0.05$).

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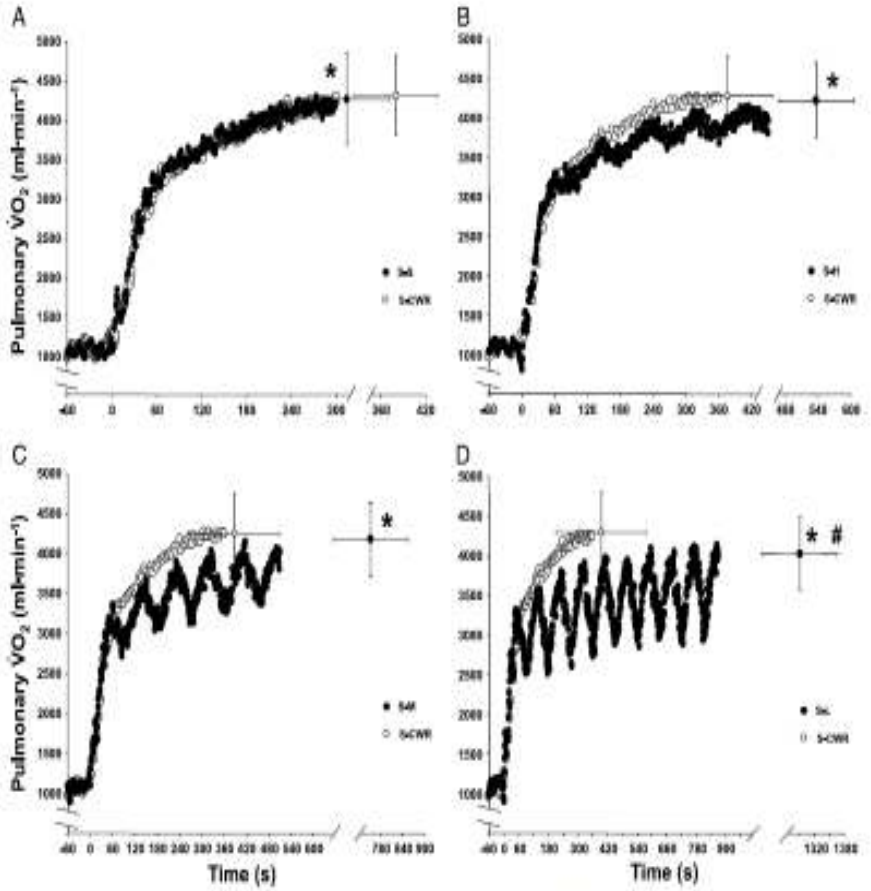

FIGURE 2—Group mean pulmonary $\dot{V}O_2$ response to S-CWR (*open circles*) compared with intermittent exercise (*closed circles*) for S–S (A), S–H (B), S–M (C), and S–L (D). The mean ± SD values at end-exercise are also shown. * T_{lim} is significantly different from S-CWR ($P < 0.05$). #End-exercise $\dot{V}O_2$ is significantly lower than S-CWR ($P < 0.05$).

for each subject in Table 2. The R^2 values ranged from 0.99 to 1.00, from 0.99 to 1.00, and from 0.95 to 1.00 for the work–time model, the 1/time model, and the intermittent model, respectively. There were no significant differences in the CP or W' estimates between the three different procedures. However, the values for CP and W' were significantly different compared to those derived using the 3-min all-out test (Table 2).

DISCUSSION

The principal original finding of this investigation was that the W'_{-CP} and limit of tolerance during intermittent exercise differed significantly when the recovery intervals were positioned in different intensity domains (<CP). During CWR or intermittent exercise performed exclusively in the severe domain, the W'_{-CP} was not significantly different from the subjects' W', as estimated from the 3-min all-out test (34). This confirms and extends the notion that, in this domain, the limit of tolerance is reached when subjects expend the finite work capacity available above CP, i.e., the W' (11). When intermittent exercise involving work bouts in the severe domain and recovery intervals in lower-intensity domains was performed, however, the limit of tolerance (and the W'_{-CP}) was extended, being greatest for S–L, intermediate for S–M, and least for S–H. The slopes describing the increases in $\dot{V}O_2$ and iEMG with time during intermittent exercise were reduced when the recovery intervals were completed at progressively lower intensities. These findings support the recent suggestion that depletion of the W' is linked to the $\dot{V}O_2$ "slow component" that is observed during S-CWR (5,38). It is feasible that both phenomena (i.e., the W' and the $\dot{V}O_2$ slow component) reflect muscle fatigue development (5,28,36,38).

There was no significant difference in W'_{-CP} between S-CWR and S–S (~22.8 kJ) and this value was also not significantly different from the W' as estimated from the 3-min all-out test (4,34,35). This is consistent with previous studies which have reported that W'_{-CP} equals W' at the limit of tolerance during severe exercise (11,14,30,42). The highest $\dot{V}O_2$ attained was not significantly different between S-CWR and S–S and was not significantly different from the $\dot{V}O_{2peak}$ measured during either the ramp incremental test or the 3-min all-out test. The attainment of a similar $\dot{V}O_{2peak}$ with different work–rate impositions (i.e., all-out sprint, ramp incremental, and CWR) within the severe domain is also consistent with previous studies (4,7,11,12,14,42).

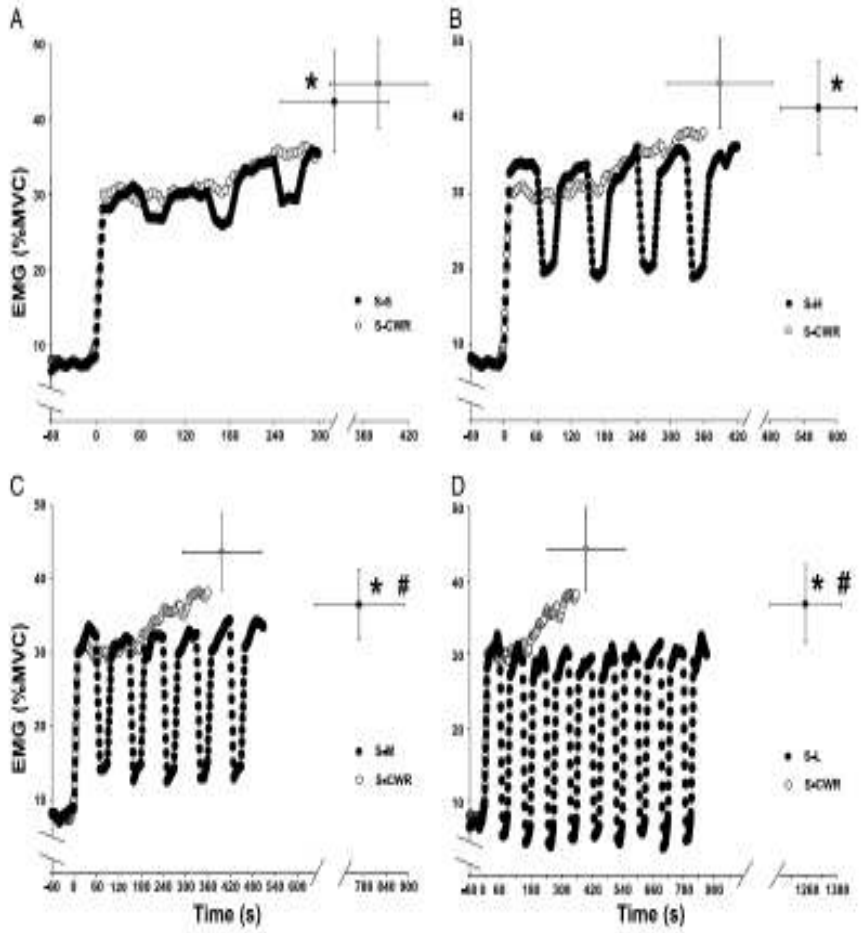

FIGURE 3—Group mean iEMG response to S-CWR (*open circles*) compared with intermittent-work exercise (*closed circles*) for S–S (A), S–H (B), S–M (C), and S–L (D). The mean ± SD values at end-exercise are also shown. * T_{lim} is significantly different from S-CWR ($P < 0.05$). # End-exercise iEMG is significantly lower than S-CWR ($P < 0.05$).

To investigate the influence of the CP and W' on performance during intermittent exercise, we asked subjects to perform intermittent exercise protocols (using a "60-s work, 30-s recovery" duty cycle) until the limit of tolerance. The intensity of the recovery intervals was selected to span the exercise-intensity domains, i.e., S–S, S–H, S–M, and S–L. In the S–S condition, the recovery interval was itself >CP, and thus theoretically, there was no opportunity for W' to be reconstituted between the work bouts (11). Accordingly, the W'_{CP} in S-CWR and S–S was not different, and the limit of tolerance during S–S could be accurately predicted by rearranging equation 1. Relative to S-CWR and S–S, the limit of tolerance became progressively longer and the W'_{CP} became progressively greater when the intensity of the recovery interval was reduced in the S–H, S–M, and S–L conditions. These findings can be explained by considering that some of the W' that is expended during a severe work bout may be reconstituted during a subsequent recovery period provided that the recovery work rate is <CP.

The contention that the W' is expended during work bouts >CP and reconstituted during recovery intervals <CP may be understood with reference to the recent study of

TABLE 2. Parameters of the power–duration relationship derived from the 3-min all-out test, the intermittent CP model, and two conventional two-parameter model equations.

	Critical Power (W)				W' (kJ)				R^2		
Subject	3 min	Intermittent	Work–Time	1/Time	3 min	Intermittent	Work–Time	1/Time	Intermittent	Work–Time	1/Time
1	211	182	188	188	28.0	43.5	39.9	39.9	0.98	0.99	0.99
2	220	195	196	196	25.4	36.5	36.0	36.0	0.99	0.99	0.99
3	213	182	183	183	22.3	37.9	36.8	36.8	1.00	1.00	1.00
4	359	279	281	281	21.5	63.9	62.9	62.9	0.98	0.99	0.99
5	277	224	224	224	18.2	39.9	40.2	40.2	1.00	1.00	1.00
6	187	151	151	151	17.8	33.0	32.9	32.9	0.99	1.00	1.00
7	221	191	195	194	14.3	22.8	20.8	21.7	0.95	0.99	0.99
Mean	241*	200	203	202	21.1*	39.6	38.5	38.6	0.98	0.99	0.99
SD	59	41	41	41	4.7	12.5	12.6	12.4	0.02	0.00	0.00

* Significantly different from other three models ($P < 0.05$).

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Jones et al. (18). Using ^{31}P magnetic resonance spectroscopy, these authors showed that, when CWR exercise was performed slightly above CP, the intramuscular [PCr] and pH continued to decrease, and inorganic phosphate concentration ($[P_i]$) continued to increase, until the limit of tolerance was reached. During exercise performed just below the CP, however, stable values for [PCr], pH, and $[P_i]$ were attained within 3 min of the start of exercise (18). From these results, it may be hypothesized that the recovery intervals during intermittent exercise allow some of the fatigue-related substrates to be resynthesized (e.g., PCr) and for fatigue-related metabolites to be cleared from the muscle (e.g., H^+), thereby delaying the attainment of a "limiting" intramuscular environment (18,36). This suggestion is consistent with the study of Coats et al. (6). These authors asked subjects to perform S-CWR to the limit of tolerance (attained in around 6 min) and then reduced the work rate to 80% GET, 90% CP, or 110% CP. The results indicated that replenishment of the W' following the limit of tolerance necessitated that work rate be reduced <CP (6).

The extent to which the W' was reconstituted in the recovery intervals during intermittent exercise can be calculated using the group mean data, as follows. Given a severe work rate of 329 W and a CP of 241 W, the W'_{CP} in one 60-s bout of severe exercise is (329 − 241) W × 60 = 5.3 kJ. Taking the S–H condition as an example, the subjects completed 6.18 work–recovery cycles on average, during which they accumulated ~ 33.2 kJ of work >CP (6 × 5.3 kJ = 31.7 kJ plus (329 − 241) × 17 s = 1.5 kJ during the final incomplete stage). The W' reconstituted during the six 30-s recovery intervals between severe work bouts can be calculated to be 2.01 kJ (i.e., (total W'_{CP} − W') / the number of recovery cycles completed; (33.2 − 21.1 kJ)/6 = 2.05 kJ). During 6.18 work–recovery cycles (T_{lim}/90 s), this would mean an additional 12.1 kJ of W'_{CP} or a total W'_{CP} (including the initial W' of 21.1 kJ) before the limit of tolerance of approximately 33.2 kJ. Using the same procedures for the S–M condition, the W' reconstituted in each recovery interval is 3.1 kJ, and the total W'_{CP} is ~45 kJ, whereas for the S–L condition, the W' reconstituted in the recovery interval is 4.0 kJ and the total W'_{CP} is ~72 kJ. These hypothesised relationships between W' depletion and reconstitution with different recovery intensities are illustrated in Figure 4. It is clear that the reconstitution of the W' was more complete when the absolute recovery work rate between the severe work bouts was lower, thereby enabling a greater W'_{CP} and extending the limit of tolerance. It may be speculated that lower recovery work rates enabled a more rapid reconstitution of the W' because a larger fraction of the muscle fibers that were metabolically active during the severe work bouts were no longer contributing to power production during the recovery interval.

Interestingly, there was a significant reduction in the slope of the relationship between $\dot{V}O_2$ and time in the conditions where the recovery work rate was <CP (S–H, S–M, and S–L) compared with >CP CWR or intermittent exercise (S-CWR and S–S, respectively). During S-CWR, $\dot{V}O_2$ rises inexorably until $\dot{V}O_{2peak}$ is attained (14,30,42). In this exercise domain, the development of the $\dot{V}O_2$ slow component and the depletion of the W' occur concomitantly (5,15,28,38). It has been proposed that these characteristic features of the physiological response to severe exercise are mechanistically linked and that both are sequelae of muscle fatigue development (5,28,38). Historically, the W' has been considered to represent a finite amount of energy in stored O_2, the high-energy phosphates, and a source related to anaerobic glycolysis that may be expended above the CP (25,26). An alternative perspective, however, is that the W' is related to the accumulation or depletion of one or more metabolites or substrates that are linked to the process of muscle fatigue until some "critical" concentration is attained, beyond which the same work rate cannot be tolerated (6,11,17,18). Consistent with this, it has been shown that the limit of tolerance during a series of S-CWR bouts was associated with the attainment of consistently low values of muscle [PCr] and pH and a consistently high value of muscle $[P_i]$ (36). The present study shows that, compared with S-CWR and S–S, the total O_2 consumed during the first 180 and 300 s of exercise was significantly and progressively reduced in S–H, S–M, and S–L. This blunting of the rate at which $\dot{V}O_2$ increased with time in these conditions resulted in a delayed attainment of $\dot{V}O_{2peak}$ and an extension of the limit of tolerance. The simultaneous blunting of W' expenditure and the $\dot{V}O_2$–time slope (reflecting a loss of oxidative metabolic efficiency) during intermittent exercise is consistent with the proposed mechanistic relationship between the W' and the $\dot{V}O_2$ slow component during S-CWR (5,15,36,38).

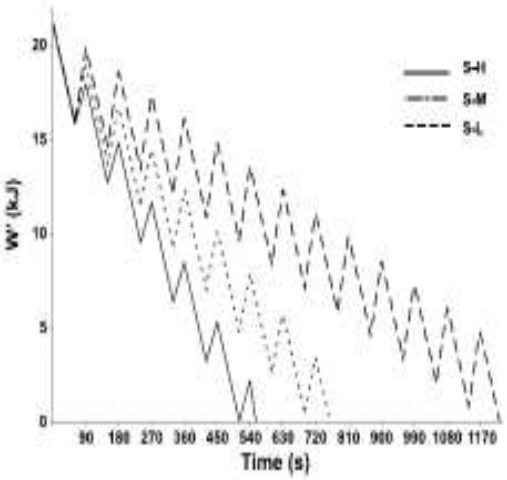

FIGURE 4—Schematic illustration of the depletion of W' during <CP work bouts and the reconstitution of W' during subsequent <CP recovery intervals. Notice that the reconstitution of W' is greater when the recovery intervals are completed at lower intensities, delaying the complete depletion of W' and enabling a longer T_{lim}. For simplicity, the model assumes that W' is reconstituted linearly during the recovery intervals and that the rate of reconstitution is the same irrespective of the magnitude of W' depletion.

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The finding that the $\dot{V}O_2$ response profile can be modulated during intermittent exercise is consistent with the study of Turner et al. (33). These authors examined pulmonary gas exchange and blood lactate dynamics during intermittent exercise protocols in which cycling work intervals at 120% of peak work rate were separated by recovery intervals at 20 W at different duty cycles (10:20 s, 30:60 s, 60:120 s, and 90:180 s). The 10:20-s duty cycle produced a physiological response profile that was akin to that observed during moderate-intensity CWR exercise (i.e., low blood [lactate] and first-order $\dot{V}O_2$ kinetics); the 30:60-s duty cycle produced responses that were similar to those observed during heavy-intensity CWR exercise (elevated but stable blood [lactate] and evidence of a supplementary but low-amplitude $\dot{V}O_2$ slow component), and the 60:120-s and 90:180-s duty cycles resulted in responses that were consistent with S-CWR (high blood [lactate] and a $\dot{V}O_2$ slow component projecting to $\dot{V}O_{2peak}$). Only two of six subjects were able to complete 30 min of exercise in the 60:120-s condition, and none of the subjects could complete 30 min of exercise in the 90:180-s condition. Collectively, the present study and that of Turner et al. (33) indicate that the trajectory of $\dot{V}O_2$ toward $\dot{V}O_{2peak}$ reflects the development of muscular fatigue and is an important determinant of exercise tolerance during intermittent exercise.

Similar to the reduction in the $\dot{V}O_2$–time slopes with decrements in recovery work rate <CP, the iEMG slopes declined in S–H, S–M, and S–L, implying that neuromuscular activity (involving recruitment of additional motor units and/or increased rate coding) was attenuated with time in comparison to S-CWR and S–S. The $\dot{V}O_2$ slow component arises predominantly from within the contracting muscles (21,29) and has been suggested to be related, at least in part, to the recruitment of fast-twitch fibers during high-intensity exercise (2,15). These fibers are considered to be less fatigue resistant and less efficient than their slow-twitch counterparts, and their recruitment would be expected to increase energy turnover and lead to a concomitant and progressive increase in $\dot{V}O_2$ (2,8,22,23,38). Collectively, the results indicate that the recovery intervals between the severe work bouts during intermittent exercise reduce the rate of fatigue development (consistent with the calculable reconstitution of the W') and, in so doing, blunt both the neuromuscular activity required to meet the demands of the exercise task and the associated rise in $\dot{V}O_2$ toward the limit of tolerance.

The end-exercise iEMG was significantly lower for S–M and S–L compared with the other conditions, and the end-exercise $\dot{V}O_2$ was significantly lower for S–L compared with the other conditions. The mean work rates sustained during S–S (309 ± 60 W) and S–H (277 ± 52 W) were significantly higher than the CP estimated from the 3-min all-out test (241 ± 59 W). The mean work rate sustained during S–M (251 ± 44 W), however, was only ~10 W higher than the estimated CP, and the mean work rate sustained during S–L (226 ± 41 W) was ~15 W lower than the estimated CP. That the end-exercise $\dot{V}O_2$ for S–L was lower than the $\dot{V}O_{2peak}$ is consistent with exercise performed in the heavy-intensity domain (19,30,42). Moreover, the lower end-exercise iEMG at work rates ≤CP may suggest exercise intensity domain-specific differences in fiber recruitment or rate coding, which may be linked to differences in fatigue mechanisms <CP compared with >CP (5,18,30).

This is the first study to apply the intermittent CP model of Morton and Billat (27) to cycling exercise. Using the data collected during the CWR and four intermittent exercise protocols, there were no significant differences in either the CP or W' parameter estimates when the work–time model, the 1/time model, and the intermittent model (27) (see Jones et al. (19) for review) were applied. It is important to note, however, that the work–time and 1/time models (equations 2 and 3) provide meaningful parameters only when applied to continuous exercise >CP. In the present study, the total work done and mean power output during intermittent exercise were modeled using these equations to illustrate the goodness of fit and absence of random error in prediction trial data. It is acknowledged that equations 2 and 3 do not account for W' recovery and make the erroneous assumption that P > CP when applied to intermittent exercise. Although equation 4 is an improvement on equations 2 and 3 in that it accounts for linear W' recovery, the precise recovery kinetics of W' during intermittent exercise warrant further study (10).

The CP and W' parameter estimates (equations 2–4) were significantly lower and higher, respectively, compared with the values derived from the 3-min all-out test—which has been shown to provide close estimates of the conventionally established CP and W' parameters during continuous exercise (34,35). These results are similar to those of Morton and Billat (27) who reported that the two-parameter intermittent CP model (equation 4) resulted in a lower CP and a higher W' during intermittent compared with continuous running. This is consistent with the notion that the W' is fixed at the onset of S-CWR but may be reconstituted during intermittent exercise where the recovery intervals are <CP. It is unclear why CP is lower during intermittent compared with continuous exercise. It is possible that this may be related to the apparent reciprocity of CP and W' estimates; for example, interventions such as endurance training and hyperoxia which increase the CP also tend to reduce the W' (35,36). Alternatively, it is possible that CP may be lower during intermittent compared with continuous exercise as a direct consequence of the more substantial nonoxidative contribution to energy turnover during repeated short bouts of high-intensity exercise (19).

In conclusion, the results of this study indicate that physiological responses and exercise tolerance during intermittent exercise can be understood in terms of the CP concept. Recovery intervals between severe work bouts enable the finite W' to be restored, with the magnitude of this reconstitution being related to the intensity and duration of the recovery interval. From a practical perspective, prior knowledge of the CP and W' for intermittent exercise

could be used to predict the limit of tolerance (37) and aid the construction of effective interval training programs (i.e., intensity and duration of the work bouts and recovery intervals) for the enhancement of fitness, functional capacity, or cardiovascular and metabolic health (9,13,24). This study has also shown that the reconstitution of W' during the recovery intervals between repeated bouts of intense exercise results in a reduced rate of change of both iEMG and $\dot{V}O_2$ as exercise proceeds. This indicates that the depletion of the W' and the associated increase in $\dot{V}O_2$ with time are portents of muscle fatigue development and important determinants of performance during intermittent exercise.

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The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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ORIGINAL ARTICLE

$\dot{V}_{O_2max}$ is not altered by self-pacing during incremental exercise

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Abstract We tested the hypothesis that incremental cycling to exhaustion that is paced using clamps of the rating of perceived exertion (RPE) elicits higher $\dot{V}_{O_2max}$ values compared to a conventional ramp incremental protocol when test duration is matched. Seven males completed three incremental tests to exhaustion to measure $\dot{V}_{O_2max}$. The incremental protocols were of similar duration and included: a ramp test at 30 W min^{-1} with constant cadence (RAMP1); a ramp test at 30 W min^{-1} with cadence free to fluctuate according to subject preference (RAMP2); and a self-paced incremental test in which the power output was selected by the subject according to prescribed increments in RPE (SPT). The subjects also completed a $\dot{V}_{O_2max}$ 'verification' test at a fixed high-intensity power output and a 3-min all-out test. No difference was found for $\dot{V}_{O_2max}$ between the incremental protocols (RAMP1 = 4.33 ± 0.60 L min^{-1}; RAMP2 = 4.31 ± 0.62 L min^{-1}; SPT = 4.36 ± 0.59 L min^{-1}; $P > 0.05$) nor between the incremental protocols and the peak $\dot{V}_{O_2}$ measured during the 3-min all-out test (4.33 ± 0.68 L min^{-1}) or the $\dot{V}_{O_2max}$ measured in the verification test (4.32 ± 0.69 L min^{-1}). The integrated electromyogram, blood lactate concentration, heart rate and minute ventilation at exhaustion were not different ($P > 0.05$) between the incremental protocols. In conclusion, when test duration is matched, SPT does not elicit a higher $\dot{V}_{O_2max}$ compared to conventional incremental protocols. The striking similarity of $\dot{V}_{O_2max}$ measured across an array of exercise protocols indicates that there are physiological limits to the attainment of $\dot{V}_{O_2max}$ that cannot be exceeded by self-pacing.

Keywords Maximal oxygen uptake · Critical power · Exercise tolerance · Ramp test · RPE

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Introduction

In 1923, Hill and Lupton studied subjects running at increasing speeds on an outdoor track and observed that oxygen intake rose steadily as speed was increased, attaining a maximum "beyond which no bodily effort can drive it" (Hill and Lupton 1923; Hill et al. 1924). This 'maximal oxygen uptake' ($\dot{V}_{O_2max}$) has since been defined as the highest $\dot{V}_{O_2}$ (i.e., rate of O_2 consumption), averaged over a 15- to 30 s period, which is attainable for a given form of exercise, as evidenced by a failure of $\dot{V}_{O_2}$ to increase further despite an increase in power output

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(Wasserman et al. 1994). Historically, $\dot{V}_{O_2max}$ has been considered to reflect the limits of the human cardio-respiratory system to transport and utilise O_2 and it has, therefore, become a cornerstone of experimental, clinical and applied exercise physiology (Wasserman et al. 1994).

Despite the widespread acceptance of $\dot{V}_{O_2max}$ as an important index of aerobic fitness and functional capacity, it continues to attract considerable conceptual and methodological controversy. Indeed, the very existence of a $\dot{V}_{O_2max}$ has been called into question (Noakes 2008, 2012). The exercise protocol used to measure $\dot{V}_{O_2max}$ has developed from the original discontinuous series of bouts at different constant power outputs (Hill and Lupton 1923; Taylor et al. 1955) to single incremental or ramp exercise tests (Whipp et al. 1981; Davis et al. 1982). These latter tests sometimes include a 'verification phase' in which a power output which is higher than the peak power output attained during the incremental test is subsequently sustained for as long as possible (Rossiter et al. 2006; Midgley et al. 2007). Evidence has been presented that, for a given mode of exercise, $\dot{V}_{O_2max}$ is reproducible across different maximal-effort testing protocols (Davis et al. 1982; Zhang et al. 1991; Takaishi et al. 1992; Bogaard et al. 1996; Burnley et al. 2006).

Intriguingly, Mauger and Sculthorpe (2012) recently reported that self-paced incremental cycling using 'clamps' of ratings of perceived exertion (RPE), i.e., five 2-min stages during which subjects incremented power output according to a prescribed RPE, resulted in the attainment of higher peak $\dot{V}_{O_2}$ compared to a traditional protocol in which power output increments were applied in a strictly linear fashion (40 ± 10 vs. 37 ± 8 ml kg^{-1} min^{-1}, respectively). Mauger and Sculthorpe (2012) suggested that allowing subjects the ability to regulate power output in an anticipatory manner enabled them to reduce the influence of afferent signalling on the perception of discomfort and thereby avoid termination of the test before a 'true' $\dot{V}_{O_2max}$ could be attained. The authors also suggested that this greater level of subject autonomy in selecting power output and pacing the maximal effort might allow for increased or 'more efficient' muscle fibre recruitment. Mauger and Sculthorpe (2012) observed a ~15 % greater peak power output during the self-paced test compared to the conventional incremental test and speculated that the protocol allowed for this higher peak power output (and the increased $\dot{V}_{O_2}$ that it may require) to be achieved at the same level of discomfort.

The highest $\dot{V}_{O_2}$ attained during incremental exercise may be influenced by the total duration of the test (Buchfuhrer et al. 1983; Astorino et al. 2004; Yoon et al. 2007; cf. Midgley et al. 2008). The reason for this discrepancy is unclear, but might be linked to an inability to reach the same absolute power output during longer tests and also to differences in the causes of fatigue (Astorino et al. 2004; Yoon et al. 2007; Jones et al. 2010). Interestingly, in the study by Mauger and Sculthorpe (2012), the mean test duration in the conventional incremental test was ~30 % longer (i.e., ~13 ± 3 min) than the self-paced test (i.e., 10 ± 0 min). It is possible, therefore, that test duration contributed to the higher peak $\dot{V}_{O_2}$ measured in the self-paced test compared to the conventional test (Mauger and Sculthorpe 2012). It should be noted that the importance of incremental test duration for eliciting $\dot{V}_{O_2max}$ has recently been questioned. In a review on the topic, Midgley et al. (2008) suggested that, on the basis of the available evidence, cycle ergometer tests should last between 7 and 26 min to elicit valid $\dot{V}_{O_2max}$ values. Nevertheless, as pointed out recently by Eston (2012), evaluating whether the differences in $\dot{V}_{O_2max}$ reported by Mauger and Sculthorpe (2012) for self-paced and conventional incremental protocols are real rather than artifactual requires a direct comparison where test duration is matched.

The purpose of this study was to investigate possible differences in $\dot{V}_{O_2max}$ measured during self-paced incremental cycling (where intensity is regulated by RPE) and $\dot{V}_{O_2max}$ measured during conventional incremental cycling where the power output increment is externally-controlled and strictly linear. We also included an incremental test in which the power output increment was linear but subjects were free to vary their cadence throughout the test. This provided a condition which had a level of subject autonomy that was more than a conventional ramp incremental test (where cadence is fixed throughout) but less than the self-paced incremental test. Importantly, we designed the study to ensure that the duration of the self-paced incremental test and the duration of the conventional incremental tests were closely matched, and we used EMG to assess possible differences in muscle activation between conditions. In light of the results of Mauger and Sculthorpe (2012), we hypothesised that $\dot{V}_{O_2max}$ would be greater for the self-paced protocol compared to the conventional incremental test protocol.

Methods

Subjects

Seven male subjects (mean ± SD: age 20 ± 1 years, stature 1.74 ± 0.11 m, body mass 75 ± 11 kg) volunteered and gave written informed consent to participate in this study, which had been approved by the University of Exeter Research Ethics Committee. The subjects were recreationally active but not highly trained and were

familiar with the experimental procedures used in the present study. On test days, subjects were instructed to report to the laboratory in a rested state, having completed no strenuous exercise within the previous 24 h, and having abstained from food, alcohol and caffeine for the preceding 3 h. Testing was conducted at the same time of day (±2 h) for each subject and laboratory visits were separated by at least 24 h.

Experimental overview

All testing was completed at sea level in an air-conditioned laboratory at a temperature of 21 °C. The subjects visited the laboratory on eight occasions over a three-week period to perform exercise tests. Subjects completed an initial ramp incremental test to determine $\dot{V}_{O_2max}$ and the gas exchange threshold (GET) on an electronically-braked cycle ergometer (Lode Excalibur Sport, Groningen, the Netherlands) that was regularly calibrated according to the manufacturer's instructions. During the second visit, subjects completed a familiarization 3-min all-out cycle sprint test which was not used in data analysis. In this maximal-effort test, subjects typically attain peak power output in the first 5 s of exercise, after which power output falls with time before stabilizing over the final ~30 s of exercise (Burnley et al. 2006; Vanhatalo et al. 2007). The 3-min all-out test was repeated during the third laboratory visit and used to determine end-test power and work above end-test power, which have been shown to closely approximate the critical power (CP) and work done above CP (W'), respectively (Burnley et al. 2006; Vanhatalo et al. 2007). During the fourth visit, subjects completed a self-paced incremental test that served to familiarise them with one of the experimental conditions that would be tested (see below). The data from this test were not considered for analysis. Following these four preliminary tests, subjects completed each of the three experimental conditions that involved incremental cycling to exhaustion to determine $\dot{V}_{O_2max}$. Two were ramp tests with fixed ramp rates (one with cadence fixed throughout the test and one in which subjects could choose to vary their cadence throughout the test) and the third involved a self-paced protocol. These three tests were completed in a randomised order. In addition, the subjects completed a maximal-intensity constant-power-output test to the limit of tolerance as a $\dot{V}_{O_2max}$ verification test (Midgley et al. 2007).

Initial ramp incremental test

The initial ramp incremental test (T_{ramp0}) consisted of 3 min of cycling against no resistance, followed by a continuous ramped increase in power output of 30 W min^{-1} until the subject was unable to continue. The subjects were instructed to maintain their preferred cadence throughout the test (i.e., cadence was 'fixed'; 80 rpm, $n = 5$; 90 rpm, $n = 2$) and tests were terminated upon volitional exhaustion or when the required cadence could no longer be maintained (i.e., dropped by >10 rpm). Time to exhaustion (s) and peak power output (P_{peak}) were recorded and pulmonary gas exchange and minute ventilation ($\dot{V}_E$) data were averaged into 10 s bins. The $\dot{V}_{O_2max}$ and maximal $\dot{V}_E$ ($\dot{V}_{Emax}$) were defined as the highest 30 s rolling-mean values recorded before termination of the test. Baseline $\dot{V}_{O_2}$ was calculated as the mean $\dot{V}_{O_2}$ measured over the final 90 s of unloaded cycling prior to the onset of the ramp. The GET was determined using standard criteria (Whipp et al. 1981; Wasserman et al. 1994). Baseline and end-exercise heart rate (HR) were defined as the mean HR measured over the final 15 s of baseline cycling and the final 15 s of exercise, respectively.

3-min all-out test

The 3-min all-out test consisted of 3 min of unloaded baseline cycling at each subject's preferred cadence, followed by a 3-min all-out effort. For the latter, subjects were asked to accelerate their cadence to ~110–120 rpm over the last 5 s of the baseline period. The resistance on the pedals during the 3-min all-out effort was set using the linear mode of the Lode Excalibur Sport ergometer such that the subject would attain the power output halfway between P_{GET} and P_{peak} on reaching their preferred cadence (linear factor = power/preferred cadence^2). Strong verbal encouragement was provided throughout the test but, to prevent pacing, the subjects were not informed of the elapsed time. Subjects were instructed to attain their peak power output as quickly as possible from the start of the test and to maintain their cadence as high as possible at all times throughout the 3-min test. The peak $\dot{V}_{O_2}$ during the 3-min all-out test was defined as the highest 30 s mean value recorded during the bout. The end-test power (used to estimate CP) was defined as the mean power output over the final 30 s of the test. The work done above end-test power (an estimate of W') was calculated as the power-time integral above end-test power (Burnley et al. 2006; Vanhatalo et al. 2007).

Experimental incremental tests

Both ramp incremental tests consisted of 3 min of unloaded cycling before the ramped power output protocol was applied. Ramped increases were: (1) 30 W min^{-1} with preferred cadence that was held constant throughout the test (RAMP1); and (2) 30 W min^{-1} with cadence free to vary throughout the test according to the subject's preference (RAMP2). RAMP1 and RAMP2 were performed on

the same ergometer as the initial incremental test (see above). The test termination criteria for these tests was the same as for the initial incremental test, and the baseline and end-exercise HR, baseline $\dot{V}_{O_2}$, $\dot{V}_{O_2max}$, $\dot{V}_{Emax}$ and P_{peak} were all defined in the same manner.

The self-paced incremental test (SPT) was performed on a Computrainer cycle ergometry system (RacerMate Computrainer, Seattle, Washington, USA), which provides reliable and valid measurements of power output during self-paced cycling (Davison et al. 2009). During the SPT, a computer screen displaying the Computrainer's software program was placed in front of the subject. This allowed the subject to watch a computer-projected simulation of himself as he cycled. The protocol consisted of seven x-second stages, where x equals the duration of the initial ramp test divided by seven, i.e., $x = T_{ramp0}/7$, during which subjects could continually vary power output but RPE (using the Borg 6–20 scale) remained clamped (Mauger and Sculthorpe 2012). Specifically, stage-1 RPE was fixed at 8, stage 2 at 10, stage 3 at 12, stage 4 at 14, stage 5 at 16, stage 6 at 18 and stage 7 at 20. Prior to the SPT, the subjects were instructed that they should pace themselves within each stage according to the prescribed RPE, that the goal was to produce a maximal effort within the final stage, and that they should reach volitional exhaustion at the end of the test. During each stage, the subjects were reminded of the RPE at which they should be cycling and the RPE scale was on view throughout the test. Power output was recorded continuously using the Computrainer software. Baseline and end-exercise HR, baseline $\dot{V}_{O_2}$, $\dot{V}_{O_2max}$ and $\dot{V}_{Emax}$ were determined in the same manner as for RAMP1 and RAMP2 (see above).

On a separate day, the subjects completed a maximal-intensity constant-power-output test to the limit of tolerance as a verification of $\dot{V}_{O_2max}$ (Rossiter et al. 2006). The power output used for this verification test was predicted to lead to exhaustion in 180 s as calculated from the following equation:

$$P = (W'/180\ \text{s}) + \text{CP} \quad (1)$$

where P is the power output, CP is the critical power and the W' is the finite work capacity >CP.

This verification bout began with 3 min of unloaded cycling after which the power output was abruptly increased. Subjects were instructed to continue for as long as possible and the test was terminated when cadence fell by more than 10 rpm.

Prior to the exercise tests, the maximum cycling iEMG of the subject's right m. vastus lateralis ($iEMG_{max}$) was assessed. This assessment (performed on the Lode cycle ergometer; see above) began with 3 min of unloaded cycling at each subject's preferred cadence followed by a 5 s all-out sprint with the resistance set using the linear mode of the ergometer. The iEMG data recorded during this 5 s sprint cycling test was processed and analyzed to determine $iEMG_{max}$ (see below). Once this assessment was completed, the subjects were allowed 5 min of rest prior to beginning the incremental test.

Measurements

During all cycling tests, pulmonary gas exchange was measured breath-by-breath with subjects wearing a nose clip and breathing through a low dead space (90 mL), low resistance (0.75 mmHg L^{-1} s^{-1} at 15 L s^{-1}) mouthpiece and impeller turbine assembly (Jaeger Triple V, Hoechberg, Germany). The inspired and expired gas volume and gas concentration signals were continuously sampled at 100 Hz, the latter using paramagnetic (O_2) and infrared (CO_2) analyzers (Jaeger Oxycon Pro, Hoechberg, Germany) via a capillary line connected to the mouthpiece. These analyzers were calibrated before each test with gases of known concentration, and the turbine volume transducer was calibrated using a 3-L syringe (Hans Rudolph, MO). The volume and concentration signals were time aligned by accounting for the delay in capillary gas transit and analyzer rise time relative to the volume signal. $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$ and $\dot{V}_E$ were calculated and displayed breath-by-breath. HR was measured every 5 s during all tests using short-range radiotelemetry (Polar S610, Polar Electro Oy, Kempele, Finland).

Neuromuscular activity of the m. vastus lateralis of the right leg was measured during the 5 s sprint cycling test and subsequent cycling test using bipolar surface EMG. The leg was initially shaved and cleaned with alcohol around the belly of the muscle, and graphite snap electrodes (Unilect, Unomedical Ltd., UK) were adhered to the prepared area in a bipolar arrangement (interelectrode distance: 40 mm). A ground electrode was positioned on the m. rectus femoris equidistant from the active electrodes. The sites of electrode placement (20 cm superior to the lateral tibial head) were chosen according to the recommendations provided in the EMG software (Mega Electronics, Kuopio, Finland). To secure electrodes and wires in place and to minimise movement during cycling, an elastic bandage was wrapped around the subject's leg. Pen marks were made around the electrodes to enable reproduction of the placement in subsequent tests. The EMG signal was recorded at a sampling frequency of 1,000 Hz using a ME3000PB Muscle Tester (Mega Electronics). The bipolar signal was amplified (amplifier input impedance >1 MΩ), and data were collected online in raw form and stored on a personal computer using MegaWin software (Mega Electronics). The raw EMG data were subsequently exported as an ASCII file and digitally filtered using Labview 8.2 (National Instruments, Newbury,

UK). Initially, the signals were filtered with a 20 Hz high-pass, second-order Butterworth filter to remove contamination from movement artifacts after which they were rectified and low-pass filtered at 500 Hz to smooth high-frequency peaks.

A blood sample from a fingertip was collected into a capillary tube over the final 20 s of baseline cycling and as soon as possible (<10 s) following the termination of exercise. These samples were subsequently analyzed to determine the blood lactate concentration (blood [lactate]) at baseline and end-exercise and the change in blood [lactate] during exercise (Δ blood [lactate]) (YSI 1500, Yellow Springs Instruments, Yellow Springs, OH).

Data analysis procedures

Total work done (W_{total}; in J) during RAMP1 and RAMP2 was calculated according to the following equation:

$$W_{total} = \left(P_{peak} \cdot T_{lim} \cdot 0.5\right) \quad (2)$$

where P_{peak} is the peak power (in Watts) achieved on the incremental test, T_{lim} is the time to exhaustion (in s) in the incremental test and 0.5 is the ramp rate (in Watts per s) that was applied during the incremental RAMP1 and RAMP2. $W_{>CP}$ during RAMP1 and RAMP2 was calculated as the power-time integral above CP. W_{total} during SPT was calculated by integrating the area under the power profile dictated by the self-paced second-by-second fluctuations in power output as measured by the Computrainer software (Microsoft Excel, 2007). $W_{>CP}$ during SPT was calculated by adjusting W_{total} to remove all work performed below the CP. The W_{total} and $W_{>CP}$ for the maximal-intensity constant-power-output verification test were calculated as previously described (Chidnok et al. 2012).

During the 5 s sprint cycling test, the iEMG data were averaged for 1 s intervals. The highest of these 1 s mean values was defined as $iEMG_{max}$. During the subsequent exercise tests, the iEMG data were averaged for 10 s intervals and these 10 s mean values were subsequently normalised to $iEMG_{max}$ to provide a temporal profile of iEMG throughout the incremental test. Baseline iEMG was defined as the mean during the 90 s preceding the onset of exercise and end-exercise iEMG was defined as the mean during the final 10 s of exercise.

Statistical analysis

A one-way repeated-measures ANOVA was employed to determine the effects on the relevant physiological and performance variables elicited by the incremental cycling protocols. Where the analysis revealed a significant difference, the origin of such effects was determined via LSD corrected t tests. All data are presented as mean ± SD. Statistical significance was accepted when $P < 0.05$.

Results

Peak power output attained for RAMP1 and RAMP2 were 385 ± 53 W and 385 ± 47 W, respectively. In the SPT, the mean power outputs for each of the stages were 80 ± 36 W for stage 1, 104 ± 41 W for stage 2, 133 ± 34 W for stage 3, 167 ± 21 W for stage 4, 217 ± 19 W for stage 5, 279 ± 46 W for stage 6 and 364 ± 75 W for stage 7. The stage durations in the SPT were 111 ± 15 s. Group mean power output and P_{peak} data are illustrated in Fig. 1. It is important to note that in the SPT, the peak power output was attained in the early part of the final stage of the test when subjects produced a final sprint at an RPE of 20 (Fig. 1). For the 3-min all-out test, peak power was 743 ± 99 W, end-test power was 270 ± 54 W, and work done above end-test power was 15.8 ± 4.1 kJ; the $\dot{V}_{O_2peak}$ was 4.33 ± 0.68 L min^{-1}. For the verification test, subjects sustained exercise for 192 ± 21 s. The $\dot{V}_{O_2max}$ was 4.32 ± 0.69 L min^{-1} and the $W_{>CP}$ was 15.8 ± 5.2 kJ.

Table 1 presents the physiological and performance data for the three incremental protocols and Fig. 2 depicts the group mean $\dot{V}_{O_2}$ response profiles for each condition. There was no significant difference in $\dot{V}_{O_2max}$ across the three incremental protocols (Table 1). Moreover, the values for $\dot{V}_{O_2max}$ measured in the incremental protocols were not significantly different from the $\dot{V}_{O_2max}$ measured in the 3-min all-out test or the constant-power-output verification test (Fig. 2). In the 3-min all-out test, the peak $\dot{V}_{O_2}$ was attained at ~60 s and remained at the peak value until the end of the test despite the continuous fall in power output. In the constant-power-output verification test, $\dot{V}_{O_2}$ rose continuously, reaching a maximum value close to the end of exercise. Baseline $\dot{V}_{O_2}$, baseline and end-exercise HR and $\dot{V}_{Emax}$ were also similar in all tests. There were no significant differences in W_{total} or $W_{>CP}$ across the three incremental protocols. The $W_{>CP}$ across the three incremental protocols was not different from the work done above end-test power in the 3-min all-out test or the $W_{>CP}$ in the constant-power-output verification test ($P > 0.05$).

Blood [lactate] and iEMG results for the three incremental tests are presented in Table 2 and the group mean iEMG response profiles are depicted in Fig. 3. There was no significant difference in baseline, end-exercise or Δ blood [lactate] between conditions. There was also no significant difference in baseline or end-exercise iEMG between the three incremental tests. For the 3-min all-out test, iEMG was 7 ± 3 % at baseline and 39 ± 6 % at end-exercise; however, the highest mean iEMG of 71 ± 8 % was reached during the first 30 s of the test after which iEMG fell, in line with the falling power output. For the

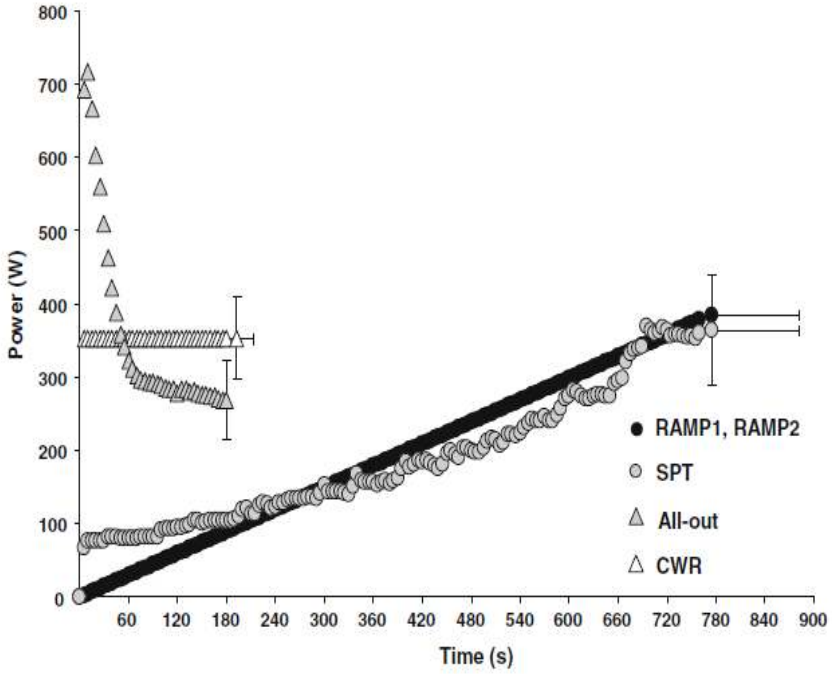

Fig. 1 Group mean power output profiles for RAMP1 and RAMP2 (*black circles*), SPT (*gray circles*), 3-min all-out sprint test (*gray triangles*), and maximal-intensity constant-power-output verification test (*open triangles*). SD *bars* are shown for the end-exercise values

Table 1 Physiological and performance parameters for the three different incremental cycling protocols

	RAMP1	RAMP2	SPT
Baseline HR (b min^{-1})	92 ± 14	92 ± 11	89 ± 16
End-exercise HR (b min^{-1})	180 ± 6	181 ± 6	177 ± 8
Baseline $\dot{V}_{O_2}$ (L min^{-1})	0.87 ± 0.19	0.95 ± 0.20	0.98 ± 0.56
$\dot{V}_{O_2max}$ (L min^{-1})	4.33 ± 0.60	4.31 ± 0.62	4.36 ± 0.59
$\dot{V}_{Emax}$ (L min^{-1})	163 ± 28	164 ± 33	159 ± 35
Time to exhaustion (s)	775 ± 107	773 ± 94	775 ± 107
W_{total} (kJ)	150.6 ± 44.9	150.9 ± 37.9	150.0 ± 32.8
$W_{>CP}$ (kJ)	14.8 ± 9.2	15.0 ± 9.9	13.0 ± 8.4

RAMP1 was a 30 W min^{-1} ramp incremental test in which the subject's preferred cadence was maintained throughout the test; RAMP2 was a 30 W min^{-1} ramp incremental test in which the subject was permitted to vary cadence throughout the test; and SPT was a self-paced incremental test in which the seven work rate increments were regulated according to a prescribed rating of perceived exertion

constant-power-output verification test, iEMG increased with time, reaching 51 ± 13 % at end-exercise (Fig. 3).

Discussion

The principal finding of this investigation was that, contrary to our experimental hypothesis, an incremental cycling protocol that allowed subjects to self pace their power output according to a prescribed RPE did not result in higher $\dot{V}_{O_2max}$ values compared to conventional incremental protocols where the ramp rate was computer-controlled and applied in a strictly linear fashion. Furthermore, $\dot{V}_{O_2max}$ was not different during the conventional tests in which cycling cadence was held constant (at the subject's initial preferred cadence) or allowed to fluctuate during the test according to subject preference. The relative constancy of the highest $\dot{V}_{O_2}$ achieved across the various protocols studied herein (i.e., RAMP1 = 4.33 ± 0.60 L min^{-1}; RAMP2 = 4.31 ± 0.62 L min^{-1}; SPT = 4.36 ± 0.59 L min^{-1}; 3-min all-out test = 4.33 ± 0.68 L min^{-1}; and constant-power-output verification test = 4.32 ± 0.69 L min^{-1}) supports the long-standing interpretation that, for a specific mode of exercise, there is a reproducible $\dot{V}_{O_2}$ that cannot be exceeded (i.e., a $\dot{V}_{O_2max}$). The physiological determinant(s) of $\dot{V}_{O_2max}$ during large muscle group exercise are debated, but are widely considered to be related to limitations in convective and diffusive O_2 transport to muscle (Bassett and Howley 2000; Wagner 2000; Gonzalez-Alonso and Calbet 2003; Mortensen et al. 2005; Saltin and Calbet 2006; Mortensen et al. 2008; cf. Noakes and Marino 2009).

The concept of a $\dot{V}_{O_2max}$ was originally formulated based upon the observation that $\dot{V}_{O_2}$ fails to rise despite an increase in power output (i.e., a '$\dot{V}_{O_2}$ plateau' exists) during a discontinuous series of graded exercise bouts (i.e.,

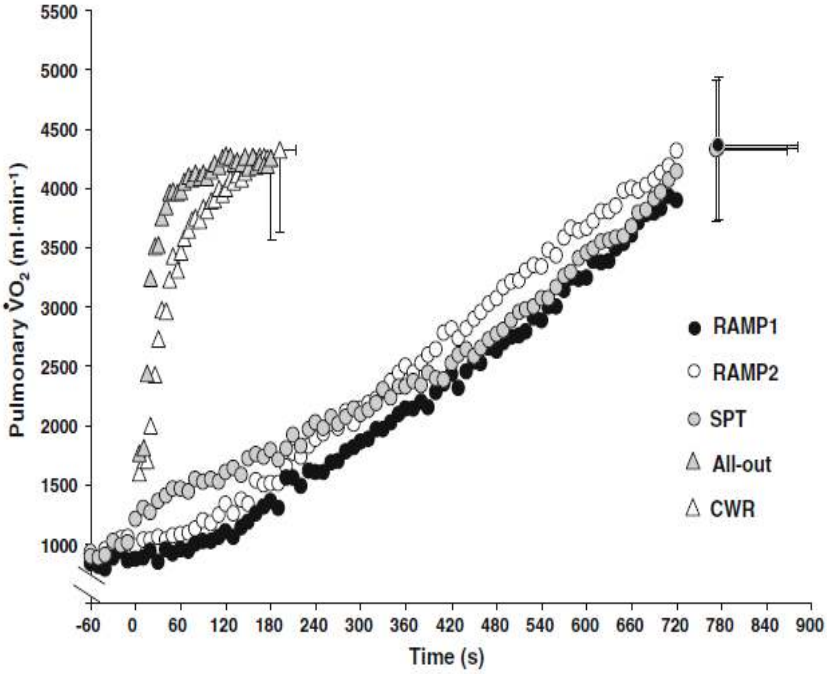

Fig. 2 Group mean pulmonary O_2 uptake response for ramp incremental cycling at 30 W min^{-1} with cadence fixed (RAMP1; *black circles*), ramp incremental cycling at 30 W min^{-1} with cadence free to fluctuate according to subject preference (RAMP2; *open circles*), and an incremental protocol that was self paced according to perceptual regulation (SPT; *gray circles*). Note the striking similarity of $\dot{V}_{O_2max}$ despite varying degrees of subject autonomy. Also shown are the pulmonary O_2 uptake profiles for the 3-min all-out sprint test (*gray triangles*) and the maximal-intensity constant-power-output verification test (*open triangles*). Notice the highly consistent maximum $\dot{V}_{O_2}$ values despite different $\dot{V}_{O_2}$ response profiles across the different protocols. SD *bars* are shown for the end-exercise values

Table 2 iEMG and blood lactate responses for the three different incremental cycling protocols

	RAMP1	RAMP2	SPT
Baseline blood [lactate] (mM)	2.2 ± 1.0	1.8 ± 0.6	1.9 ± 0.5
End-exercise blood [lactate] (mM)	7.6 ± 1.4	7.0 ± 1.7	7.2 ± 2.3
Δ blood [lactate] (mM)	5.4 ± 1.4	5.2 ± 1.4	5.4 ± 2.3
Baseline iEMG (% $iEMG_{max}$)	8 ± 4	9 ± 3	7 ± 7
End-exercise iEMG (% $iEMG_{max}$)	40 ± 9	41 ± 7	43 ± 9

RAMP1 was a 30 W min^{-1} ramp incremental test in which the subject's preferred cadence was maintained throughout the test; RAMP2 was a 30 W min^{-1} ramp incremental test in which the subject was permitted to vary cadence throughout the test; and SPT was a self-paced incremental test in which the seven work rate increments were regulated according to a prescribed rating of perceived exertion

discrete square-wave transitions to constant-power-outputs typically performed on separate days; Hill and Lupton 1923; Taylor et al. 1955; Mitchell et al. 1958). These tests were replaced by continuous-graded protocols (i.e., single exercise tests where power output was increased periodically; e.g., every 1 or 3 min) after it was reported that the peak $\dot{V}_{O_2}$ during such tests was no different than $\dot{V}_{O_2max}$ from the discontinuous protocol (Maksud and Coutts 1971; McArdle et al. 1973). More recently, continuous-load tests involving the increment of power output as a linear function of time (i.e., 'ramp' tests) have become popular because they provide information regarding other important indices of aerobic function such as GET sub-maximal and the $\dot{V}_{O_2}$ power output relationship (Whipp et al. 1981). Importantly, it was shown that these tests produced similar peak $\dot{V}_{O_2}$ values when compared to continuous-graded protocols (Zhang et al. 1991; Bogaard et al. 1996) and across ramp protocols utilizing different ramp slopes (Davis et al. 1982; Takaishi et al. 1992). However, a common feature of both continuous-graded and ramp incremental protocols is that a substantial proportion of subjects do not exhibit a $\dot{V}_{O_2}$ plateau prior to the termination of exercise (Day et al. 2003; Doherty et al. 2003).

Day et al. (2003) found no discernible $\dot{V}_{O_2}$ plateau in 83 % of subjects performing a ramp incremental cycling test that lead to exhaustion in ~10–15 min. However, the peak $\dot{V}_{O_2}$ on such a test was not different from the $\dot{V}_{O_2max}$ measured during a series of severe-intensity constant-power-output exercise bouts for a sub-group of subjects (Day et al. 2003). The authors concluded that the peak $\dot{V}_{O_2}$ during ramp incremental exercise is likely to closely approximate $\dot{V}_{O_2max}$ despite the fact that a plateau is not an obligatory consequence of this type of testing (Day et al.

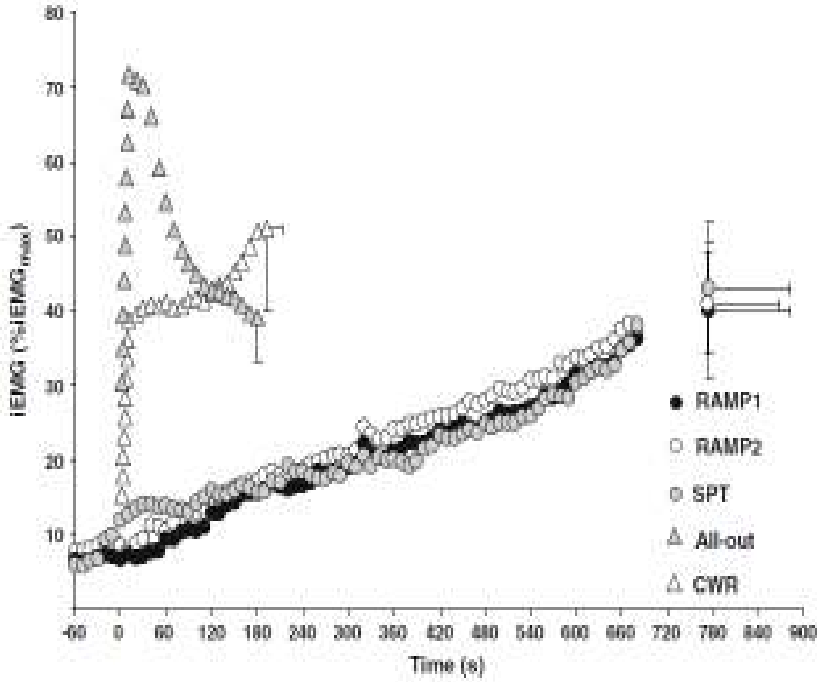

Fig. 3 Group mean iEMG response for RAMP1 (*black circles*), RAMP2 (*open circles*), SPT (*gray circles*), 3-min all-out sprint test (*gray triangles*), and maximal-intensity constant-power-output verification test (*open triangles*). iEMG values are normalised to iEMG$_{max}$ during a 5 s all-out sprint. SD *bars* are shown for the end-exercise values

2003). Conversely, other researchers have used the absence of a universal plateau during incremental exercise to question the existence of a true $\dot{V}_{O_2max}$. For example, Noakes et al. (2001) have suggested that the brain acts subconsciously in an anticipatory (i.e., feed-forward) manner to restrict skeletal muscle fibre recruitment during 'maximal' exercise in order to prevent catastrophic challenges to biological homeostasis. It has been proposed that this 'central governor' would intervene to terminate exhaustive exercise prior to subjects achieving maximal cardio-respiratory capacity, thereby ensuring that the 'true' maximal rate of O_2 transport/utilization (i.e., the traditional notion of a $\dot{V}_{O_2max}$) cannot be achieved (Noakes and Marino 2009).

Mauger and Sculthorpe (2012) reported that an incremental cycling protocol comprising five 2-min stages during which subjects progressively increased power output in a self-paced manner according to clamps of RPE allowed the attainment of higher $\dot{V}_{O_2max}$ values compared to a conventional incremental test protocol. This apparent ability to surpass the '$\dot{V}_{O_2max}$' measured during traditional exercise testing supports the notion that the limitation to $\dot{V}_{O_2max}$ may not be entirely somatic (Noakes 2008; Noakes and Marino 2009; Beltrami et al. 2012). That is, if the highest $\dot{V}_{O_2}$ achieved during the conventional incremental test truly reflects the upper limit of cardio-respiratory function (Bassett and Howley 2000; Wagner 2000; Gonzalez-Alonso and Calbet 2003; Hawkins et al. 2007), it is difficult to reconcile how this could be surpassed by incorporating a different incremental test paradigm. Conversely, if the brain plays a significant role (e.g., by modulating patterns of central motor command in order to optimise performance; Noakes 2008), such regulation could conceivably be circumvented by changing the subject's perception of the challenge at hand. Mauger and Sculthorpe (2012) suggested that the novel protocol they employed achieved this objective by providing a closed-loop format that enabled subjects to pace themselves according to a known exercise end-point. The authors also suggested that their protocol provided subjects with the ability to vary their power output for a given RPE, enabling better management of afferent signalers (which may include the accumulation of fatigue-related muscle metabolites, and changes in motor unit recruitment, core temperature and pulmonary ventilation) and thereby reducing the perception of pain and discomfort. However, a potential confounding factor in that study was test duration, which was different between the self-paced test (10 min) and the conventional incremental test (~13 min). It is possible that the duration of an incremental test can influence the measured $\dot{V}_{O_2max}$ (Buchfuhrer et al. 1983; Astorino et al. 2004; Yoon et al. 2007; cf. Midgley et al. 2008). With this in mind, we investigated possible differences in $\dot{V}_{O_2max}$ between self-paced and conventional incremental protocols when the total test duration was closely matched.

Contrary to our hypothesis, the self-paced protocol did not result in the achievement of higher $\dot{V}_{O_2max}$ values

compared to conventional ramp incremental protocols. We believe this underscores the need to pay close attention to test duration when designing $\dot{V}_{O_2max}$ protocols and interpreting resultant data. At exhaustion, cardiac output, stroke volume and O_2 pulse are lower during long compared to short incremental tests, which may be related in part to a greater rise in core temperature that mandates enhanced peripheral vasodilatation and reduced venous return (McCole et al. 2001; Astorino et al. 2004). This means that the lower $\dot{V}_{O_2max}$ observed during longer incremental tests might reflect a tightening of the already-existing central circulatory restriction (Gonzalez-Alonso and Calbet 2003). This effect might, in part, explain the findings of Mauger and Sculthorpe (2012), i.e., a lower peak $\dot{V}_{O_2}$ for the 13-min conventional incremental protocol compared to the 10-min self-paced protocol despite the prevalence of a $\dot{V}_{O_2}$ plateau in most of the conventional tests.

In the present study, we sampled surface EMG from the m. vastus lateralis and found that at end-exercise, iEMG was ~40–43 % of $iEMG_{max}$ for RAMP1, RAMP2 and SPT. It should be noted, however, that this figure represents the mean of a 10 s sample as a proportion of the highest 1 s value recorded during an initial 5 s sprint cycling bout. This is similar to what has been reported previously for similar protocols (Scheuermann et al. 2002). While the use of iEMG to infer motor unit activation (i.e., recruitment and/or rate coding) is complex, these results suggest that increasing subject autonomy, either by allowing cadence to vary for the same increment in power output (RAMP2) or by allowing subjects to control the power output increment according to RPE (SPT), had little effect on central motor command during incremental exercise tests of the same duration. Importantly, however, completing an all-out 3-min sprint test, which presumably results in near-maximal motor unit recruitment from the onset of exercise (mean iEMG over the first 30 s of exercise was ~71 % of $iEMG_{max}$), does not result in a peak $\dot{V}_{O_2}$ that is higher than $\dot{V}_{O_2max}$ (present study; Burnley et al. 2006; Vanhatalo et al. 2007). Moreover, the end-exercise iEMG in the constant-power-output verification test tended to be higher than the end-exercise iEMG in the incremental tests (~51 vs. ~40–43 %) but $\dot{V}_{O_2max}$ was not significantly different. This suggests that although muscle activation might not be 'maximal' at the termination of incremental cycle exercise, a 'true' $\dot{V}_{O_2max}$ is still attained. Overall, the present study indicates that the same $\dot{V}_{O_2max}$ is attained for a variety of high-intensity cycle exercise protocols (all-out, constant-power-output, and different types of incremental exercise), irrespective of differences in motor unit activation profiles and the degree of central motor drive, provided that the subject exercises to volitional exhaustion.

The CP model of human bioenergetics proposes that two endogenous energy supply components (CP and W') interact to dictate the limit of tolerance during high-intensity exercise (Monod and Scherrer 1965; Jones et al. 2010). According to this model, if an individual exercises at a power output that is less than CP, energetic demands can be met principally by aerobic means such that exercise can continue for a considerable period of time. When power output exceeds CP, however, the rate of aerobic energy supply is insufficient to meet demand and the resultant shortfall must be satisfied using the capacity-limited W'. In the present study, we calculated the work that subjects had performed above their CP ($W_{>CP}$) at the point of exhaustion during the incremental tests. It is known that for constant-power-output exercise >CP, $\dot{V}_{O_2max}$ is attained at or just before the limit of tolerance (Poole et al. 1988). We have suggested that the depletion of the W' and the development of the $\dot{V}_{O_2}$ 'slow component' that sets $\dot{V}_{O_2}$ on a trajectory to $\dot{V}_{O_2max}$ during >CP exercise may be related (Burnley and Jones 2007; Vanhatalo et al. 2010). Therefore, in the present study, we were interested in whether possible differences in the $W_{>CP}$ might explain possible differences in $\dot{V}_{O_2max}$ between the incremental protocols we investigated. The results indicate that neither the $\dot{V}_{O_2max}$ nor the $W_{>CP}$ (~13–15 kJ) were significantly different across the three incremental protocols. Moreover, the $W_{>CP}$ values measured in the incremental tests were not different from the work done above end-test power during the 3-min all-out sprint test or the $W_{>CP}$ during the constant-power-output verification test, both of which produced very similar $\dot{V}_{O_2max}$ values. The striking similarity of $W_{>CP}$ across ramp incremental tests performed at different cadences, and with different levels of subject autonomy with respect to power output increment, is consistent with recent studies which have confirmed the applicability of the CP concept during high-intensity exercise in which power output varies as a function of time (e.g., ramp, intermittent and all-out exercise) (Morton et al. 1997; Burnley et al. 2006; Vanhatalo et al. 2007; Chidnok et al. 2012). Importantly, these results support the notion that the depletion of a finite capacity for $W_{>CP}$ (W') and the achievement of a reproducible $\dot{V}_{O_2max}$ occur consistently when exhaustion ensues across different types of high-intensity (>CP) exercise protocols (Jones et al. 2010). These events appear to coincide with the attainment of some critical level of high-energy phosphate depletion and/or metabolite accumulation (Poole et al. 1988; Jones et al. 2008; Vanhatalo et al. 2010). Interestingly, there is evidence that exercise above the intensity corresponding to the CP is associated with increasing recruitment of highly glycolytic muscle fibres in the rat (Copp et al. 2010) and may coincide with a blunted increase in O_2 delivery to the

leg muscles during cycle exercise in humans (Mortensen et al. 2005).

In conclusion, the results of the present study indicate that incremental cycling to exhaustion is characterised by a similar $\dot{V}_{O_2max}$ regardless of whether the power output is applied linearly by the experimenter or regulated by the subject's own perception of effort. The highest $\dot{V}_{O_2}$ values measured across an array of protocols (i.e., a continuous-graded protocol that was self-paced according to a prescribed RPE; a ramp incremental protocol with cadence allowed to fluctuate according to subject preference; a ramp incremental protocol with fixed cadence; a high-intensity constant-power-output test to the limit of tolerance; and a 3-min all-out test during which power output declined precipitously over time) were strikingly similar (within 1 %). These results appear to be consistent with the traditional interpretation of $\dot{V}_{O_2max}$, i.e., a $\dot{V}_{O_2}$ that cannot be exceeded for a specific mode of exercise due to a limitation of physiological origin (Hill and Lupton 1923; Bassett and Howley 2000; Wagner 2000; Gonzalez-Alonso and Calbet 2003; Saltin and Calbet 2006; Hawkins et al. 2007). Finally, our results are consistent with the CP model of bioenergetics and suggest that $\dot{V}_{O_2max}$ is attained when the W' is fully expended during exhaustive >CP exercise.

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Effects of Pacing Strategy on Work Performed above Critical Power during High-Intensity Exercise

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[1]*Sport and Health Sciences, College of Life and Environmental Sciences, University of Exeter, Exeter, Devon, UNITED KINGDOM;* [2]*Human Performance Laboratory, Health Studies, Physical Education and Human Performance Sciences, Adelphi University, Garden City, NY; and* [3]*Teachers College, Department of Biobehavioral Sciences, Columbia University, New York, NY*

ABSTRACT

CHIDNOK, W., F. J. DIMENNA, S. J. BAILEY, D. P. WILKERSON, A. VANHATALO, and A. M. JONES. Effects of Pacing Strategy on Work Performed above Critical Power during High-Intensity Exercise. *Med. Sci. Sports Exerc.*, Vol. 45, No. 7, pp. 00–00, 2013. **Purpose**: We investigated the influence of pacing strategy on the work completed above critical power (CP) before exhaustion (W'_{CP}) and the peak $\dot{V}O_2$ attained during high-intensity cycling. **Methods**: After the determination of $\dot{V}O_{2max}$ from a ramp incremental cycling (INC) test and the estimation of the parameters of the power–duration relationship for high-intensity exercise (i.e., CP and W') from a 3-min all-out cycling test (AOT), eight male subjects completed a cycle test to exhaustion at a severe-intensity constant work rate (CWR) estimated to result in exhaustion in 3 min and a self-paced 3-min cycling time trial (SPT). **Results**: The $\dot{V}O_{2max}$ determined from INC was 4.24 ± 0.69 L·min^{-1}, and the CP and the W' estimated from AOT were 260 ± 60 W and 16.5 ± 4.0 kJ, respectively. W'_{CP} during SPT was not significantly different from W'_{CP} during CWR (15.3 ± 5.6 and 16.6 ± 7.4 kJ, respectively), and these values were also similar to W'_{CP} during INC (16.4 ± 4.0 kJ) and W' estimated from AOT. The peak $\dot{V}O_2$ during SPT was not significantly different from peak $\dot{V}O_2$ during CWR (4.20 ± 0.77 and 4.14 ± 0.75 L·min^{-1}, respectively), and these values were similar to the $\dot{V}O_{2max}$ determined from INC and the peak $\dot{V}O_2$ during AOT (4.10 ± 0.79 L·min^{-1}). **Conclusion**: Exhaustion during high-intensity exercise coincides with the achievement of the same peak $\dot{V}O_2$ ($\dot{V}O_{2max}$) and the completion of the same W'_{CP}, irrespective of the work rate forcing function (INC or CWR) or pacing strategy (enforced pace or self-paced). These findings indicate that exhaustion during high-intensity exercise is based on highly predictable physiological processes, which are unaffected when pacing strategy is self-selected. **Key Words**: CRITICAL POWER, W', SELF-PACED, CONSTANT WORK RATE, FATIGUE

It is well established that the limit of tolerance during high-intensity constant work rate (CWR) exercise conforms to a hyperbolic work rate/time function (26,27) (for a review, see Jones et al. [16]). For example, during cycle exercise at a power output (P) above what has been termed the "critical power" (CP; i.e., the asymptote of the power/time hyperbola), time to exhaustion (T_e) can be predicted as follows:

$$T_e = W'/(P - CP) \quad [1]$$

where W' represents the hyperbola's curvature constant. This constancy of W' for supra-CP work rates predicts that T_e during high-intensity exercise will coincide with the depletion of a fixed capacity for work above CP (W'_{CP}) (12,16,26). Recent research indicates that the same model applies to exhaustive exercise where work rate is not held constant, for example, "ramp" incremental cycling (INC) where work rate is increased as a linear function of time (e.g., 1 W every 2 s) (28,30). For this type of exercise, T_e is predicted by a modified version of equation 1:

$$T_e = CP/S + \text{sqrt}(2W'/S) \quad [2]$$

where S represents the ramp slope (e.g., 0.5 W·s^{-1}) (28). This indicates that regardless of how an exercise bout is configured, the time spent working above CP (e.g., from exercise onset for CWR and once CP is surpassed during ramp INC) is limited by the same finite energetic reserve. The CP model also predicts T_e during intermittent cycling where bouts of high-intensity exercise are interspersed with recovery intervals at reduced work rates (6).

Unlike the aforementioned testing protocols where structured work rate forcing functions are applied by an investigator (i.e., pace is enforced), athletes in sports competitions have the opportunity for self-pacing, and this is known to

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have important implications for performance (1,2,9,11). Pacing strategies vary according to event duration and an athlete's level of experience but are believed to reflect an attempt to optimize performance without incurring premature or intolerable challenges to homeostasis (35). Interestingly, there is evidence to suggest that self-pacing reduces the metabolic stress associated with a given performance. For example, it has been shown that allowing subjects to self-pace at a fixed RPE enabled them to complete a 5000-m rowing bout in the same amount of time, but with less physiological perturbation, compared with enforced-pace CWR exercise where the same mean power output was maintained (21). Given that CWR exercise above CP is theoretically terminated when the W' is fully used and $\dot{V}O_2$ reaches its maximum value (4,16,33), it may be speculated that self-pacing increases the W' and/or the $\dot{V}O_{2max}$, but this possibility has not been investigated.

In addition to exhaustive high-intensity CWR and INC, all-out sprint exercise results in the attainment of $\dot{V}O_{2max}$ as long as it is maintained for a sufficient period (13). For example, when subjects cycle all out against a fixed resistance for 3 min, the peak $\dot{V}O_2$ (typically attained ~60 s into the test and maintained from that point onwards) is not different from the $\dot{V}O_{2max}$ measured during exhaustive INC or CWR exercise in the severe-intensity domain (3,40). Interestingly, although power output (which is solely a function of cadence within this paradigm) declines precipitously throughout the initial stages of this test, it ultimately reaches a nadir (i.e., end-test power; EP) that is not significantly different from the CP derived from conventional testing procedures (3,37,38). It has also been shown that the total work performed above EP during this 3-min AOT (WEP) approximates W' (37,38). Although it has been reported that the EP and the WEP can be used to accurately predict T_e during CWR (40), it is presently not clear if the same is true for T_e during INC (i.e., via equations 1 and 2). Also, although it has been reported that the CP is significantly correlated with cycling time trial performance (34), the degree to which EP and WEP can together predict performance during self-paced high-intensity exercise is not known.

The primary purpose of this investigation was to determine whether exhaustion during high-intensity exercise coincides with completion of the same $W'_{>CP}$ and attainment of the same peak $\dot{V}O_2$ (i.e., a reproducible W' and $\dot{V}O_{2max}$, respectively) during enforced-pace CWR, self-paced 3-min cycling time trial (SPT) at a similar mean power output, INC, and all-out sprint cycling. We hypothesized that subjects would achieve the same peak $\dot{V}O_2$ and complete the same amount of work in excess of CP at the point at which exercise was terminated irrespective of the test protocol. A secondary purpose was to examine how well parameters derived from a 3-min all-out cycling test (AOT) can predict performance during exhaustive exercise tests using different work rate forcing functions. Specifically, we hypothesized that EP and WEP could be used to accurately predict T_e for CWR and INC and performance during SPT.

METHODS

Subjects. Eight male subjects (mean ± SD; age = 21 ± 4 yr, stature = 1.75 ± 0.11 m, body mass = 76.0 ± 10.0 kg) volunteered and gave written informed consent to participate in this study, which had been approved by the University of Exeter Research Ethics Committee. The subjects were all recreationally active and were familiar with the experimental procedures used in the study. On test days, subjects were instructed to report to the laboratory in a rested state, having completed no strenuous exercise within the previous 24 h and having abstained from food, alcohol, and caffeine for the preceding 3 h. Testing was conducted at the same time of day (±2 h) for each subject and laboratory visits were separated by at least 48 h.

Experimental overview. All testing was completed at sea level in an air-conditioned laboratory at a temperature of 21°C. The subjects visited the laboratory on six occasions for a 3-wk period to perform exercise tests on an electronically braked cycle ergometer (Lode Excalibur Sport; Lode BV, Groningen, the Netherlands) and a Computrainer cycle ergometry system (Racermate Computrainer, Seattle, WA). Both ergometers were calibrated using a Lode Calibrator 2000 (Lode BV). On the first visit, subjects completed a ramp incremental exercise test to determine $\dot{V}O_{2max}$ and gas exchange threshold (GET). During the second visit, subjects completed a familiarization 3-min AOT, which was not used in data analysis. This test was repeated during the third laboratory visit to determine EP and WEP. On the fourth visit, subjects performed a 3-min self-paced familiarization trial, which was not used in data analysis. After these preliminary tests, subjects completed two cycling tests. One test comprised enforced-pace CWR at a power output predicted to result in exhaustion in 3 min according to EP and WEP measured in the 3-min AOT (see equation 3 below). The other test was a 3-min self-paced trial. These tests were presented to subjects on separate days in a counterbalanced order.

Exercise tests. The ramp incremental exercise test (INC) consisted of 3 min of pedaling at 0 W, followed by a continuous ramped increase in work rate of 30 W·min^{-1} until the subject was unable to continue. The subjects were instructed to maintain their preferred cadence (80 rpm, n = 5; 90 rpm, n = 3), and tests were terminated upon volitional exhaustion or when the required pedal rate could no longer be maintained (i.e., fell by >10 rpm). T_e and peak power output (P_{peak}) were recorded, and $\dot{V}O_{2max}$ was defined as the highest 30-s mean value achieved. The GET was determined as the first disproportionate increase in carbon dioxide output ($\dot{V}CO_2$) from visual inspection of individual plots of $\dot{V}CO_2$ versus $\dot{V}O_2$ (41). The power output halfway between P_{GET} and P_{peak} (i.e., 50% Δ) was calculated, taking into account the lag time for $\dot{V}O_2$ relative to power output during incremental exercise, as previously described (38,40).

The 3-min AOT comprised 3 min of "unloaded" (i.e., 0 W) baseline pedaling at the subject's preferred cadence followed by the 3-min all-out effort. The resistance on the

pedals during all-out cycling was set using the linear mode of the Lode Excalibur Sport ergometer such that the subject would attain a power output corresponding to 50% Δ on reaching their preferred cadence (linear factor = power/preferred cadence^2). Strong verbal encouragement was provided throughout the test, but no information was given regarding elapsed time to prevent pacing. Subjects were instructed to accelerate their cadence to approximately 110–120 rpm over the last 5 s of the baseline period and to attain their peak power output as quickly as possible after the start of the test. They were told to maintain their cadence as high as possible at all times from that point onwards.

EP during AOT was defined as the mean power output over the final 30 s of the test. WEP was calculated as the power–time integral above EP. The power output that would be predicted to lead to exhaustion in 3 min (P_3; i.e., the work rate that subjects would maintain during CWR) was then calculated from the algebraically equivalent version of equation 1:

$$P = (W'/T_e) + CP \quad [3]$$

where T_e is the predicted time to exhaustion (i.e., 180 s) and EP and WEP are substituted for CP and W', respectively. We also used EP and WEP from the 3-min AOT in place of CP and W' (38) in equation 2 to predict T_e for the ramp test that the subjects had already completed (S = 0.5 W·s^{-1}). This allowed for a comparison of actual versus predicted T_e to assess the degree to which parameters derived from the 3-min AOT can be used to predict T_e during INC. After returning to the laboratory on the fourth visit for familiarization to self-paced cycling on the Computrainer, subjects completed CWR and SPT in counterbalanced order (four subjects did CWR first and the other four did SPT first) on visits 5 and 6.

CWR (performed on the Lode Excalibur Sport) began with 3 min of 0-W baseline cycling after which work rate was abruptly increased to P_3. Subjects were instructed to continue for as long as possible at the prescribed cadence (see previous paragraphs), and T_e was recorded to the nearest second with exhaustion defined as a fall in cadence >10 rpm. Subjects were not informed of the work rate, elapsed time, or expected time to exhaustion during this test. SPT was performed on the Computrainer, which provides a reliable measure of power output when guidelines regarding temperature and calibration are followed (8). This ergometer was used for SPT because, unlike the Lode where self-pacing is difficult because the linear factor is fixed for the duration of the test, the Computrainer permits the subject to change gears during exercise. Before each test, the Computrainer was calibrated in accordance with manufacturer recommendations. During each test, a computer screen that displayed the Computrainer software program was placed in front of subjects, allowing them to view a computer-projected simulation of themselves and the distance they had cycled. The test began with 3 min of 0-W baseline cycling after which subjects were asked to perform as much work as possible within 3 min using a self-selected pacing strategy. Strong verbal encouragement was provided during CWR and SPT.

Before performing AOT, CWR, and SPT, the maximum iEMG of subjects' right musculus vastus lateralis ($iEMG_{max}$) during cycling was determined. The leg was initially shaved and cleaned with alcohol around the belly of the muscle, and graphite snap electrodes (Unilect; Unomedical Ltd., UK) were adhered to the prepared area in a bipolar arrangement (interelectrode distance: 40 mm). To secure electrodes and wires in place and to minimize movement during cycling, an elastic bandage was wrapped around the subject's leg. Pen marks were made around the electrodes to enable the reproduction of the placement in subsequent tests. For the $iEMG_{max}$ assessment, the test began with 3 min of unloaded baseline pedaling at each subject's preferred cadence, followed by a 5-s all-out sprint with the resistance on the pedals set using the linear mode of the Lode Excalibur Sport ergometer (linear factor = 0.035 W·(r·min^{-1})$^{-2}$). Once this assessment was completed, 5 min of rest was allowed before beginning the subsequent bout. iEMG values recorded during AOT, CWR, and SPT were normalized to the $iEMG_{max}$ that preceded the bout.

Measurements. During all cycling tests, pulmonary gas exchange was measured breath by breath with subjects wearing a nose clip and breathing through a low dead space, low-resistance mouthpiece, and impeller turbine assembly (Jaeger Triple V, Jaeger, Hoechberg, Germany). The inspired and expired gas volume and gas concentration signals were continuously sampled at 100 Hz, the latter using paramagnetic ($\dot{V}O_2$) and infrared ($\dot{V}CO_2$) analyzers (Jaeger Oxycon Pro; Jaeger) via a capillary line connected to the mouthpiece. These analyzers were calibrated before each test with gases of known concentration, and the turbine volume transducer was calibrated using a 3-L syringe (Hans Rudolph, Kansas City, KS). The volume and concentration signals were time aligned by accounting for the delay in capillary gas transit and analyzer rise time relative to the volume signal. $\dot{V}O_2$, $\dot{V}CO_2$, and $_E$ were displayed breath by breath. HR was measured every 5 s during all tests using short-range radiotelemetry (Polar S610; Polar Electro Oy, Kempele, Finland).

The neuromuscular activity of the musculus vastus lateralis of the right leg was measured using bipolar surface electromyography. The electromyographic signal was recorded using an ME3000PB Muscle Tester (Mega Electronics Ltd., Kuopio, Finland) at a sampling frequency of 1000 Hz. The bipolar signal was amplified (amplifier input impedance > 1 MΩ), and data were collected online in raw form and stored on a personal computer using MegaWin software (Mega Electronics). The raw electromyographic data were subsequently exported as an ASCII file and digitally filtered using Labview 8.2 (National Instruments, Newbury, UK). Initially, the signals were filtered with a 20-Hz high-pass, second-order Butterworth filter to remove contamination from movement artifacts. A linear envelope was then produced via rectification and low-pass (500 Hz) filtering to smooth high-frequency

peaks (e.g., signals in the external environment detected by the electrodes).

During all exercise tests, a blood sample from a fingertip was collected into a capillary tube over the final 20 s of baseline cycling and immediately upon termination of exercise. These samples were subsequently analyzed to determine baseline and end-exercise blood [lactate] (YSI 1500; Yellow Springs Instruments, Yellow Springs, OH). Blood lactate accumulation (Δ blood [lactate]) was calculated as the difference between blood [lactate] at end exercise and blood [lactate] at baseline.

Data analysis procedures. To assess the amount of work performed above CP ($W_{>CP}$; expressed in kilojoules) during CWR, we used the following equation:

$$W_{>CP} = [(P_3 \times T_e) - (CP \times T_e)]/1000 \quad [4]$$

where T_e is the measured time to exhaustion. The total work performed in the SPT was calculated as

$$W = (CP \times 180\,s) + W' \quad [5]$$

To assess $W_{>CP}$ during SPT, we calculated the power–time integral during the 3-min self-paced cycling bout and subtracted the work performed below CP (i.e., CP × 180 s). To assess $W_{>CP}$ during INC, we calculated the power–time integral for all of the data before exhaustion and subtracted the work performed below CP (i.e., all work performed before CP was reached).

For CWR, SPT, and AOT, baseline $\dot{V}O_2$ was defined as the mean $\dot{V}O_2$ measured over the final 90 s of cycling before the onset of exercise, whereas peak $\dot{V}O_2$ was defined as the highest 30-s mean value recorded during the test. The time-to-attain $\dot{V}O_{2max}$ in the AOT was determined for each individual as the time required for the 5 s rolling-averaged $\dot{V}O_2$ to rise to a value that was within 1 SD of the $\dot{V}O_{2max}$ (using the criterion established in the ramp incremental test) (40). Baseline and end-exercise HR were defined as the mean HR measured over the final 15 s of baseline cycling and exercise, respectively.

Mean iEMG was calculated for 1-s intervals during the 5-s sprint cycling assessment, and $iEMG_{max}$ was defined as the highest 1-s value. Mean iEMG was calculated for 10-s intervals throughout both the baseline and the exercise periods of AOT, CWR, and SPT, and these values were normalized to the $iEMG_{max}$ that preceded the bout. Baseline iEMG was defined as the mean iEMG during the 90-s preceding the onset of exercise, and end-exercise iEMG was defined as the mean iEMG during the final 10 s of exercise. We also calculated peak iEMG, which was defined as the highest 10-s value recorded during the test. Finally, we calculated mean iEMG for 30-s time bins for each experimental condition (i.e., mean = 1–30, 31–60, 61–90, 91–120, 121–150, and 151–180 s) to assess the extent to which iEMG changed as exercise proceeded for the three different protocols.

Statistical analysis. A one-way repeated-measures ANOVA was used to determine the differences between trials in relevant physiological and performance variables. Where the analysis revealed a significant difference, individual paired *t*-tests were used to determine the origin of such effects. Paired *t*-tests were also used to identify significant differences between iEMG at 30-s and iEMG at 180-s for each condition. Paired *t*-tests and intraclass correlation coefficients were used to examine the relationship between actual and predicted T_e for INC and CWR and actual and predicted performance for SPT. The coefficient of variation (CV) was calculated for each individual as the standard deviation of the predicted and actual measure relative to the mean of the predicted and actual measure and expressed as a percentage. All data are presented as mean ± SD. Statistical significance was accepted when $P < 0.05$.

RESULTS

The $\dot{V}O_{2max}$ for INC was 4.24 ± 0.69 L·min^{-1} with GET occurring at 1.80 ± 0.31 L·min^{-1}. P_{peak} and P_{GET} were 376 ± 57 and 117 ± 38 W, respectively. EP and WEP from the AOT were 260 ± 60 W and 16.5 ± 4.0 kJ, respectively. P_3 (i.e., the work rate for CWR estimated via equation 3) was 345 ± 58 W. The actual T_e for CWR was 185 ± 24 s (range = 145–217 s), which was not significantly different from 180 s (i.e., the predicted value), and the actual and the predicted T_e for CWR were significantly correlated ($r = 0.99$, $P < 0.01$). The CV between predicted and actual T_e in the CWR was 8% ± 5%. The actual T_e for INC (753 ± 121; range = 622–939 s) was not significantly different from the predicted T_e (754 ± 122 s; range = 605–943 s), and these values were also highly correlated ($r = 0.92$, $P < 0.01$; see Fig. 1). The CV between predicted and actual T_e in the INC was 3% ± 3%. The actual total work performed in SPT (63.1 ± 10.6 kJ) was not significantly different from the predicted total work performed (62.6 ± 10.4 kJ), and the actual and predicted values were significantly correlated ($r = 0.94$, $P < 0.01$). The

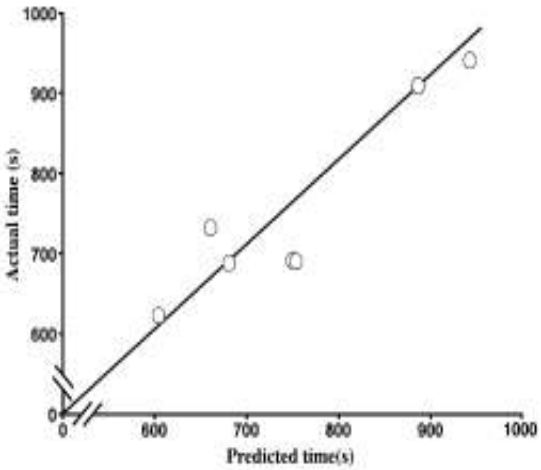

FIGURE 1—Actual versus predicted T_e for INC ($r = 0.92$ for $n = 8$, $P < 0.01$). Prediction was made using parameters derived from a 3-min AOT (i.e., EP and WEP).

CV between predicted and actual word performed in the SPT was 3% ± 3%.

$W'_{>CP}$ values for CWR and SPT (16.6 ± 7.4 and 15.3 ± 5.6 kJ, respectively) were not different from one another or from WEP for AOT (16.5 ± 4.0 kJ, $P > 0.05$). Furthermore, these values were not different ($P > 0.05$) from $W'_{>CP}$ for INC (16.4 ± 4.0 kJ). The CV for $W'_{>CP}$ among conditions was 19% ± 19% between AOT and INC, 17% ± 25% between AOT and CWR, and 12% ± 11% between AOT and SPT. This similarity of the capacity to perform work above CP for four different protocols is depicted for a representative subject in Figure 2. A schematic representation of the different ways in which this capacity was depleted along with the associated power profiles is provided in Figure 3.

Table 1 presents physiological responses to AOT, CWR, and SPT. The peak $\dot{V}O_2$ values for these three protocols were not different from one another or from $\dot{V}O_{2max}$ for INC ($P > 0.05$). The CV for $\dot{V}O_{2max}$ among conditions was 5% ± 4% between INC and AOT, 4% ± 2% between INC and CWR, and 3% ± 1% between INC and SPT. However, the time to achieve the peak $\dot{V}O_2$ was shorter for AOT compared with the other two conditions, which did not differ (Table 1). Group mean $\dot{V}O_2$ response profiles for AOT, CWR, and SPT are shown in Figure 4. There was no significant difference in end-exercise blood (lactate) or end-exercise iEMG between conditions; however, iEMG profiles during the bouts were different. Specifically, peak iEMG was greater for AOT and iEMG decreased by 44% from the initial to the final 30 s of the test. In contrast, iEMG increased from the initial to the final 30 s of CWR and SPT (by 19% and 15%, respectively; Table 1). Group mean iEMG response profiles are shown in Figure 5.

DISCUSSION

The results of this investigation indicate that the total amount of work that can be performed before the limit of tolerance during exercise exceeding the CP (i.e., $W'_{>CP}$) is similar across different high-intensity exercise protocols that expend the W' at different rates. Specifically, in agreement with our first hypothesis, regardless of whether it was produced by enforced-pace exercise (either INC or CWR) or a protocol where subjects were allowed to choose their own pacing strategy (SPT), the termination of exercise coincided with a similar $W'_{>CP}$. This is consistent with the CP model of human bioenergetics, which proposes that the power–duration relationship for high-intensity exercise is defined by two constants: a power asymptote (CP) and a curvature constant (W'), with the latter (expressed in joules) representing the finite capacity for $W'_{>CP}$. The $W'_{>CP}$ across SPT, CWR, and INC was similar to the total amount of work that subjects could complete above EP during the 3-min AOT. Together, the CP and W' estimated using the AOT enabled the prediction of the T_e during CWR with moderate accuracy (CV ~8%) and the prediction of T_e during INC and the total work performed during SPT with good accuracy (CV ~3% for both).

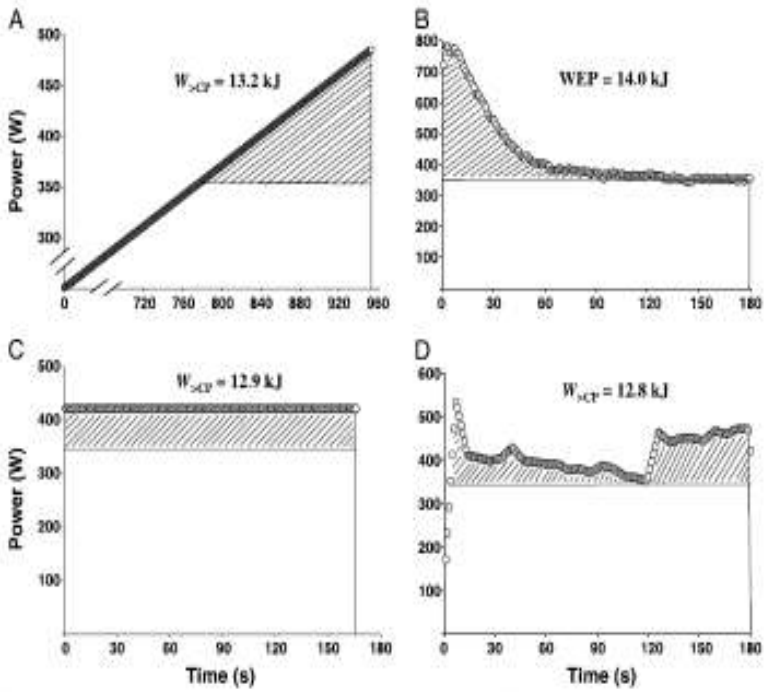

FIGURE 2—Power profile for a representative subject during INC (A), AOT (B), CWR (C), and SPT (D). The horizontal line superimposed on each panel indicates the subject's CP (i.e., as estimated by EP from AOT; e.g., 353 W for this subject), and the striped area represents the work the subject performed above that power output for each protocol. Notice the similarity of this capacity for $W'_{>CP}$ for four distinctly different protocols.

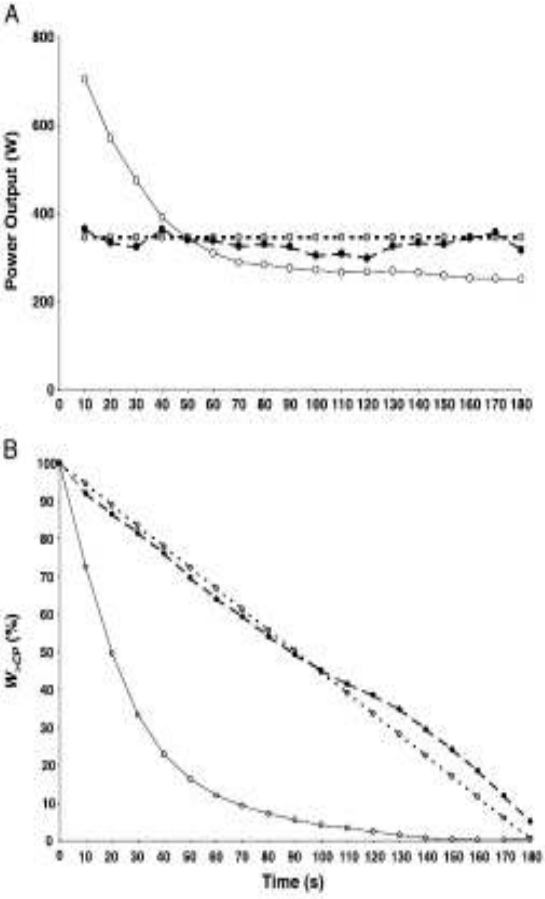

FIGURE 3—A. The power profiles for AOT (open circles), CWR (*closed circles with gray fill*), and SPT (*closed circles with black fill*). B. A schematic representation of depletion of the capacity for $W_{>CP}$ for the same three conditions. These depictions reflect group mean data for work performed at 10-s intervals throughout the exercise bouts. Error bars have been excluded for clarity. Notice how the end of exercise coincides with complete depletion/use of a finite capacity for work regardless of the power profile. See text for further details.

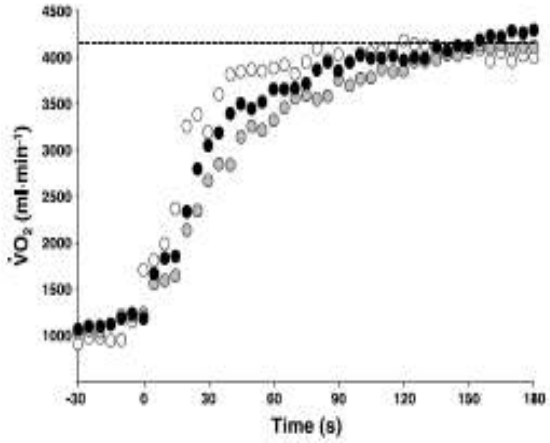

FIGURE 4—Group mean $\dot{V}O_2$ response profiles for AOT (*open circles*), CWR (*closed circles with gray fill*), and SPT (*closed circles with black fill*). Dashed horizontal line indicates $\dot{V}O_{2max}$ from INC. Notice that despite the different protocols that were used, $\dot{V}O_2$ reached a peak value that was not different from $\dot{V}O_{2max}$.

The precise determinants of W' (i.e., the factors that dictate the capacity for $W_{>CP}$) are not yet well understood. According to the classical interpretation, it represents an "anaerobic work capacity"; that is, it is linked to the ability to derive energy from high-energy phosphate hydrolysis and anaerobic glycolysis with a small contribution from myoglobin- and hemoglobin-bound $\dot{V}O_2$ stores (10,25–27). However, this is now understood to be an oversimplification (16). An alternative view is that W' is related to the attainment of some critical level of metabolite accumulation (e.g., $[H^+]$ and/or $[P_i]$ and/or [ADP]) (7,12,17,18,39). Interestingly, CP represents the highest external power output that can be maintained without a continued rise in $\dot{V}O_2$ to the $\dot{V}O_{2max}$ (16,33). Therefore, another feature of CWR and INC performed above CP is that a reproducible $\dot{V}O_{2max}$ should be attained when the W' has been expended.

In the present study, we used the parameters of the power–duration relationship for high-intensity exercise to predict the CWR that subjects could sustain for 3 min (see equation 3) and the total work that could be performed in the SPT. We also predicted in retrospect the amount of time subjects could sustain an INC test at a given ramp slope (i.e., 0.5 W·s^{-1}; see equation 2). However, instead of deriving these parameters in the conventional manner (i.e., constructing the power–duration hyperbola by plotting T_e from multiple CWR bouts performed to exhaustion at different work rates), we estimated them from the data obtained during a single 3-min AOT. We have noted previously that in some individuals, the ability of the AOT parameters to predict T_e during CWR exercise may be less accurate at higher compared with lower severe-intensity work rates (39). This may be partly

TABLE 1. Physiological parameters for 3-min AOT, CWR test with predicted duration of 3 min, and 3-min self-paced time trial (SPT).

	AOT	CWR	SPT
Baseline HR (beats·min^{-1})	95 ± 10	98 ± 7	92 ± 20
End-exercise HR (beats·min^{-1})	174 ± 4	177 ± 8	171 ± 7
Baseline $\dot{V}O_2$ (L·min^{-1})	0.95 ± 0.25	1.06 ± 0.21	1.10 ± 0.81
Peak $\dot{V}O_2$ (L·min^{-1})	4.10 ± 0.79	4.14 ± 0.75	4.20 ± 0.77
Time to peak $\dot{V}O_2$ (s)	59 ± 24*	140 ± 22	136 ± 24
Baseline blood (lactate) (mM)	1.6 ± 0.4	2.1 ± 0.4	1.8 ± 0.8
End-exercise blood (lactate) (mM)	8.7 ± 1.7	7.6 ± 1.8	8.2 ± 1.7
Δ Blood (lactate) (mM)	7.1 ± 1.4	5.4 ± 1.9	6.3 ± 2.2
Baseline iEMG (% $iEMG_{max}$)	8 ± 3	10 ± 5	7 ± 7
End-exercise iEMG (% $iEMG_{max}$)	39 ± 6	48 ± 12	47 ± 6
Peak iEMG (% iEMGmax)	74 ± 11**	51 ± 15	50 ± 6

*Significantly different from CWR and SPT ($P < 0.05$).
†Significantly different from SPT ($P < 0.05$).

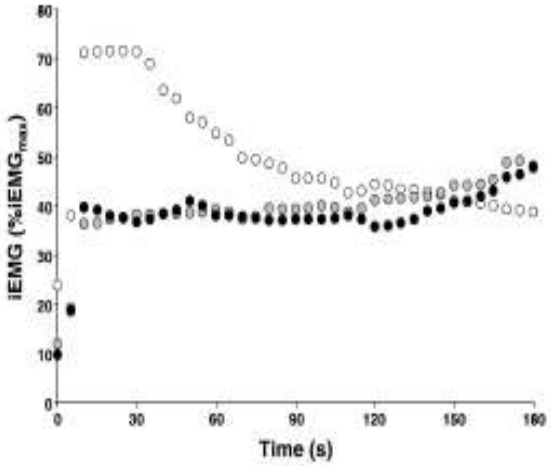

FIGURE 5—Mean iEMG response expressed relative to $iEMG_{max}$ for AOT (*open circles*), CWR (*closed circles with gray fill*), and SPT (*closed circles with black fill*). Notice how iEMG reaches a higher peak but subsequently decreases during all-out exercise compared with the increase in iEMG with time that is observed for the other two protocols. Notice also that the iEMG profile is similar in the CWR (enforced-pace) and SPT (self-paced) tests. Values represent 10-s mean values expressed as a percentage of the highest 1-s iEMG measured during a preceding 5-s sprint.

due to relatively poor test–retest reliability of W' compared with CP whether established using conventional or all-out protocols (3,14,15,36) as well as the greater relative contribution of W' to total work performed in shorter exercise bouts. In the present study, the accuracy of the predicted T_e in CWR, which was performed at approximately 90%Δ, was moderately good (CV = ~8%), whereas the predictions of T_e and performance in INC and SPT were quite precise (CV of ~3%). This largely confirms our second hypothesis and provides further support for the notion that the EP and WEP during 3 min of all-out cycling can be used to estimate CP and W', respectively (3,37,38).

Our ability to use estimates of CP and W' to predict T_e during CWR and INC and performance during SPT is consistent with previous research, which shows that $W_{>CP}$ is similar regardless of the chosen work rate(s) above CP or the forcing function by which the work rate(s) is applied. For example, it has been established that $W_{>CP}$ is similar for CWR exercise, INC exercise, and exercise, where stepwise variations of power output above CP are present (6,12,14,28,29). However, it is important to note that pacing was enforced during these previous investigations; that is, the investigator controlled the external power output(s) that the subject was expected to produce throughout the test. This is unlike athletic competition where the pacing strategy is selected by the subject to achieve the optimal performance (35). This point is relevant when investigating $W_{>CP}$ because it has been suggested that alterations in power output that occur during self-paced exercise are established through central neural control. Specifically, it has been suggested that the brain regulates muscle activation in response to feedback information from peripheral receptors and feedforward anticipation of ongoing events (20,24,31,32). This "central governor" would presumably be related in some way to the cascade of events that pertain at the limit of tolerance (i.e., $\dot{V}O_2$ failing to continue to rise to a higher value and the inability to accumulate further $W_{>CP}$). If true, it is possible that affording subjects greater autonomy over pacing strategy during high-intensity exercise might reduce the sensations of fatigue and allow performance to be enhanced (23). Interestingly, self-pacing during the 5000-m rowing time trials has been shown to result in similar performance as an enforced-pace protocol while producing less homeostatic disturbance (e.g., less blood [lactate] accumulation and temperature elevation) (21).

In the present study, we asked the subjects to cycle as far as possible in 3 min on an ergometer that provided feedback on distance covered. This allowed us to compare $W_{>CP}$ during a self-paced exhaustive 3-min bout of high-intensity cycling (SPT) with $W_{>CP}$ during an enforced-pace approximately 3-min (i.e., 185 ± 24 s) CWR test. Consistent with our first hypothesis, $W_{>CP}$ during SPT was not significantly different from $W_{>CP}$ during CWR. Furthermore, this value was also similar to $W_{>CP}$ during INC and the WEP measured during the AOT. Our study design also allowed us to compare the consistency of the peak $\dot{V}O_2$ across the different exercise protocols. The peak $\dot{V}O_2$ during SPT was not significantly different from the peak $\dot{V}O_2$ during CWR, and this value was also similar to the $\dot{V}O_{2max}$ from INC and the peak $\dot{V}O_2$ measured during AOT. These latter results are consistent with our previous study (5) in which we reported that there was no difference in the peak $\dot{V}O_2$ attained during INC when subjects were allowed to self-pace compared with when the work rate increment was linear and controlled externally. The findings of the present study indicate that the functional link between the depletion of a reproducible capacity for $W_{>CP}$, the attainment of a reproducible $\dot{V}O_2$ peak, and, ultimately, the factors that lead to the termination of exercise during high-intensity exercise is not affected when subjects are allowed to self-pace.

Although the cause(s) of fatigue during supra-CP exercise may ultimately be the same, the pattern of pacing selected in SPT (i.e., greater than mean power output at the beginning and end of exercise) resulted in a different profile of $W_{>CP}$ expenditure compared with CWR, as illustrated in Figure 3. In CWR, the $W_{>CP}$ expenditure was linear throughout exercise, whereas in SPT, it fell more steeply over the first approximately 100 s and then less steeply for most of the remaining approximately 80 s. It is possible that this self-selected pacing strategy allows subjects to feel more comfortable over a greater period of the exercise trial (i.e., more of the total available $W_{>CP}$ is remaining from ~100 to ~170 s), while still permitting a similar $W_{>CP}$ to be accomplished. This may explain the lower RPE but similar performance reported by Lander et al. (21) during a self-paced compared with an enforced-pace 5000 m rowing time trial. A relatively higher power output at the beginning of high-intensity exercise (i.e., a fast-start pacing strategy) also

speeds $\dot{V}O_2$ kinetics (see Fig. 4) and may benefit performance (1,2,19). Interestingly, although the $W'_{>CP}$ was not significantly different between any of the protocols investigated in the present study, it was lowest for SPT (15.3 kJ compared with ~16.5 kJ for the other tests). It is possible that this was a function of the subjects' relative lack of familiarity with the SPT protocol because they completed only one familiarization trial. It is known that pacing strategy can be optimized with additional experience of the exercise task (22,24).

We used iEMG to assess muscle activation during the different protocols in the present study. There are limitations to inferring muscle activation from iEMG during cycling (especially when cadence is not constant; e.g., during AOT). Moreover, the iEMG results in Figure 5 represent the mean of a 10-s sample as a proportion of the highest 1-s value recorded during an initial 5-s sprint cycling bout. Therefore, it is only possible to make qualitative comparisons between the iEMG profiles recorded in the different protocols. End-exercise iEMG was not significantly different for AOT, CWR, and SPT. However, iEMG reached an early peak during AOT, after which it declined, whereas CWR and SPT were characterized by a progressive increase in iEMG until the termination of exercise. The muscle activation profile in AOT would allow $W'_{>CP}$ to be expended rapidly (see Fig. 3B), and power output would fall precipitously to reach its nadir once $W'_{>CP}$ was exhausted (see Fig. 3A). Conversely, during CWR and SPT, $W'_{>CP}$ is expended at a slower rate (Fig. 3B), and muscle activation increases (Fig. 5) to maintain the same external power output in the face of mounting fatigue. Importantly, the similarity of the iEMG profile for CWR and SPT (Fig. 5 and Table 1) suggests that affording subjects the autonomy to pace their exercise efforts does little to change the sequence of events that defines performance capacity during high-intensity exercise (see Figs. 2D, 3, and 4).

In conclusion, the results of this study indicate that the use of a "fixed" capacity for $W'_{>CP}$ (i.e., W') and the associated increase in $\dot{V}O_2$ to a reproducible peak $\dot{V}O_2$ (i.e., $\dot{V}O_{2max}$) are consistently associated with the attainment of the limit of tolerance during high-intensity (>CP) exercise. Specifically, we have shown that high-intensity cycling that involves different profiles of external power production will cause $W'_{>CP}$, iEMG, and $\dot{V}O_2$ to change at different rates; ultimately, however, performance in all these conditions is associated with a "complete" depletion of W', as indicated by similar $W'_{>CP}$ across all exercise trials. Although affording subjects the ability to expend this capacity of their own accord by selecting their preferred pacing strategy may alter the profiles of $W'_{>CP}$ expenditure and $\dot{V}O_2$ kinetics, and perhaps perception of effort, it does not alter either the capacity for $W'_{>CP}$ or the peak $\dot{V}O_2$ that can be attained. Finally, the CP and W' parameters estimated in the 3-min AOT enabled the prediction of time to exhaustion and performance in INC and SPT with high accuracy (CV ~3%) and the prediction of time to exhaustion during CWR test with moderate accuracy (CV ~8%). This test also elicits a peak $\dot{V}O_2$ response that is not different from $\dot{V}O_{2max}$. Consequently, this single test can be used to determine the three physiological parameters (i.e., CP, W', and $\dot{V}O_{2max}$) that together determine high-intensity exercise tolerance.

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The results of this study do not constitute endorsement by the American College of Sports Medicine.

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Muscle metabolic determinants of exercise tolerance following exhaustion: relationship to the "critical power"

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[1]Sport and Health Sciences, College of Life and Environmental Sciences, St. Luke's Campus, University of Exeter, Devon, United Kingdom; [2]Peninsula National Institute for Health Research Clinical Research Facility, St. Luke's Campus, University of Exeter, Devon, United Kingdom; [3]Human Performance Laboratory, Health Studies, Physical Education and Human Performance Sciences, Adelphi University, Garden City, New York; and [4]Teachers College, Department of Biobehavioral Sciences, Columbia University, New York, New York

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Chidnok W, Fulford J, Bailey SJ, DiMenna FJ, Skiba PF, Vanhatalo A, Jones AM. Muscle metabolic determinants of exercise tolerance following exhaustion: relationship to the "critical power". *J Appl Physiol* 115: 000–000, 2013. First published May 2, 2013; doi:10.1152/japplphysiol.00334.2013.—We tested the hypothesis that muscle high-energy phosphate compounds and metabolites related to the fatigue process would be recovered after exhaustion during recovery exercise performed below but not above critical power (CP) and that these changes would influence the capacity to continue exercise. Eight male subjects completed single-leg, knee-extension exercises to exhaustion (for ~180 s) on three occasions, followed by a work-rate reduction to severe-intensity exercise (>CP), heavy-intensity exercise (<CP), or a 10-min passive recovery period, in random order. The muscle metabolic responses to exercise were assessed using ^{31}P magnetic resonance spectroscopy. There was a significant difference between the sustainable exercise duration during the recovery from exhaustive exercise between the <CP and >CP conditions (at least 10 min and 39 ± 31 s, respectively; $P < 0.05$). During passive recovery and <CP recovery exercise, muscle phosphocreatine concentration ([PCr]) increased rapidly after the exhaustion point, reaching ~96% and ~76% of baseline values, respectively, after 10 min ($P < 0.05$). Moreover, pH increased abruptly, reaching 7.0 ± 0.0 and 7.0 ± 0.2, respectively, after 10 min recovery ($P < 0.05$). However, during >CP recovery exercise, neither muscle [PCr] nor pH recovered, reaching ~37% of the initial baseline and 6.6 ± 0.2, respectively. These results indicate that the muscle metabolic dynamics in recovery from exhaustive >CP differ according to whether the recovery exercise is performed below or above the CP. These findings confirm the importance of the CP as an intramuscular metabolic threshold that dictates the accumulation of fatigue-related metabolites and the capacity to tolerate high-intensity exercise.

critical power; W'; exercise tolerance; constant work rate; fatigue

THE PHYSIOLOGICAL RESPONSES during constant work-rate (CWR) exercise are highly predictable depending on the exercise-intensity domain in which an individual is exercising (12, 21, 29, 30). The asymptote of the hyperbolic relationship between power output (P) and the time to exhaustion (T_{lim}) during high-intensity exercise [critical power (CP)] marks the boundary between the heavy (<CP)- and severe (>CP)-intensity exercise domains [see (13) for review; 17, 18]. The CP therefore represents an important physiological threshold that approximates the so-called maximal lactate steady state (22, 24) and is considered to represent the highest sustainable rate of oxidative metabolism (13, 19). During sustained >CP, a slowly developing oxygen consumption ($\dot{V}O_2$) "slow component" will eventually result in the attainment of maximal $\dot{V}O_2$ ($\dot{V}O_{2max}$), with the T_{lim} attained shortly thereafter (9, 12, 21, 30). In the >CP, tolerance can be closely predicted based on the hyperbolic relationship between P and T_{lim} (13, 17, 18). The curvature constant of the power-duration hyperbola (W') represents a fixed amount of work that can be performed above CP (13, 17, 18). This constancy of W' for the entire range of supra-CP work rates means that T_{lim}, during any high-intensity exercise, coincides with the complete depletion of a fixed capacity for work above CP ($W_{>CP}$) (5, 8, 13, 17), the physiological determinants of which are uncertain (8, 13a, 26). Consistently, low values of muscle phosphocreatine concentration ([PCr]) and pH have been reported at the limit of tolerance during CWR exercise above CP (14, 26).

It has been suggested that once the W' has been exhausted and the T_{lim} attained during supra-CP exercise, the work rate must be reduced below the CP for W' to be reconstituted and for exercise to be continued (7). Coats and coworkers (7) asked subjects to complete severe-intensity CWR to T_{lim} (attained in ~6 min) and then immediately reduced the work rate to 80% of the gas exchange threshold (GET), 90% CP, or 110% CP; the subjects then attempted to complete 20 min of exercise. It was reported that all six subjects completed the 20-min target time at 80% GET, only two subjects completed the 20-min target time at 90% CP, whereas none of the subjects completed the 20-min target time at 110% CP (mean ± SD exercise time: 30 ± 12 s). The authors interpreted these findings to suggest that the W' recovers in an intensity-dependent manner following supra-CP exercise with important implications for exercise tolerance (7). Consistent with this, we have reported that recovery intervals between repeated severe-intensity work bouts enable the finite W' to be restored, with the magnitude of this reconstitution related to the intensity of the recovery interval (6). Specifically, recovery work rates below CP allow for a partial recharge of W', whereas "recovery" work rates above CP continue to deplete W', albeit at a slower rate than is observed during the work interval (6, 23). However, the intramuscular bases for this intensity-dependent W' recovery have yet to be investigated.

Therefore, the purpose of the present study was to use ^{31}P-magnetic resonance spectroscopy (^{31}P-MRS) to investigate the mechanistic bases for the intensity-dependent changes in the reconstitution of the W' and exercise tolerance immediately

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following exhaustive >CP. We did this by assessing the responses of intramuscular phosphorus-linked metabolites and pH during recovery exercise performed at different intensities following severe-intensity CWR to exhaustion. We hypothesized that recovery exercise <CP would be sustainable for an appreciable duration (at least 10 min) without significant fatigue development after the exhaustive exercise and that muscle [PCr] and pH would be recovered significantly. We also hypothesized that exercise tolerance would be severely limited during recovery exercise >CP as a consequence of an inability to recover [PCr] and pH.

METHODS

Subjects. Eight male subjects (mean ± SD: age 23 ± 5 years, stature 1.78 ± 0.03 m, body mass 76.7 ± 8.2 kg) volunteered and gave written, informed consent to participate in this study, which has been approved by the University of Exeter Research Ethics Committee. The subjects were all recreationally active and were familiar with the experimental procedures used in the study. On test days, subjects were instructed to report to the laboratory in a rested state, having completed no strenuous exercise within the previous 24 h and having abstained from food, alcohol, and caffeine for the preceding 3 h. Testing was conducted at the same time of day (±2 h) for each subject, and laboratory visits were separated by at least 48 h.

Experimental overview. The study was conducted in two parts. The power–duration relationship was first established in the laboratory during the single-leg, knee-extension exercise for each subject from four separate exercise bouts. From this relationship, the CP and W′ were estimated. Subsequently, with the use of the same ergometer, subjects performed the single-leg, knee-extension exercise to exhaustion within a magnetic resonance scanner. Muscle high-energy phosphate compounds and metabolites [PCr, ADP, inorganic phosphate (P_i), and pH] were assessed continuously at a severe-intensity CWR, estimated to result in exhaustion in 3 min (P_3; 26 ± 3 W; ~153% of CP). Immediately following exhaustion, subjects underwent a 10-min passive recovery period, or the work rate was reduced to a >CP (21 ± 4 W) or <CP (13 ± 5 W) single-leg, knee-extension exercise. These conditions were presented in random order.

Part I: derivation of the power–duration relationship and estimation of CP and W′. The subjects initially completed four severe-intensity CWR single-leg, knee-extension exercise bouts at different work rates to determine the hyperbolic power–T_{lim} relationship. The work rates for the trials were selected to yield a range of T_{lim}, varying from ~2 min for the shortest trial to ~12 min for the longest trial (26). The exercise bouts were completed on separate days and presented in random order. Subjects were placed in a prone position and secured to the ergometer bed with Velcro straps at the thigh, buttocks, and lower back to minimize extraneous movement during the exercise protocol. The ergometer consisted of a nylon frame secured on top of the bed close to the subject's feet and a base unit placed at the distal end of the bed. The subject's right foot was connected to a rope running along the top of the frame to the base unit, on which a mounted pulley system permitted brass-weight plates to be lifted and lowered. Exercise was performed at the rate of 40 contractions/min, with the subject lifting and lowering the weight over a distance of ~0.22 m, in accordance with a visual cue presented on a monitor and an audible cue timed to the bottom of the down stroke. A shaft encoder (type BDK-06; Baumer, Swindon, UK) was fitted within the pulley system to record the distance traveled by the load, alongside a nonmagnetic load cell (type F250; Novatech Measurements, St Leonards On Sea, East Sussex, UK) to record applied force, which allowed the calculation of work rate.

During all exercise tests, the subjects were verbally encouraged to continue exercising for as long as possible. The T_{lim}, which was recorded to the nearest second, was defined as the time at which the subject could no longer keep pace with the required rate of P. Subjects were not informed of the work rates or their performance until the entire project had been completed. Individual CP and W′ estimates were derived from the prediction trial by least-squares fitting of the following regression models: nonlinear P vs. time (T)

$$T = W'/(P - CP) \quad (1)$$

linear work (W) vs. time model

$$W = CP \cdot T + W' \quad (2)$$

linear P vs. 1/time model

$$P = (1/T) \cdot W' + CP \quad (3)$$

The parameter estimates from *Eqs. 1–3* were compared to ensure goodness of fit, and the model with the lowest standard error of the estimate (SEE) was chosen for further analysis (10). The 95% confidence intervals for the estimation of CP were used to calculate the work rates that were just below and just above the CP. We reasoned that this approach would provide reasonable assurance that the work rates were truly below and above the CP for all subjects.

Part II: ^{31}P-MRS assessment of muscle metabolic responses to high-intensity exercise. After completion of the predictive trials for estimation of the CP and W′, the subjects reported to the MRS laboratory at the Peninsula Magnetic Resonance Research Unit (Exeter, UK) on three separate sessions. Exhaustive >CP was performed with simultaneous measurement of muscle metabolic responses by ^{31}P-MRS using a 1.5 T superconducting magnetic resonance scanner (Intera; Philips, Amsterdam, the Netherlands) and using the same ergometer as for *part I*. To collect the ^{31}P data during the exercise protocol within the scanner, a 6-cm ^{31}P transmit/receive surface coil was placed within the ergometer bed, and the subject was positioned such that the coil was centered over the quadriceps muscle of the right leg. Initially, fast-field echo images were acquired to determine correct positioning of the muscle relative to the coil. Placement of cod-liver oil capsules, which yield high-intensity signal points within the image adjacent to the coil, allowed its orientation relative to the muscle volume under examination to be assessed. A number of pre-acquisition steps were carried out to optimize the signal from the muscle under investigation. Tuning and matching of the coil were performed to maximize energy transfer between the coil and the muscle. An automatic shimming protocol was then undertaken within a volume that defined the quadriceps muscle to optimize homogeneity of the local magnetic field, thereby leading to maximal signal collection.

Subjects were required to exercise to T_{lim} at a severe-intensity CWR, predicted to result in P_3 [with the use of the CP and W′ derived from *part I*; P = [W′/T_{lim} of 180 s (T_{180})] + CP]. Immediately following exhaustion, subjects either: *1*) continued to perform exercise at an above CP (>CP) or below CP (<CP) work rate, with exercise continued for 10 min or as long as possible, or *2*) ceased exercise completely for a 10-min period. The three tests were undertaken on separate days in random order. During the entire exercise and recovery periods, ^{31}P data were acquired every 1.5 s with a spectral width of 1,500 Hz. Phase cycling with four phase cycles was used, leading to a spectrum acquired every 6 s. The subsequent spectra were quantified by peak fitting with the assumption of prior knowledge, using the advanced method for accurate, robust, and efficient spectral (AMARES) fitting algorithm in the jMRUI (version 3) software package. Spectra were fitted with the assumption that P_i, PCr, ATP, and phosphodiester peaks were present. In all cases, relative amplitudes were corrected for partial saturation due to the repetition time relative to T1 relaxation time. The T1 saturation was corrected via a spectrum consisting of 48 individually acquired spectra that were acquired with a long relaxation time before the beginning of data acquisition.

Intracellular pH was calculated using the chemical shift of the P_i spectra relative to the PCr peak (25). The [PCr] and P_i concentra-

Table 1. *Work rates and limit of tolerance during exhaustive severe-intensity constant work-rate (CWR) exercise and subsequent resting or exercising [heavy- and severe-intensity critical power exercise (<CP and >CP, respectively)] recovery*

	Resting	<CP	>CP
CWR exercise bout, W	26 ± 3	26 ± 3	26 ± 3
T_{lim} during CWR exhaustive exercise, s	171 ± 22	182 ± 26	173 ± 23
$W_{>CP}$ during CWR exhaustive exercise, kJ	1.44 ± 0.53	1.55 ± 0.61	1.45 ± 0.55
Recovery WR, W	0 ± 0	13 ± 5	21 ± 4
T_{lim} during CWR recovery exercise, s		600 ± 0	39 ± 31*

Data are means ± SD. T_{lim}, time to exhaustion; $W_{>CP}$, work above CP. *Significantly different from resting and <CP ($P < 0.05$).

tion ([P_i]) were expressed as percentage change relative to resting baseline, which was assumed to represent 100%. Resting and end-exercise values of [PCr], [P_i], and pH were calculated over the last 90 s of the rest or the last 18 s of the exercise period. The ADP concentration ([ADP]) was calculated as described by Kemp et al. (16), assuming a baseline ATP concentration ([ATP]) of 8.2 mM. Total creatine was assumed to be the sum of PCr and free creatine, where the latter was determined based on the stoichiometry of free creatine and P_i.

Statistical analysis. One-way ANOVA was used to compare CP with W′ among the three models. Two-way, repeated-measures ANOVA were used to determine differences among the ^{31}P-MRS data ([PCr], [P_i], [ADP], and pH) and the T_{lim} for the resting, <CP, and >CP recovery conditions. Where the analysis revealed a significant difference, individual paired *t*-tests were used with least significant difference correction to determine the origin of such effects. All data are presented as mean ± SD. Statistical significance was accepted when $P < 0.05$.

RESULTS

All subjects successfully completed four CWR exercise trials for the estimation of the CP and W′. There was no significant difference in the parameter estimates derived from the three models for CP (17 ± 4, 17 ± 4, and 17 ± 4 W for *models 1*, *2*, and *3*, respectively; $P > 0.05$) or W′ (1.62 ± 0.51, 1.52 ± 0.46, and 1.52 ± 0.53 kJ for *models 1*, *2*, and *3*, respectively; $P > 0.05$). The coefficients of variation were lowest for *model 3* (6 ± 5 and 10 ± 4% for CP and W′, respectively), and the CP and W′ from *model 3* were therefore used to calculate the work rates to be used in the main experiment.

Subjects exercised at 26 ± 3 W for the CWR exercise bout (i.e., P_3). The T_{lim} during the initial exhaustive exercise bouts were 171 ± 22 s, 182 ± 26 s, and 173 ± 23 s for the resting, <CP, and >CP conditions, respectively. The T_{lim} for these three protocols were not different from one another or from the predicted T_{180} ($P > 0.05$). In addition, there was no significant difference in $W_{>CP}$ across the three conditions during the initial exhaustive exercise bouts (resting = 1.44 ± 0.53 kJ; <CP = 1.55 ± 0.61 kJ; >CP = 1.45 ± 0.55 kJ), and the $W_{>CP}$ were not significantly different from the subjects' W′, as estimated from the power–duration relationship (1.52 ± 0.53 kJ; $P > 0.05$). The subsequent recovery WR for the two experimental conditions (<CP and >CP) was 13 ± 5 and 21 ± 4 W, respectively. There was a significant difference between the exercise duration sustained during the recovery periods following the initial attainment of T_{lim} for the <CP and >CP conditions ($P < 0.05$). During <CP recovery exercise, all subjects were able to complete the targeted 10-min recovery exercise period without difficulty. However, when the recovery work rate was >CP, exercise was sustained for only 39 ± 31 s beyond the point of initial exhaustion (Table 1). This duration of continued exercise required 0.17 ± 0.18 kJ of additional $W_{>CP}$, which was significantly greater ($P < 0.05$) than the $W_{>CP}$ of 0.04 ± 0.12 kJ, theoretically available if the subjects had reached the predicted T_{180}. The amount of additional $W_{>CP}$ was also significantly greater ($P < 0.05$) than the SEE for W′ (0.15 ± 0.11 kJ) and was not significantly correlated with the T_{lim} recorded in the initial exhaustive exercise bout ($r = -0.67$; $P > 0.05$).

Table 2 presents the muscle metabolic responses during the CWR exercise with different recovery intensities. The muscle high-energy phosphate and metabolite concentrations were not significantly different at the point of exhaustion among conditions (Table 2). The muscle [PCr] decreased rapidly with T_{lim} in all conditions (approximately 38–40% of the initial baseline value). Moreover, muscle [ATP] fell by approximately 15–17% over the course of the exhaustive exercise bout, and [P_i] and [ADP] rose immediately after the onset of exercise and increased until exhaustion (approximately 524–586% and approximately 51–55 μM above the initial baseline, respectively). Muscle pH fell precipitously to reach ~6.7 at the termination of exhaustive exercise.

During resting and <CP recovery exercise, muscle [PCr] increased significantly and rapidly after the exhaustion point by 56 ± 6% and 36 ± 14%, respectively ($P < 0.05$; see Fig. 2 and Table 2). Moreover, pH increased significantly and abruptly (reaching 7.0 ± 0.0 and 7.0 ± 0.2 after 10 min), and [P_i] and [ADP] fell significantly after exhaustion ($P < 0.05$). However,

Table 2. *Muscle metabolic responses during severe-intensity CWR exercise and subsequent recovery at different intensities*

	Baseline	End-CWR Exercise	End-Recovery Period
[PCr], %			
Rest	100 ± 0	40 ± 8*	96 ± 4*†
<CP	100 ± 0	40 ± 7*	76 ± 12*†‡
>CP	100 ± 0	38 ± 6*	37 ± 8*‡§
pH			
Rest	7.0 ± 0.1	6.7 ± 0.2*	7.0 ± 0.0†
<CP	7.0 ± 0.0	6.7 ± 0.2*	7.0 ± 0.2†
>CP	7.0 ± 0.0	6.7 ± 0.2*	6.6 ± 0.2‡§
[P_i], %			
Rest	100 ± 0	586 ± 105*	68 ± 21*†
<CP	100 ± 0	524 ± 117*	178 ± 105†‡
>CP	100 ± 0	534 ± 115*	545 ± 133*‡§
[ADP], μM			
Rest	6 ± 1	51 ± 12*	9 ± 3*†
<CP	7 ± 2	54 ± 16*	23 ± 12*†‡
>CP	7 ± 1	55 ± 14*	51 ± 10*‡§
[ATP], %			
Rest	100 ± 0	83 ± 5*	93 ± 6*†
<CP	100 ± 0	82 ± 7*	87 ± 6*
>CP	100 ± 0	85 ± 4*	79 ± 4*‡§

Data are means ± SD. [PCr], phosphocreatine concentration; [P_i], inorganic phosphate concentration; [ADP] and [ATP], ADP and ATP concentration, respectively. *Significantly different from Baseline; †significantly different from End exercise; ‡significantly different from Rest; §significantly different from <CP.

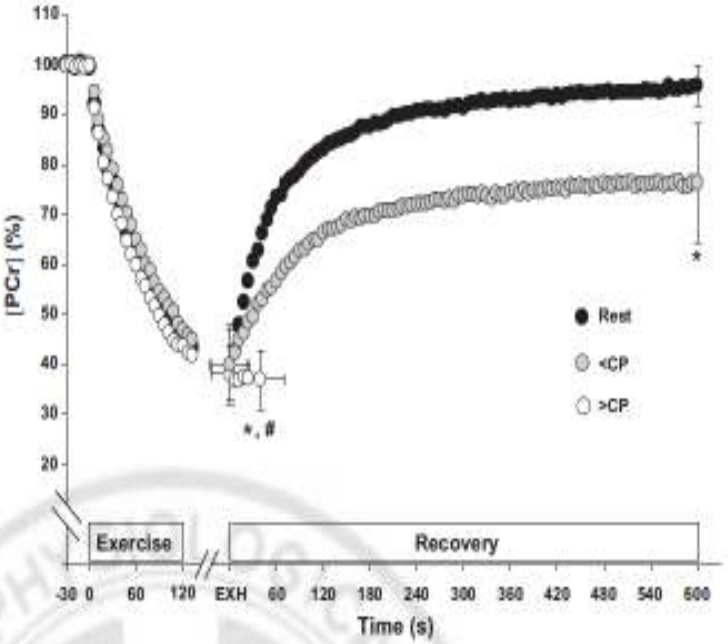

Fig. 1. Muscle phosphocreatine concentration ([PCr]) responses to constant work-rate (CWR) severe-intensity critical power exercise (>CP) and subsequent recovery exercise for resting (black circles), heavy-intensity CP exercise (<CP; gray circles), and >CP (open circles) conditions. Horizontal error bars show mean ± SD time to exhaustion (T_{lim}) for the initial exhaustive exercise bout (EXH) and the >CP recovery condition, and vertical error bars show mean ± SD [PCr] at end of exhaustive exercise and subsequent "recovery" exercise. *End-recovery muscle [PCr] was significantly less for >CP and <CP recovery compared with resting ($P < 0.05$). #End-recovery muscle [PCr] was significantly less for >CP recovery compared with resting and <CP recovery ($P < 0.05$).

during >CP recovery exercise, muscle [PCr] and [ATP] remained stable with time until exercise was terminated (~37% and 79% of the initial baseline, respectively; $P > 0.05$). There was no further change in [P_i] and [ADP] between the exhaustion point of the initial exhaustive bout and the end of recovery exercise (~545% and ~51 μM, respectively; $P > 0.05$), and the muscle pH was not significantly different from the value reached at the initial T_{lim} (6.6 ± 0.2; $P > 0.05$).

In the recovery period, the muscle [PCr], [ATP], [P_i], [ADP], and pH were significantly different among conditions (Table 2). End-recovery muscle [PCr], [ADP], and [P_i] were significantly less for >CP recovery compared with resting and <CP recovery and for <CP recovery compared with resting recovery conditions ($P < 0.05$). In addition, there was a significant difference in end-recovery exercise [ATP] and pH for >CP recovery compared with resting and <CP recovery ($P < 0.05$; Table 2). The group mean muscle metabolic response profiles are depicted in Figs. 1–4.

DISCUSSION

The principal, novel finding of this investigation was that the muscle metabolic response profile (as assessed by ^{31}P-MRS) and T_{lim} differed significantly when the exercise intensity used in the recovery from exhaustive exercise was positioned in different intensity domains (<CP and >CP). The results of the study were consistent with our hypotheses and indicate that <CP recovery exercise can be sustained for an appreciable

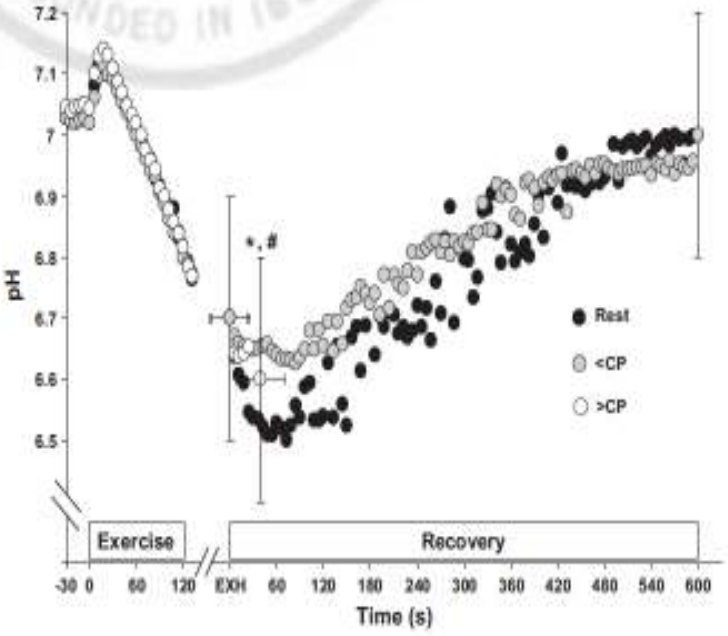

Fig. 2. Muscle pH responses to CWR >CP and subsequent recovery exercise for resting (black circles), <CP (gray circles), and >CP (open circles) conditions. Note that the recovery of pH is dependent on the rate of [PCr] recovery (see Fig. 1). Horizontal error bars show mean ± SD T_{lim} for the initial exhaustive exercise bout and the >CP recovery condition, and vertical error bars show mean ± SD pH at end of exhaustive exercise and subsequent recovery exercise. *End-recovery muscle pH was significantly less for >CP recovery compared with resting ($P < 0.05$). #End-recovery muscle pH was significantly less for >CP recovery compared with resting and <CP recovery ($P < 0.05$).

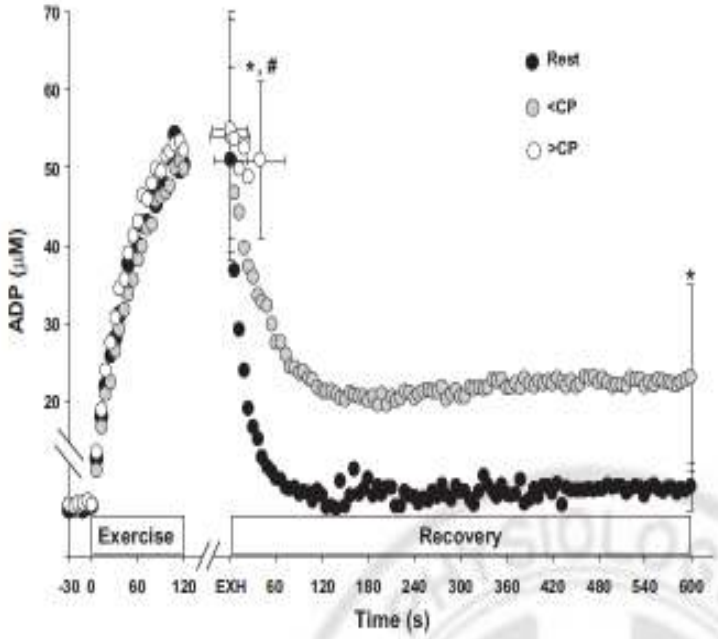

Fig. 3. Muscle ADP concentration ([ADP]) responses to CWR >CP and subsequent recovery exercise for resting (black circles), <CP (gray circles), and >CP (open circles) conditions. Horizontal error bars show mean ± SD T_{lim} for the initial exhaustive exercise bout and the >CP recovery condition, and vertical error bars show mean ± SD [ADP] at end of exhaustive exercise and subsequent recovery exercise. *End-recovery muscle [ADP] was significantly high for >CP and <CP recovery compared with resting ($P < 0.05$). #End-recovery muscle [ADP] was significantly high for >CP recovery compared with resting and <CP recovery ($P < 0.05$).

duration without significant fatigue development after initial exhaustive exercise, with muscle [PCr] and pH increasing significantly in the recovery period. However, exercise tolerance was severely limited during >CP recovery exercise, and there was no recovery in intramuscular high-energy phosphate compounds or metabolites until exercise was terminated. The results indicated that replenishment of the W′ following the limit of tolerance necessitated that work rate be reduced <CP to allow some of the fatigue-related, high-energy phosphate compounds or metabolites to be resynthesized (e.g., ATP and PCr) or cleared from the muscle [e.g., hydrogen ion (H^+)], thereby delaying the attainment of a "limiting" intramuscular environment (1, 14, 26).

There was no significant difference in $W_{>CP}$ across the three conditions during the initial exhaustive exercise bouts (approximately 1.44–1.55 kJ), and these $W_{>CP}$ were not significantly different from the subjects' W′ (1.52 kJ). This is consistent with previous studies that have reported that $W_{>CP}$ equals W′ at the limit of tolerance during >CP and confirms the notion that in this domain, the limit of tolerance is reached when subjects expend the finite work capacity available above CP; i.e., the W′ (8, 9, 21, 30). Historically, the W′ has been considered to represent a finite amount of energy available from oxygen stores (e.g., in blood and tissue), the high-energy phosphates, and a source related to anaerobic glycolysis that may be expended above the CP (17, 18). An alternative

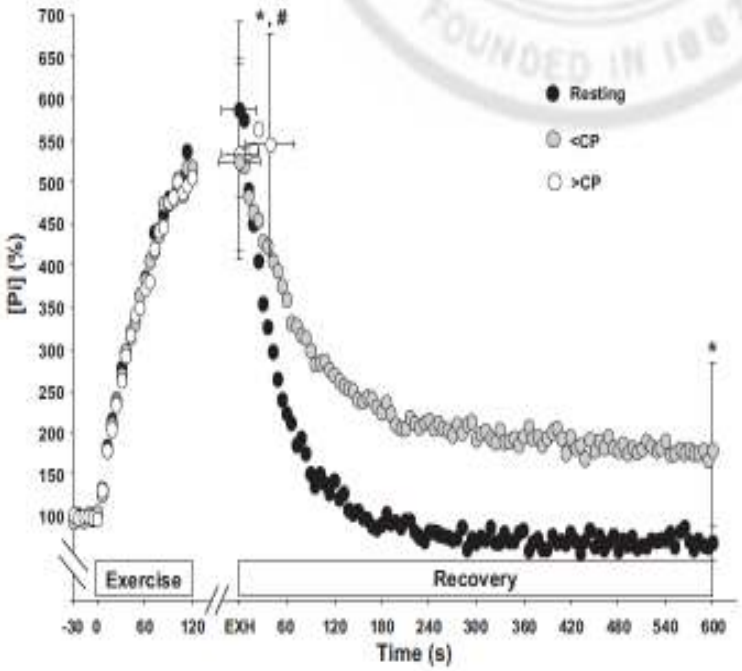

Fig. 4. Muscle inorganic phosphate concentration ([P_i]) responses to CWR >CP and subsequent recovery exercise for resting (black circles), <CP (gray circles), and >CP (open circles) conditions. Horizontal error bars show mean ± SD T_{lim} for the initial exhaustive exercise bout and the >CP recovery condition, and vertical error bars show mean ± SD [P_i] at end of exhaustive exercise and subsequent recovery exercise. *End-recovery muscle [P_i] was significantly high for >CP and <CP recovery compared with resting ($P < 0.05$). #End-recovery muscle [P_i] was significantly high for >CP recovery compared with resting and <CP recovery ($P < 0.05$).

perspective is that the W′ is related to the accumulation or depletion of one or more metabolites or substrates that are linked to the process of muscle fatigue until some "critical" concentration is attained, beyond which, the same work rate cannot be tolerated (7, 8, 13a, 14, 26). Consistent with this and with the previous study of Vanhatalo et al. (26), the present study shows that T_{lim}, during a series of severe CWR exercise bouts, was associated with the attainment of consistently low values of muscle [PCr] and pH and consistently high values of muscle [P_i] and [ADP]. We have proposed previously that changes in muscle [PCr], [ADP], [P_i], and H^+ concentration ([H^+]) are linked to the use of the W′ during >CP, with these perturbations in the muscle metabolic milieu also driving the continued development of the $\dot{V}O_2$ slow component (3, 13, 28). In this way, exercise intolerance during >CP is associated with the complete use of the W′, the attainment of some critical combination of muscle high-energy phosphate compound and/or metabolite concentrations (of which [PCr], [ATP], [ADP], [P_i], and [H^+] may act directly or serve as proxies), and the achievement of $\dot{V}O_{2max}$ (3, 5, 13, 20, 21, 28).

To our knowledge, the present study is the first to investigate the muscle metabolic responses to recovery exercise performed in different exercise-intensity domains (i.e., below and above CP) following exhaustive exercise. During resting recovery, a steady state in muscle metabolites was achieved relatively rapidly. For example, muscle [PCr] and [ATP] had recovered, on average, to ~96% and ~93% of their respective baseline values, and pH had increased to the resting value within 10 min. Following 10 min of recovery exercise, performed at a work rate that was below CP, muscle [PCr] and [ATP] had recovered, on average, to ~76% and ~87% of their respective baseline values, and pH had increased by 0.2 U from the value recorded at exhaustion. In all cases, the subjects were able to complete 10 min of <CP recovery exercise. The muscle metabolic response to recovery exercise performed above CP after exhaustive CWR exercise was markedly different from the response observed below the CP and in resting conditions. During >CP recovery exercise, despite a 19% reduction in work rate, [PCr] and [ATP] remained stable at ~37% and ~79% of their respective baseline values at the end of the recovery work rate (Fig. 1). Moreover, there was no further change in the muscle pH, [P_i], and [ADP] between the exhaustion point and the end of recovery exercise. These results support the notion that reconstitution of intramuscular metabolites following T_{lim} necessitates that the work rate be reduced below CP (6, 7, 8, 19).

Our results are consistent with the study of Coats et al. (7). These authors asked subjects to perform severe CWR cycle exercise to T_{lim} (attained in ~6 min) and then reduced the work rate to 80% GET, 90% CP, or 110% CP. The results suggested that replenishment of the W′ following T_{lim} necessitated that work rate be reduced <CP (7). The results of the present study are also consistent with our previous study (6), in which we reported that recovery intervals between severe-intensity work bouts enable the finite W′ to be restored, with the magnitude of this reconstitution related to the intensity of the recovery interval. That the W′ is expended during work bouts >CP and reconstituted during recovery intervals <CP may be understood with reference to the study of Jones et al. (14). With the use of ^{31}P-MRS, these authors showed that when CWR exercise was performed slightly above CP, [PCr] and pH continued to decrease, and [Pi] continued to increase until T_{lim} was reached. During exercise performed just below the CP, however, stable values for [PCr], pH, and [P_i] were attained within 3 min of the start of exercise, suggesting significant metabolic reserve (14). The results of the present investigation confirm that <CP recovery after exhaustive >CP allows some of the fatigue-related, high-energy phosphate compounds and metabolites to be recovered (e.g., ATP and PCr) or cleared from the muscle (e.g., H^+), thereby delaying the attainment of a limiting intramuscular environment (11, 14, 26).

In the present study, the tolerable duration for >CP recovery exercise after the initial exhaustion was ~39 s, which would have required the expenditure of a further ~0.17 kJ of $W_{>CP}$. Theoretically, there was no opportunity for W′ to be reconstituted between the work bouts, given that the work rate remained above the CP (8). The ability of the subjects to sustain exercise for any duration is therefore surprising. It should be considered that this additional $W_{>CP}$ might be related to variability in the estimation of W′ or to a less than complete use of the W′ in the initial exhaustive exercise bout. However, the additional $W_{>CP}$ was significantly greater than the standard error associated with the estimation of W′, significantly greater than the ~0.04 kJ of the W′, theoretically left "unexpended" after the initial exhaustive exercise bout (due to the T_{lim} of ~173 s being slightly shorter than the predicted T_{lim}), and not significantly correlated with the variability in the T_{lim} for the initial exhaustive exercise bout. That all the subjects in the present study were able to continue for some period of time during >CP recovery following initial exhaustion suggests that this is a "real" phenomenon that cannot be explained by experimental error. It is of interest that this finding is consistent with the study of Coats et al. (7), in which cycle exercise at a lower severe-intensity work rate could be continued for 30 ± 12 s following initial exhaustion.

The explanation for this ability of subjects to continue to exercise for some (albeit limited) duration following a reduction of work rate within the severe domain is unclear. It is possible that although the subjects were unable to maintain the required work rate at the point of exhaustion in the initial CWR exercise bout, the small reduction in the target work rate allowed a small, further reserve of W′ to be used, thus extending the net-tolerable duration of >CP exercise. In other words, fatigue impacted the maximal rate at which the W′ could be expended during the initial CWR exercise bout, and the decrease of the work rate at the point of exhaustion reduced the rate of W′ expenditure required and enabled a further small reserve of W′ to be expended. Whereas a constant W′ is an implicit assumption in the conventional two-parameter hyperbolic CP model that is derived from several CWR exercise bouts performed to the limit of tolerance, it is possible that different rules apply in other types of exercise tests and/or when work rate is manipulated close to the limit of tolerance (when W′ tends toward zero). Consistent with this, repeated maximal voluntary contractions result in a progressive reduction in maximum torque until the "critical torque" is attained (2, 4), indicating that fatigue impacts on maximal force generating capacity. Also, during a 3-min, all-out cycling sprint, the P (and therefore, the maximum rate of W′ expenditure) declines with time, as fatigue accumulates until the CP is attained (27, 28). These studies indicate that the maximal rate of W′ expenditure falls as fatigue develops but that W′ con-

tinues to be used (albeit at a progressively slower rate) until it is exhausted completely, at which point, the sustainable P = CP. In this model, prior fatiguing sprint exercise simultaneously reduces both the W′ and the peak P that can be achieved during a subsequent 3-min, all-out test (27), further suggesting that there may be a link between the absolute W′ and the peak rate at which it can be expended.

In light of these findings, the classical definition of W′ as a work capacity that is not rate limited (17–19, 21) might require reconsideration. W′ may instead be more accurately described as a finite work capacity above CP, whose maximum rate of expenditure is reduced progressively as the size of W′ remaining decreases with continued exercise >CP. Theoretically, W′ might be, at least to some extent, protocol dependent, with the W′ estimated from a series of CWR tests slightly less than the W′, which may be available if work rate is reduced (within the severe domain) as exhaustion approaches. Another factor that may contribute to the subjects' ability to continue to exercise for a short time >CP following initial exhaustion is that the reduction in work rate altered muscle tension and may have reduced afferent signaling and perhaps the perception of effort, at least temporarily, thereby facilitating an extension of exercise (1). However, it is noteworthy that intramuscular high-energy phosphate and metabolite concentrations were not altered significantly during recovery exercise >CP, such that afferent traffic may not have been reduced substantially.

In conclusion, the results of this study indicate that the muscle metabolic responses and exercise tolerance during recovery from exhaustive exercise can be understood with reference to the CP concept. The dynamics of the muscle metabolic response to recovery exercise following exhaustive >CP can be differentiated according to whether the recovery exercise is performed below or above the CP. Specifically, the <CP recovery exercise can be sustained for an appreciable duration without significant fatigue development after the exhaustive exercise, with, for example, muscle [PCr] and pH increasing significantly and rapidly after the initial point of exhaustion. However, exercise tolerance is limited during >CP recovery exercise, wherein intramuscular, high-energy phosphate compound and metabolite concentrations remained stable with time until exercise was terminated. This provides further evidence for the importance of the CP in determining the ability to maintain intramuscular homeostasis and the capacity to tolerate high-intensity exercise.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

Author contributions: J.F., A.V., and A.M.J. conception and design of research; W.C., J.F., and P.F.S. performed experiments; W.C., J.F., F.J.D., P.F.S., and A.V. analyzed data; W.C., F.J.D., A.V., and A.M.J. interpreted results of experiments; W.C. prepared figures; W.C., J.F., S.J.B., A.V., and A.M.J. drafted manuscript; W.C., J.F., S.J.B., F.J.D., P.F.S., A.V., and A.M.J. edited and revised manuscript; W.C., J.F., S.J.B., F.J.D., P.F.S., A.V., and A.M.J. approved final version of manuscript.

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Muscle metabolic responses during recovery intervals of high-intensity intermittent exercise

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Running Head: Intermittent exercise and recovery intervals.

Abstract

Purpose: To investigate the responses of intramuscular phosphate-linked metabolites and pH (as assessed by ^{31}P-MRS) during intermittent severe-intensity exercise protocols performed with different recovery-interval durations. **Methods:** Following estimation of the parameters of the power-duration relationship (i.e., CP and W′) for severe constant-work-rate exercise, nine male subjects completed three intermittent exercise protocols to exhaustion where periods of high-intensity exercise (60 s) were separated by different durations of passive recovery (18 s, 30 s and 48 s). **Results:** The tolerable duration of exercise was 304 ± 68 s, 516 ± 142 s and 847 ± 240 s for the 18-s, 30-s and 48-s recovery protocols, respectively (P<0.05). The work done >CP ($W_{>CP}$) was significantly greater for all intermittent protocols compared to the subjects' W′ (estimated from constant-work-rate exercise trials) and this augmentation became progressively greater as recovery-interval duration was increased. Similarly, the amplitude of [PCr] restoration during recovery was greatest, intermediate and least for 48-s, 30-s and 18-s of recovery, respectively (P<0.05). **Conclusion:** During high-intensity interval training, recovery intervals allow intramuscular homeostasis to be restored, with the degree of restoration related to the duration of the recovery interval. Consequently, the ability to perform $W_{>CP}$ and, ultimately, limit of tolerance during this type of exercise increases in a predictable manner when recovery-interval duration is extended.

Key Words: Critical power, W′, exercise tolerance, interval training, fatigue

Introduction

Physiological responses during constant work rate exercise are highly predictable depending upon the intensity domain in which an individual is exercising (24, 33). For example, unlike moderate-intensity exercise where a steady-state rate of O_2 consumption ($\dot{V}O_2$) is achieved rapidly (e.g., in 2-3 minutes in healthy, young adults), during heavy exercise, steady state is delayed (e.g., for up to 15 minutes) due to the emergence of a $\dot{V}O_2$ slow component, which elevates the $\dot{V}O_2$ cost of work (i.e., the $\dot{V}O_2$ 'gain') above that which is present in the moderate domain (e.g., 9-11 $ml \cdot min^{-1} \cdot W^{-1}$) (32). The $\dot{V}O_2$ slow component also characterizes severe-intensity exercise; however, in this case, a steady state can not be achieved because $\dot{V}O_{2max}$ is attained after which exhaustion is imminent (14, 24).

The power output that corresponds to the boundary between the heavy and severe domains is called the critical power (CP). Accordingly, CP is equivalent to the asymptote of the hyperbolic relationship between power output (P) and limit of exercise tolerance (T_{lim}) while the curvature constant of this hyperbola (W′) represents a fixed amount of work that can be performed above CP (9, 15, 21, 22). The physiological determinant(s) of W′ are presently uncertain; however, it is apparent that exhaustion of this provision and development of the $\dot{V}O_2$ slow component occur concomitantly such that T_{lim} ultimately depends upon the interaction between the finite capacity for W′ and the magnitude and trajectory of the $\dot{V}O_2$ slow component, in addition to the 'ceiling' imposed by $\dot{V}O_{2max}$ (2, 13, 30).

It has been shown that high-intensity interval training (HIIT; i.e., repeat repetitions of relatively brief bouts of exercise performed with 'all-out' effort or at an intensity close to that which elicits $\dot{V}O_{2max}$ interspersed with periods of rest or recovery exercise) is an

effective way to improve endurance performance by athletes and also has potential applicability in the clinical setting (6, 10, 19, 20). However, the specific paradigm to employ in order to achieve maximum benefit from HIIT (e.g., number and duration of 'work' intervals, high-intensity work rate, duration of and activity pattern during recovery intervals, etc.) remains to be determined. There is a growing body of research which suggests that the critical power concept also applies to intermittent exercise (3, 23). Consequently, potential physiological determinant(s) of W′ would be important to consider when prescribing HIIT variables.

It has been reported that if work is performed immediately after T_{lim} (e.g., during an 'active' recovery interval following an all-out work interval performed to exhaustion), the recovery work rate must be <CP in order for exercise to be continued (5). Furthermore, we have found that 30 s of recovery at a work rate <CP interspersed between repeat 60-s severe-intensity cycling bouts enables W′ restoration (and, therefore, prolongation of T_{lim} for the repeat series) compared to 30 s of recovery work at a work rate >CP, which continues to exhaust the provision, albeit at a slower rate (3). Furthermore, the magnitude of W′ restoration when <CP recovery work is allowed is inversely related to the intensity of the recovery work. For example, using group mean data, W′ reconstitution during moderate recovery was ~50% greater compared to heavy recovery (i.e., ~3 kJ per 30-s interval vs. ~2 kJ), which allowed ~200 s of additional intermittent exercise to be performed prior to T_{lim} (3). However, in addition to this intensity dependency during recovery work, magnitude of W′ restoration should also depend on recovery-interval duration and this has yet to be investigated.

Muscle metabolic responses during recovery from severe-intensity exercise can be assessed via ^{31}P-magnetic resonance spectroscopy (^{31}P-MRS). For example, we have

recently confirmed an intramuscular basis for the aforementioned intensity dependency by showing that <CP recovery allows muscle phosphocreatine concentration ([PCr]) and pH to increase and inorganic phosphate concentration ($[P_i]$) and ADP concentration ([ADP]) to decrease following exhaustion whereas >CP work rates are characterized by constancy of all of these variables (4). These results are consistent with the notion that replenishment of W′ following T_{lim} requires a work rate that allows some resynthesis and/or clearance of fatigue-related substrates and/or metabolites to assuage a limitation or limitations imposed within the intramuscular environment (16, 29). However, the degree to which these intramuscular effects are duration dependent is unclear.

The purpose of this investigation was to determine the responses of intramuscular phosphate-linked variables ([PCr], $[P_i]$ and [ADP]) and pH during intermittent severe-intensity exercise protocols performed with recovery intervals (passive rest) of different duration. We hypothesised that, during an exhaustive intermittent knee-extension exercise protocol in which severe 60-s work intervals were separated by either 18-s, 30-s or 48-s recovery intervals, the total amount of work performed above CP ($W_{>CP}$) would be greater than W′ estimated from a series of constant work rate bouts. Furthermore, we hypothesized that $W_{>CP}$ and T_{lim} for the protocol would be greatest/longest, intermediate and least/shortest for the protocols that employed the 48-s, 30-s and 18-s recovery intervals, respectively. Finally, we hypothesized that, despite the ability to continue the intermittent protocol for a longer period of time when longer recovery intervals were allowed, similar values for ^{31}P-MRS variables ([PCr], $[P_i]$, [ADP] and pH) would be present at exhaustion for all three conditions.

Methods

Subjects

Nine male subjects (mean ± SD: age 22 ± 3 years, stature 1.75 ± 0.04 m, body mass 76 ± 10 kg) volunteered and gave written informed consent to participate in this study, which had been approved by the University of Exeter Research Ethics Committee. The subjects were all recreationally active and were familiar with the experimental procedures used in the study. On test days, subjects were instructed to report to the laboratory in a rested state, having completed no strenuous exercise or consumed alcohol within the previous 24 hours, and having abstained from food and caffeine for the preceding 3 hours. Testing was conducted at the same time of day (±2 hours) for each subject and laboratory visits were separated by at least 48 hours.

Experimental Overview

This study was conducted in two parts. The power-duration relationship for single-leg knee-extension exercise was first established for each subject from four separate exercise bouts. From this relationship, the CP and W′ were estimated. The subjects then performed a single-leg knee-extension intermittent exercise protocol to exhaustion within a magnetic resonance scanner. Muscle metabolites ([PCr], [P_i], [ADP]) and pH were assessed continuously during the protocol where intervals of high-intensity exercise (60 s) were separated by three different durations of passive-recovery intervals. Recovery-interval durations were 18 s, 30 s and 48 s and these three conditions were presented to subjects in a randomised order.

Part I: Estimation of CP and W′

The subjects initially completed four severe-intensity constant work rate prediction trials at different work rates in order to determine the hyperbolic power – T_{lim} relationship. The work rates for the trials were selected in order to yield a range of T_{lim}

varying from ~2 min for the shortest trial to ~12 min for the longest trial (29). The single-leg knee-extension exercise bouts were completed on separate days and presented in randomised order. Subjects were placed in a prone position and secured to the ergometer bed with Velcro straps at the thigh, buttocks and lower back to minimize extraneous movement during the exercise protocol. The ergometer consisted of a nylon frame secured on top of the bed close to the subject's feet and a base unit placed at the distal end of the bed. The subject's right foot was connected to a rope running along the top of the frame to the base unit, on which a mounted pulley system permitted brass weight plates to be lifted and lowered. Exercise was performed at the rate of 40 contractions·min^{-1}, with the subject lifting and lowering the weight over a distance of ~0.22 m in accordance with a visual cue presented on a monitor and an audible cue timed to the bottom of the down stroke. A shaft encoder (type BDK-06, Baumer Electrics, Swindon, UK) was fitted within the pulley system to record the distance travelled by the load, alongside a non-magnetic load cell (type F250, Novatech Measurements, St Leonards-On-Sea, UK) to record applied force, which was then used to calculated the work rate.

During all exhaustive tests, the subjects were verbally encouraged to continue exercising for as long as possible. The T_{lim}, which was defined as the time at which the subject could no longer keep pace with the required rate of muscle contraction, was recorded to the nearest second. Subjects were not informed of the work rates or their performance until the entire project had been completed. Individual CP and W′ estimates were derived from the prediction-trial data by least squares fitting of the following regression models:

(1) Non-linear power (P) *versus* time (T):

$$T = W' / (P - CP) \quad (1)$$

(2) Linear work (W) *versus* time model:

$$W = CP \times T + W' \quad (2)$$

(3) Linear Power (P) versus 1/time model:

$$P = (1/T) \times W' + CP \quad (3)$$

The parameter estimates from equations (1), (2) and (3) were compared to ensure goodness of fit, and the model with the lowest standard error of the estimate (SEE) was chosen for further analysis.

Part II: ^{31}P-MRS assessment of muscle metabolic responses to high-intensity intermittent exercise

After completion of the prediction trials for estimation of the CP and W′, the subjects reported to the MRS laboratory at the Peninsula Magnetic Resonance Research Unit (Exeter, UK) on three separate sessions. Exhaustive intermittent protocols were performed with simultaneous measurement of muscle metabolic responses by ^{31}P-MRS using a 1.5 T superconducting magnetic resonance scanner (Intera, Philips, Amsterdam, the Netherlands) and employing the same ergometer as for Part I. In order to collect the ^{31}P-MRS data during the protocol, a 6 cm ^{31}P transmit/receive surface coil was placed within the ergometer bed, and the subject was positioned such that the coil was centred under the quadriceps muscle of the right leg. Initially, fast field echo images were acquired to determine correct positioning of the muscle relative to the coil. Placement of cod liver oil capsules, which yield high-intensity signal points within the image, adjacent to the coil, allowed its orientation relative to the muscle volume under examination to be assessed. A number of pre-acquisition steps were carried out to optimize the signal from the muscle under investigation. Tuning and matching of the coil was performed to maximize energy transfer between the coil and the muscle. An automatic shimming protocol was then undertaken within a volume that defined the

quadriceps muscle to optimize homogeneity of the local magnetic field, thereby leading to maximal signal collection.

Subjects were required to perform single-leg knee-extension exercise using three intermittent protocols where periods of high-intensity exercise (60 s) were separated by different durations of passive recovery intervals. Recovery-interval durations for the three conditions were 18 s, 30 s and 48 s and all bouts were continued to T_{lim}, which was defined and recorded as it was for Part I (see above). The high-intensity work rate for the work intervals of these protocols was calculated using the CP and W′ estimates from Part I according to the intermittent CP model (i.e., *Eqn.* 4; 23) in order to provide a work rate that was predicted to elicit exhaustion after 4, 6 and 8 completed work/recovery cycles (i.e., after 312 s, 540 s and 864 s of intermittent exercise) for the 18-s, 30-s and 48-s recovery conditions, respectively.

$$T_{lim} = n\,(t_w + t_r) + [W' - n\,\{(P_w - CP)\,t_w - (CP - P_r)\,t_r\}] / (P_w - CP) \quad (4)$$

where T_{lim} is total protocol time, n is the number of completed work/recovery cycles, t_w and t_r are the durations and P_w and P_r are the power outputs of the work and recovery intervals, respectively. The three protocols were undertaken on separate days in random order.

During the entire exercise and recovery periods, ^{31}P-MRS data were acquired every 1.5 s with a spectral width of 1500 Hz and 1000 data points. Phase cycling with four phase cycles was employed, leading to a spectrum being acquired every 6 s. The subsequent spectra were quantified by peak fitting, with the assumption of prior knowledge, using the AMARES fitting algorithm in the jMRUI (version 3) software package. Spectra were fit with the assumption that P_i, PCr, ATP and phosphodiester peaks were present. In all cases, relative amplitudes were corrected for partial saturation due to the

repetition time relative to T1 relaxation time. The T1 saturation was corrected via a spectrum of 48 individually acquired spectra that were acquired with a long relaxation time prior to the beginning of data acquisition.

Intracellular pH was calculated using the chemical shift of the P_i spectra relative to the PCr peak (27). The [PCr] and [P_i] were expressed as percentage change relative to resting baseline (i.e., prior to initiation of the protocol), which was assumed to represent 100%. The [ADP] was calculated as described by Kemp *et al*. (18).

Data Analysis Procedures

To estimate the work done above CP ($W_{>CP}$; expressed in kJ) during the intermittent exercise protocols, we used the following equation:

$$W_{>CP} = [(P_w \times t_w) - (CP \times t_w)]/1000 \quad (5)$$

where P_w is the high-intensity work rate, t_w is the cumulative time spent at P_w during the intermittent protocol and CP is the critical power.

Pre-protocol (i.e., "Baseline") values for [PCr], [P_i], [ADP] and pH were defined as the mean values measured over the final 120 s of rest (i.e., prior to initiation of the first high-intensity work interval) while end-protocol (i.e., "End-exercise") values for these variables were defined as the mean values measured over the final 18 s of exercise. The changes in [PCr], [P_i], [ADP] and pH across the protocol (Δ[PCr], Δ[P_i], Δ[ADP], ΔpH) were then calculated as the difference between end-protocol and pre-protocol values.

For each recovery interval, the pre-recovery [PCr] ($[PCr]_{pre}$) was defined as the value measured during the final 6 s of the preceding work interval and the post-recovery [PCr] (PCr_{post}) was defined as the value measured during the final 6 s of that specific recovery

interval. The amplitude of [PCr] restoration during each recovery interval was then calculated as the difference between $[PCr]_{post}$ and $[PCr]_{pre}$. These values were calculated for all completed intervals and averaged across each intermittent protocol to provide one average [PCr] restoration amplitude for each condition.

In addition to calculating the magnitude of [PCr] restoration for each protocol, we were also interested in comparing the time course of [PCr] restoration across both protocol (e.g., for the 48-s recovery interval compared to the 18-s recovery interval) and time (e.g., for the final recovery interval compared to the initial recovery interval). However, quantifying the response by fitting it with the higher-order model that would likely be necessary under these conditions (8) was impossible due to sparsity of data points (e.g., only four points available in the 18-s recovery condition). Consequently, we determined a [PCr] restoration mean response time for the initial and final recovery interval (i.e., MRTi and MRTf, respectively) of each protocol by fitting the first four data points collected during the recovery interval with a single exponential function with the amplitude term fixed based on the assumption that full restoration (i.e., restoration to the pre-protocol baseline value of 100%; see above) would take place. Consequently, the first four [PCr] data points during the initial and final recovery interval for each protocol were fit with an exponential function of the form:

$$[PCr]_t = [PCr]_{pre} + [PCr]_{\Delta}(1 - \exp^{(-t / MRT)}) \quad (6)$$

where $[PCr]_t$ is the [PCr] at any given time t during the recovery interval, $[PCr]_{pre}$ is the [PCr] prior to initiating the recovery interval, $[PCr]_{\Delta}$ is the [PCr] amplitude that would be required for full restoration (i.e., 100 - $[PCr]_{pre}$) and MRT is the [PCr] mean response time (i.e., the time required to achieve 63% complete restoration with no distinction made for various phases of the response).

Statistical Analysis

One-way ANOVA was employed to compare CP and W′ between the three models. One-way repeated-measures ANOVAs were employed to determine differences between the three intermittent protocols for the ^{31}P-MRS data ([PCr], [P_i], [ADP] and pH), T_{lim} and $W_{>CP}$. A two-way repeated-measures ANOVA was used to assess differences in [PCr] MRT between the first and last recovery intervals across the three intermittent protocols. Where the analysis revealed a significant difference, simple contrasts with Fisher's LSD were used to determine the origin of such effects. All data are presented as mean ± SD. Statistical significance was accepted when $P<0.05$.

Results

All subjects successfully completed the four constant work rate exercise trials for the estimation of the CP and W′ (i.e., Part I; see above). There were no significant differences between models for the CP estimates (16 ± 4 W, 17 ± 4 W and 15 ± 4 W for models 1, 2, and 3, respectively, $P > 0.05$) or for the W′ estimates (1.90 ± 0.67 kJ, 1.83 ± 0.64 kJ and 2.16 ± 0.74 kJ for models 1, 2, and 3, respectively, $P > 0.05$). Using these estimates, the high-intensity work rate for the work intervals of the intermittent protocols (i.e., P_w derived via *Eqn.* 4; see above) was 33 ± 7 W.

Table 1 presents the values for T_{lim} and $W_{>CP}$, and the ^{31}P-MRS data for the three high-intensity intermittent exercise protocols. T_{lim} was significantly different between the three protocols. Specifically, T_{lim} was 304 ± 68 s for the high-intensity intermittent protocol that employed the 18-s recovery intervals, 516 ± 142 s (i.e., ~69% longer) for the one that employed the 30-s recovery intervals ($P<0.05$) and 847 ± 240 s (i.e., ~179% longer) for the one that employed the 48-s recovery intervals ($P<0.05$). Similarly, $W_{>CP}$

and [PCr] restoration amplitude also became progressively greater as recovery-interval duration was increased with all three values significantly different from one another (Table 1). Furthermore, $W_{>CP}$ for all three intermittent protocols was greater than the W′ estimated from the conventional constant work rate trials (see above; $P<0.05$). Conversely, the [PCr] MRT was not significantly different across conditions for either the initial or final recovery interval. However, two-way ANOVA revealed a significant main effect by time indicating that across protocols, the [PCr] MRT was lengthened (i.e., [PCr] restoration kinetics were slower) during the final compared to initial recovery interval ($F=14.1$, $P=0.006$, effect size=0.64). Finally, there was no significant difference between baseline, end-exercise or Δ values for any of the intramuscular variables measured via ^{31}P-MRS. For example, [PCr] at T_{lim} was ~40% of the pre-protocol value and pH was ~6.6 (i.e., a Δ across the protocol of ~60% and ~0.4 for [PCr] and pH, respectively) regardless of recovery-interval duration that was allowed (see Figures 1, 2 and 3).

Discussion

The principal original finding of this investigation was that the $W_{>CP}$ became progressively greater and limit of tolerance became progressively longer as recovery-interval duration was increased during an intermittent single-leg knee-extension exhaustive exercise protocol. However, at exhaustion, similar values for ^{31}P-MRS intramuscular variables (i.e., [PCr], [P_i], [ADP] and pH) were observed. These results are consistent with our hypotheses and indicate that longer recovery intervals during high-intensity intermittent exercise delay the attainment of the limiting intramuscular environment that ultimately determines limit of tolerance (16).

The present findings are consistent with the critical power model of bioenergetics, which describes a two-component system comprising an oxidative component that is limited in rate, but unlimited in capacity (CP), and a supplementary component (W′) that reflects a finite capacity to perform work above CP (15). Within this schema, while W′ can be expended at different rates, a consistent feature for all constant work rate exercise above CP is that limit of tolerance will coincide with complete depletion of W′. Furthermore, we have previously confirmed that this highly-predictable nature of the ability to perform supra-CP work also applies when intervals of recovery are interspersed between severe-intensity 'work' intervals (e.g., during high-intensity interval training; HIIT) (3). Specifically, $W_{>CP}$ can be increased and T_{lim} prolonged if recovery intervals are performed at work rates that allow some degree of W′ recharge (i.e., work rates below CP) (3, 26). In the present study, we employed recovery intervals of passive rest to maximize W′ repletion; however, we provided only relatively short periods of time (i.e., 18 s, 30 s and 48 s) before asking subjects to initiate the subsequent 60-s work interval (i.e., work/recovery ratios of 3.3:1, 2:1 and 1.25:1 for the 18-s, 30-s and 48-s recovery conditions, respectively). Nevertheless, in all three conditions, the $W_{>CP}$ was greater than the W′ estimated from the constant work rate trials suggesting that some degree of W′ repletion had occurred during the protocol.

In the present study, we used ^{31}P-MRS to measure intramuscular metabolic responses during the exhaustive protocols and found that regardless of recovery-interval duration, the values for intramuscular variables were similar at exhaustion. For example, [PCr] had decreased to ~40% of its pre-exercise value, pH had dropped from ~7.0 to ~6.6 and P_i had increased more than 5-fold. These findings are consistent with the notion that W′ is related to the accumulation or depletion of one or more metabolites or substrates

linked to the process of muscle fatigue (15, 16). In this regard, we have previously shown that recovery of these intramuscular variables following exhaustive severe-intensity exercise depends on whether the recovery period involves exercise below or above the CP (4). Specifically, [PCr] and pH increased significantly and [P_i] and [ADP] fell significantly during 10 minutes immediately following exhaustive exercise when either passive rest was allowed or exercise <CP was performed (4). However, these variables could not recover when >CP exercise was attempted and, consequently, exercise tolerance in that case was severely limited (e.g., only an additional ~39 s of exercise could be performed) (4). In the present study, the 48-s recovery interval provided the greatest opportunity for partial recovery of these variables, which explains why the greatest augmentation of $W_{>CP}$ (and, therefore, T_{lim}) was observed for this condition.

In the present study, the average amplitude of [PCr] restoration during 48-s recovery intervals was approximately twice that which occurred when 18-second periods of recovery were allowed. Interestingly, $W_{>CP}$ during the entire protocol employing the 48-s recovery intervals was also approximately doubled (see Table 1). While the precise physiological determinants of W′ remain to be determined, this finding is consistent with prior ones which indicate that [PCr] plays a significant role. For example, another consistent feature of supra-CP exercise is that $\dot{V}O_2$ rises inexorably (due to the presence of a $\dot{V}O_2$ 'slow component') such that once T_{lim} is attained (and, therefore, W′ is exhausted), $\dot{V}O_{2max}$ will have been reached (11). The kinetics of pulmonary $\dot{V}o_2$ have been shown to be similar to those of muscle [PCr] (16, 25), which supports the notion that the maximum rate of O_2 consumption corresponds with the maximum rate of [PCr] depletion. Furthermore, the link between [PCr] and $\dot{V}O_2$ is

believed to reflect feedback control of oxidative phosphorylation through one or more of the reactants and/or products of high-energy phosphate hydrolysis. Specifically, [ADP] is believed to play an important role (1). However, the degree to which [ADP] can rise during high-intensity exercise is limited by increased [H^+] due to greater activation of glycolysis (6, 17). Consequently, the only way to continue performing supra-CP exercise long enough for $\dot{V}O_2$ to rise to $\dot{V}O_{2max}$ (i.e., to achieve the 'maximum' $\dot{V}O_2$ slow component; 30) would be by reducing [PCr] to offset the fall in pH. This explains how restrictions imposed by the ability of [PCr] to continue to fall might ultimately limit the ability for $\dot{V}O_2$ to continue to rise and, therefore, work rate to be continued to be maintained such that W′ ultimately reflects this capacity.

In addition to assessing the magnitude of [PCr] restoration during the recovery intervals employed in the present study, we also characterized the [PCr] restoration time course. Unfortunately, in this regard, our methodology involved relatively short periods of recovery, which means we had limited data points available for modelling the response. Consequently, we could not use the higher-order model that would likely be required for this type of exercise (e.g., see 8) and our results should, therefore, be interpreted with caution. However, as would be expected, we did find that the [PCr] MRT (i.e., the time taken to achieve 63% of the overall response amplitude assuming a single exponential response profile that allowed for full restoration) was not significantly different between conditions, but did become longer for all conditions as the high-intensity protocol preceded (i.e., for the final recovery interval compared to the initial one). For example, for the 48-s recovery protocol, the [PCr] restoration MRT increased by ~16 s, which means that 'complete' restoration (from a functional standpoint, considered to have occurred after MRT x 4 s; 31) would have required an additional 64

s (i.e., 312 s v. 248 s). This slowing of PCr restoration as the intermittent protocol continued (likely a consequence of the progressive decline in pH; 12, 28) is important to consider when prescribing HIIT recovery-interval variables because if the goal is to provide for a consistent degree of restoration prior to initiating a subsequent work interval, recovery-interval duration should be progressively lengthened over the course of the bout.

In conclusion, the results of this study indicate that the muscle metabolic responses and exercise tolerance during high-intensity intermittent exercise protocols utilizing recovery intervals of different durations can be explained in accordance with the CP model of bioenergetics. Specifically, when an interval of passive rest (e.g., for 18 s) is interspersed between 60-s intervals of high-intensity single-leg knee-extension exercise, $W_{>CP}$ is enhanced compared to the W′ measured for constant work rate exercise. Furthermore, when recovery-interval duration is extended during the same protocol (e.g., to 30 s and 48 s), the augmentation of $W_{>CP}$ is also progressively increased, which allows T_{lim} to be extended (e.g., by ~550 s for the 48-s compared to 18-s recovery condition). However, regardless of recovery-interval duration, similar values for ^{31}P-MRS variables (i.e., [PCr], [P_i], [ADP] and pH) were present at exhaustion. Our findings also suggest that the degree of [PCr] restoration is a likely contributor to the enhanced capacity for $W_{>CP}$ under these circumstances as the average [PCr] restoration amplitude during recovery intervals of the 48-s protocol was ~2-times greater than that for the 18-s recovery condition. However, for all conditions, the [PCr] restoration time course became slower as the high-intensity protocol progressed. This lengthening of recovery kinetics during progressive rest intervals of high-intensity intermittent exercise is important to consider when prescribing a high-intensity interval training program.

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Figures

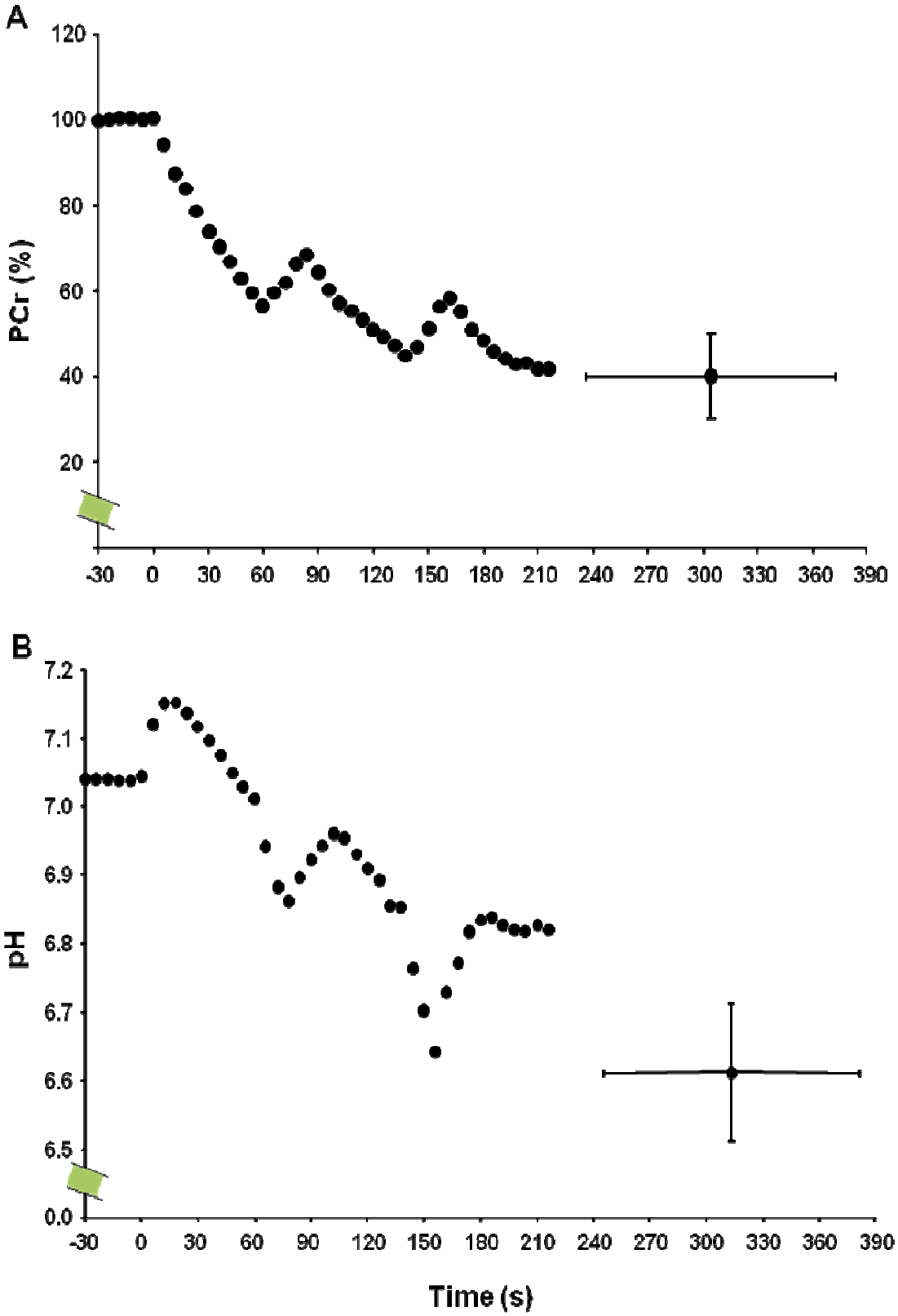

Figure 1: Muscle [PCr] and pH responses during high-intensity intermittent exercise with 18 s recovery intervals.

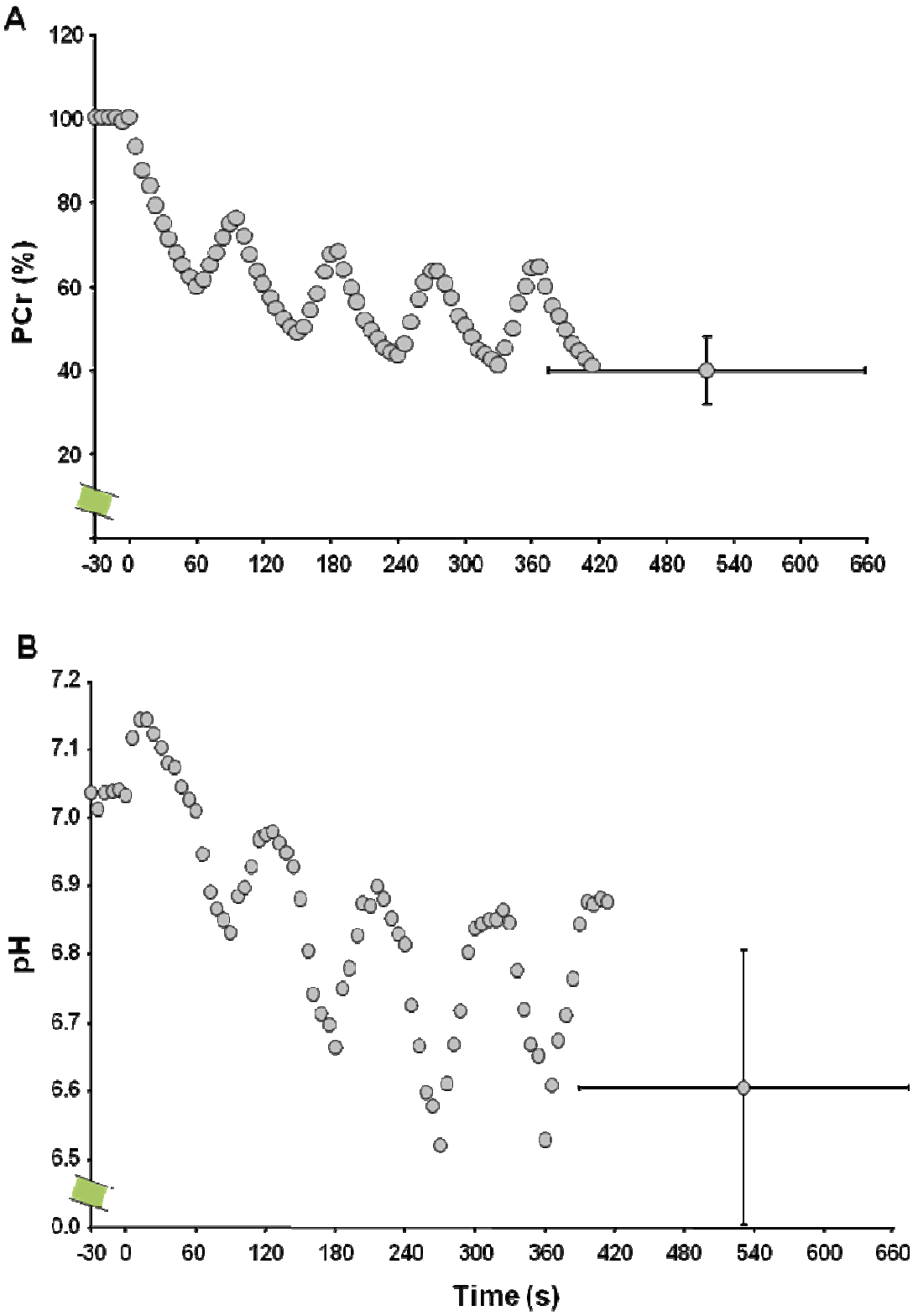

Figure 2: Muscle [PCr] and pH responses during high-intensity intermittent exercise with 30 s recovery intervals.

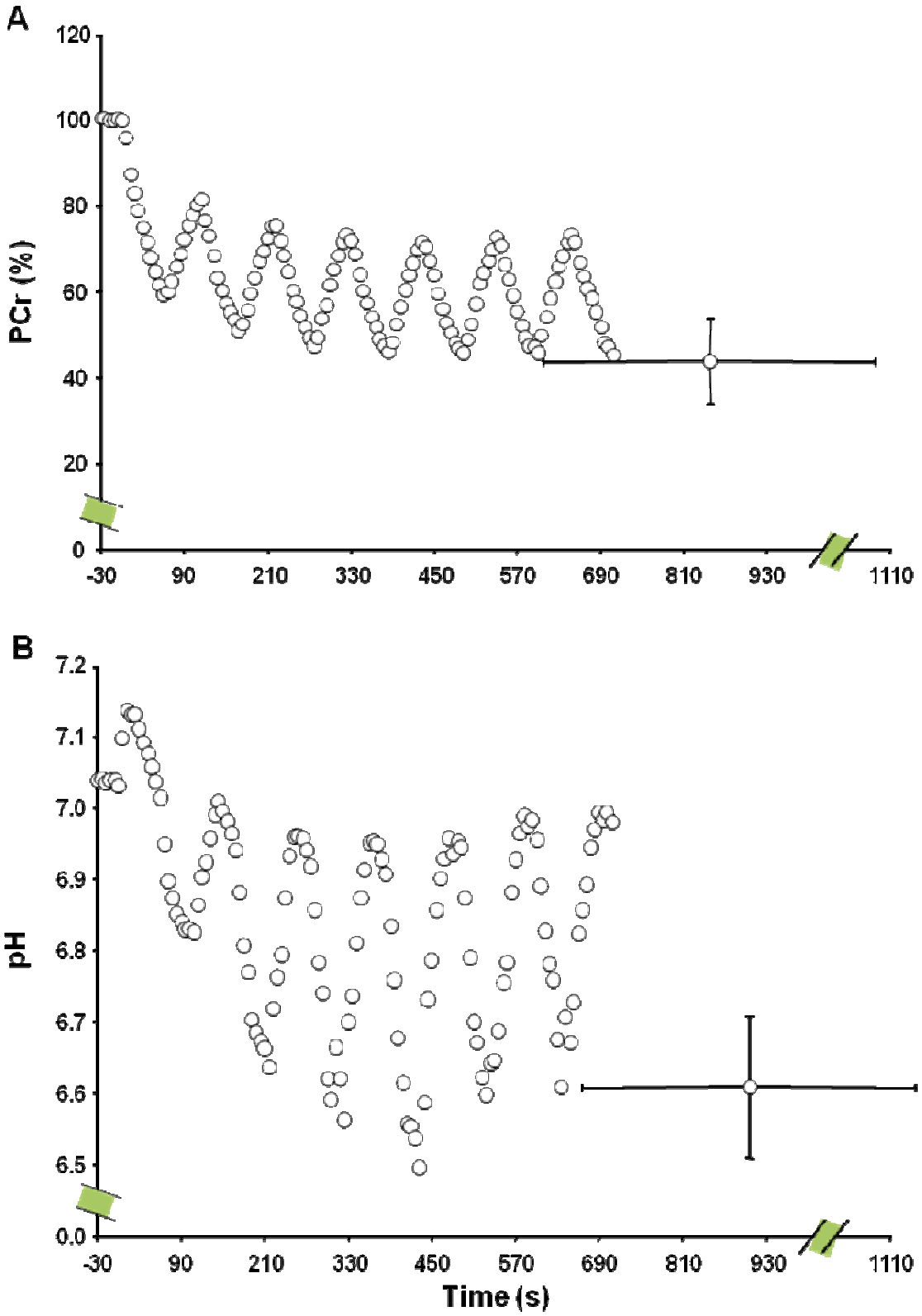

Figure 3: Muscle [PCr] and pH responses during high-intensity intermittent exercise with 48 s recovery intervals.

Table 1: Selected muscle metabolite values and limit of tolerance during intermittent high-intensity exercise protocols with different recovery durations.

	18 s	**30 s**	**48 s**
Baseline [PCr] (%)	100 ± 0	100 ± 0	100 ± 0
End-exercise [PCr] (%)	40 ± 10	40 ± 8	44 ± 10
Δ[PCr] (%)	60 ± 10	60 ± 8	56 ± 11
Baseline [P_i] (%)	100 ± 0	100 ± 0	100 ± 0
End-exercise [P_i] (%)	592 ± 285	519 ± 218	555 ± 236
Δ[Pi] (%)	492 ± 285	419 ± 218	455 ± 236
Baseline [ADP] (μM)	6 ± 3	6 ± 2	5 ± 2
End-exercise [ADP] (μM)	65 ± 24	72 ± 52	69 ± 37
Δ[ADP] (μM)	58 ± 23	66 ± 51	64 ± 37
Baseline pH	7.0 ± 0	7.0 ± 0	7.0 ± 0
End-exercise pH	6.6 ± 0.2	6.6 ± 0.2	6.6 ± 0.2
ΔpH	0.4 ± 0.1	0.4 ± 0.2	0.4 ± 0.2
[PCr] restoration amplitude (%)	13 ± 5	21 ± 4a	27 ± 7a,b
[PCr] restoration MRT_i (s)	67 ± 30	60 ± 16	62 ± 16
[PCr] restoration MRT_f (s)	71 ± 26	62 ± 8	78 ± 26
T_{lim} (s)	304 ± 68	516 ± 142a	847 ± 240a,b
$W_{>CP}$ (kJ)	3.8 ± 1.0	5.6 ± 1.8a	7.9 ± 3.1a,b

a: significantly different from 18 s (P<0.05); b: significantly different from 30 s (P<0.05).

Chapter 9 Discussion

The relationship between external work/power and time has been extensively investigated. It is now well established that, during CWR exercise, the relationship between external power output and the time to fatigue is hyperbolic (Carnevale and Gaesser, 1991; Gaesser et al., 1995; Hill, 2004; Hill et al., 2002; Hill and Smith, 1999) and is defined by two parameters: an asymptote (critical power; CP) reflecting the highest sustainable rate of oxidative metabolism, and a curvature constant (W′), which indicates the fixed amount of work that can be completed above CP ($W_{>CP}$) (Jones et al., 2010). These two parameters form the basis for the CP model of human bioenergetics which proposes that two endogenous energy supply components (CP and W′) interact to dictate the limit of tolerance during severe-intensity exercise (Jones et al., 2008, 2010; Poole et al., 1988). Specifically, the CP model proposes that W′ utilisation is obligatory during severe-intensity exercise (>CP) with exhaustion occurring when W′ is expended. Indeed, it has been reported that $W_{>CP}$ equals W′ at the limit of tolerance during severe-intensity CWR exercise (Fukuba, 2003; Hill, 2002; Poole et al., 1988). It has been further suggested that the tolerable duration of CWR exercise within the severe-intensity domain is dependent upon the interaction between W′, the $\dot{V}_{O_2}$ slow component and the $\dot{V}_{O_{2max}}$ since the development of the $\dot{V}_{O_2}$ slow component parallels the depletion of W′ such that $\dot{V}_{O_{2max}}$ attainment and complete W′ utilisation occur in unison (Burnley and Jones, 2007; Jones et al., 2011). Although its mechanistic bases have yet to be completely defined, W′ appears to be linked to ATP turnover through PCr hydrolysis and anaerobic glycolysis, and the attendant accumulation of intra-muscular (H^+, ADP and Pi) and extra-muscular (potassium [K^+]) metabolites (Fukuba et al., 2003; Jones et al., 2008; Monod and Scherrer, 1965; Moritani et al., 1981; Poole et al., 1988; Vanhatalo et al., 2010). Therefore, the termination of severe-intensity CWR exercise coincides with the complete depletion of W′, which occurs in concert with the

attainment of the $\dot{V}_{O_{2max}}$ and a critically low level of anaerobic substrate (PCr and glycogen) availability and/or critically high levels metabolite accumulation (H^+, ADP, P_i and K^+) within the muscle, at least during CWR exercise (Jones et al., 2008; Poole et al., 1988; Vanhatalo et al., 2010).

The extent to which the CP model can explain the degree of metabolic perturbation and performance during exercise tests using different methods of work rate imposition is less clear. Recently, it has been proposed that the CP model might be able to predict exercise tolerance in incremental and intermittent exercise tests. However, empirical evidence to support the efficacy of the CP model in these settings is currently lacking. It is also unclear whether the CP model can explain performance during an exercise performance trial where participants are able to freely regulate their own pace. If the CP model can help predict metabolic perturbation and exercise performance in tests where the work is not held constant, this will have important practical applications in physical activity and competitive sports. Moreover, resolving the mechanistic bases of W′ will improve our understanding of fatigue during high-intensity exercise. Therefore, the overriding purpose of this thesis was to assess the effectiveness of the CP model to predict metabolic perturbation and performance across exercise tests employing a range of different work rate forcing functions. This thesis was also concerned with further exploring the mechanistic bases for W′ using ^{31}P-MRS and its role in dictating the tolerable duration of intermittent exercise performance.

9.1 Research Questions Addressed

To investigate the extent to which performance could be explained by the CP model during variable work rate exercise, this thesis set about answering the following questions in the five experimental chapters:

1) Can exercise tolerance during intermittent high-intensity exercise with recovery intervals at different exercise intensities be explained by the CP model?
2) Does incremental cycling to exhaustion that is paced according to the RPE permit a higher $\dot{V}_{O2max}$ and increased $W_{>CP}$ compared to a conventional ramp incremental protocol when test duration is matched?
3) Does self-paced exercise influence the work completed above CP ($W_{>CP}$) and the peak $\dot{V}o_2$ attained during high-intensity cycling compared to a bout of duration-matched high-intensity CWR cycling?
4) How do muscle [PCr], [ADP], [P_i] and pH respond after exhaustive exercise during recovery exercise performed above and below CP?
5) How do muscle [PCr], [ADP], [P_i] and pH respond during intermittent severe-intensity exercise bouts separated by recovery bouts of different durations?

9.2 Summary of the Main Findings

Critical power concept during intermittent high-intensity exercise

The $W_{>CP}$ and limit of tolerance during intermittent exercise differed significantly when the recovery intervals were positioned in different intensity domains (<CP). During constant-work-rate or intermittent exercise performed exclusively in the severe domain, the $W_{>CP}$ was not significantly different from the subjects' W′, as estimated from the 3-min all-out test. This confirms and extends the notion that, in this domain, the limit of tolerance is reached when subjects expend the finite work capacity available above CP, i.e. the W′. When intermittent exercise involving work bouts in the severe domain and recovery intervals in lower intensity domains was performed, however, the limit of tolerance (and the $W_{>CP}$) was extended, being greatest for S-L, intermediate for S-M, and least for S-H. The slopes describing the increases in $\dot{V}_{O_2}$ and iEMG with time during intermittent exercise were reduced when the recovery intervals were completed at progressively lower intensities. The results of this study indicate that physiological responses and exercise tolerance during intermittent exercise can be understood in terms of the CP concept. Recovery intervals between severe work bouts enable the finite W′ to be restored, with the magnitude of this reconstitution being related to the intensity and duration of the recovery interval. This study showed that the reconstitution of W′ during the recovery intervals between repeated bouts of intense exercise results in a reduced rate of change of both iEMG and $\dot{V}_{O_2}$ as exercise proceeds and influenced the tolerable duration of exercise consistent with the CP model of bioenergetics.

Link between $\dot{V}_{O2max}$ and W′ during incremental exercise

An incremental cycling protocol that allowed subjects to self pace their power output according to a prescribed RPE did not result in higher $\dot{V}_{O2max}$ values compared to conventional incremental protocols where the ramp rate was computer-controlled and

applied in a strictly linear fashion. Furthermore, $\dot{V}_{O2max}$ was not different during the conventional tests in which cycling cadence was held constant (at the subject's own preferred cadence) or allowed to fluctuate throughout the test according to subject preference. The relative constancy of the highest $\dot{V}_{O2}$ achieved across the various protocols studied herein supports the long-standing interpretation that, for a specific mode of exercise, there is a reproducible $\dot{V}_{O2}$ that cannot be exceeded (i.e., a $\dot{V}_{O2max}$). The results of this study indicate that incremental cycling to exhaustion is characterized by a similar peak $\dot{V}_{O2}$ response regardless of whether the power output is applied linearly by the experimenter or regulated by the subject's own perception of effort. The peak $\dot{V}_{O2}$ values measured across an array of protocols (i.e., a continuous-graded protocol that was self-paced according to a prescribed RPE, a ramp incremental protocol with cadence allowed to fluctuate according to subject preference, a ramp incremental protocol with fixed cadence, a high-intensity constant-power-output test to the limit of tolerance, and a 3-min all-out test during which power output declined precipitously over time) were strikingly similar (within 1%). There was no also no significant difference in $W_{>CP}$ across the protocols. These consistent $\dot{V}_{O2max}$ and $W_{>CP}$ values were attained in the five different exercise tests despite clear differences in muscle activation profiles, whereas increasing subject autonomy did not appreciably alter the degree of muscle activation at end-exercise during the three iso-duration incremental protocols. These results are consistent with the CP model of bioenergetics and suggest that $\dot{V}_{O2max}$ is attained when the W′ is fully expended during exhaustive >CP exercise.

The ability to self-pace does not increase $\dot{V}_{O2max}$ and W′ during high-intensity exercise

The total amount of work that could be performed prior to the limit of tolerance during exercise exceeding the CP (i.e., $W_{>CP}$) was similar across different high-intensity

exercise protocols that expended this capacity (W′) at different rates. Specifically, regardless of whether it was produced by enforced-pace exercise (either INT or CWR) or a protocol where subjects were allowed to choose their own pacing strategy (SPT), the termination of exercise coincided with a similar $W_{>CP}$ and $\dot{V}_{O2max}$. Together, the CP and W′ estimated using the AOT enabled the prediction of the T_{lim} during CWR with moderate accuracy (CV ~8%), and the prediction of T_{lim} during INC and the total work done during SPT with good accuracy (CV ~3% for both). The results of this study indicate that utilization of a 'fixed' capacity for $W_{>CP}$ (i.e., W') and the associated increase in $\dot{V}_{O_2}$ to a reproducible peak $\dot{V}o_2$ (i.e., $\dot{V}o_{2max}$) are consistently associated with attainment of the limit of tolerance during high-intensity (>CP) exercise irrespective of the work-rate forcing function. These findings support the use of the CP model to predict the amount of supra-CP work that can be completed before exercise termination even if the subject has autonomy over their pacing strategy.

Mechanistic bases for W′ recovery after exhaustive exercise

The muscle metabolic response profile (as assessed by ^{31}P-MRS) and T_{lim} differed significantly in the recovery from exhaustive exercise when the recovery exercise was positioned in different exercise intensity domains (<CP and >CP). Specifically when the work rate in the recovery bout was <CP, exercise was sustained for an appreciable duration (at least 10 min), muscle [PCr] and pH increased and muscle [P_i] and [ADP] declined significantly in the recovery period. However exercise tolerance was severely limited during >CP recovery exercise (39 ± 31 s), with there being no recovery in intramuscular metabolites until exercise was terminated. The results of this study indicate that the muscle metabolic responses and exercise tolerance during recovery from exhaustive exercise can be understood in terms of the CP concept. Indeed, the dynamics of the muscle metabolic response in recovery exercise completed immediately

after exhaustive severe intensity exercise can be differentiated according to whether the recovery exercise is performed below or above the CP. This provides further evidence to support the importance of the CP concept in determining the degree of muscle metabolic perturbation and the capacity to tolerate high-intensity exercise, and supports the notion that the capacity for $W_{>CP}$ (i.e., W') is linked to the degree of anaerobic substrate availability and muscle metabolite accumulation.

Mechanistic bases for W' recovery during high-intensity intermittent exercise

The muscle metabolic response profile (as assessed by ^{31}P-MRS) and T_{lim} during high-intensity intermittent exercise bouts were a function of the recovery interval duration. Indeed, during intermittent exercise involving work bouts in the severe-intensity domain and resting recovery intervals, muscle PCr reconstitution and metabolite clearance in the recovery interval, $W_{>CP}$ and T_{lim} were greatest for the 48 s recovery trial, intermediate for the 30 s recovery, and least for the 18 s recovery bout. Similar to the results presented in chapter 4, this study indicated that physiological responses and exercise tolerance during intermittent exercise can be understood in terms of the CP concept. Recovery intervals between severe work bouts enabled the finite W′ to be restored, with the magnitude of this reconstitution being related to the duration of the recovery interval. This study also showed that the greater increase in W′ as recovery duration was extended was accompanied by enhanced PCr reconstitution in the recovery interval such that the attainment of a 'critical' muscle PCr was delayed and the tolerable duration of exercise was enhanced. The attainment of critical levels of muscle pH, $[P_i]$ and [ADP] was also delayed as the recovery duration increased. These data suggest that the increased $W_{>CP}$ (W′), and therefore T_{lim}, as the recovery interval is extended during severe-intensity intermittent exercise is linked to changes in the muscle metabolic milieu, consistent with the CP model of bioenergetics.

The thesis set out to address two fundamental questions:

1) To what extent can the CP concept (CP and W′) explain fatigue during different types of high-intensity exercise?

2) What are the physiological bases for the W′ during different types of high-intensity exercise?

The following sections will discuss how the studies of this thesis have improved our understanding of these important questions. Potential applications of the findings and areas for further research will also be highlighted.

9.3 Exercise performance and the critical power model of bioenergetics

The CP model of bioenergetics proposes that during exercise completed above CP (the maximal sustainable rate of oxidative metabolism), the capacity limited W′ is utilized until W′ is depleted and severe-intensity exercise is terminated (Jones et al., 2010). Several studies have shown that the work completed above CP ($W_{>CP}$) during severe-intensity CWR exercise is not significantly different from the W′ derived from conventional testing procedures (Fukuba, 2003; Hill, 2002; Poole et al., 1988) or the work done above end-test power in the 3 min all-out test (Vanhatalo et al., 2011), which is equivalent to W′ (Vanhatalo et al., 2007). Consistent with these findings, data presented in Chapters 4-6 showed that $W_{>CP}$ during CWR exercise was not significantly different from the work above end-test power in the 3 min all-out test, while data presented in Chapter 7 showed that $W_{>CP}$ during CWR exercise was not significantly different from the W′ determined from 4 CWR tests to T_{lim}. However, an important aspect of this thesis was to determine whether the CP concept could help explain

performance in exercise tasks where the work rate was not held constant, which is more characteristic of work rate patterns that occur during everyday locomotion and sporting competitions.

In Chapter 5 it was shown that $W_{>CP}$ was not significantly different across incremental exercise tests that imposed a continuous-graded protocol that was self-paced according to a prescribed RPE, a ramp incremental protocol with cadence allowed to fluctuate according to subject preference, and a ramp incremental protocol with fixed cadence. Moreover, these $W_{>CP}$ values were not significantly different from $W_{>CP}$ during the 3 min all-out test or a CWR trial completed to exhaustion. These results support the findings of Morton et al. (1997) who demonstrated that $W_{>CP}$ was not significantly different between CWR and four different rates of ramp incremental exercise. However, these data conflict with Mauger and Sculthorpe (2012) who reported that a graded incremental test that was self-paced according to perception of exertion permitted the attainment of a greater peak power compared to a traditional incremental test which applied linear increments in work rate. The increased peak power achieved in the protocol of Mauger and Sculthorpe (2012) might be expected to reflect an increased $W_{>CP}$. Mauger and Sculthorpe (2012) suggested that their protocol provided subjects with the ability to vary their power output for a given RPE, enabling better management of afferent signallers (which may include the accumulation of fatigue-related muscle metabolites, and changes in motor unit recruitment, core temperature and pulmonary ventilation) and thereby reducing the perception of pain and discomfort. However, a potential confounding factor in that study was test duration, which was different between the self-paced test (~10 min) and the conventional incremental test (~13 min). It is known that the duration of an incremental test can influence the measured peak work rate (Astorino, 2004; Yoon, 2007). To address this concern,

Chapter 5 administered a self-paced perceptually regulated incremental test based on the methods of Mauger and Sculthorpe (2012), but matched the test duration to that attained in a preliminary ramp incremental test. When the duration of the self-paced perceptually regulated incremental test was matched to the ramp incremental test, there was no difference in $W_{>CP}$ across the protocols. Moreover, the $W_{>CP}$ was also not different compared to a ramp incremental protocol with cadence allowed to fluctuate according to subject preference, a high-intensity constant-power-output test to the limit of tolerance and a 3-min all-out test. This supports the CP model by demonstrating that a fixed $W_{>CP}$ can be completed prior to exhaustion during continuous exercise, and suggests that the CP model also has application in exhaustion incremental exercise.

Since self-paced exercise has been reported to reduce metabolic stress compared to CWR exercise (Lander et al., 2009), self paced exercise might be expected to increase $W_{>CP}$. This was investigated in Chapter 6 where it was shown that $W_{>CP}$ was not significantly different during a 3 min all-out cycling test, a constant work rate bout predicted to lead to exhaustion in 3 min and a self-paced 3 min work-trial. Therefore, irrespective of the method of work rate imposition, and thus the rate at which W' was depleted, $W_{>CP}$ was not significantly different across the exercise tests at end-exercise. It has been shown previously that $W_{>CP}$ is similar in duration-matched all-out exercise and CWR exercise (Vanhatalo et al., 2011), but the data presented in Chapter 6 extend this by showing that $W_{>CP}$ is also consistent across duration-matched CWR, all-out and self-paced exercise. Taken together with the findings from Chapter 5, these data suggest that the $W_{>CP}$ during continuous exercise is fixed, being similar across CWR, all-out, self-paced and incremental exercise. These data support the critical power concept as a model to explain fatigue during exercise across a variety of work rate forcing functions.

While the data presented in Chapters 5 and 6 suggest that the CP model can explain exercise performance during continuous exercise (a fixed $W_{>CP}$ is completed at end-exercise), the extent to which the CP model can explain performance during intermittent exercise is complicated by the potential for W′ recharge in the recovery intervals. In an attempt to account for this W′ resynthesis, Morton and Billat (2004) adapted the two-parameter CP model for intermittent exercise by including four independent variables (*P* and *t* during both work and recovery phases) during intermittent exercise (see *Equation 7*). Consistent with the findings of Morton and Billat (2004), the findings in Chapter 4 showed that the W′ was increased and CP was reduced during intermittent exercise compared to the 3 min all-out test. The greater W′ is likely reflective of W′ resynthesis in the recovery intervals during the intermittent exercise and it was shown that rate of W′ resynthesis, and thus the potential to tolerate supra-CP work, was greater as the intensity of the recovery interval was lowered. Importantly, when both the work and recovery intervals of the intermittent exercise protocol were in the severe-intensity exercise domain $W_{>CP}$ was not significantly different from $W_{>CP}$ in the severe-intensity CWR test to T_{lim} or the W′ measured in the 3 min all-out test. In the context of the CP model these results demonstrate that, while W′ is fixed and completely expended at the point of fatigue during severe-intensity CWR exercise or intermittent exercise conducted exclusively within the severe-intensity exercise domain, W′ can be resynthesised in an intensity-dependent manner when the recovery work rate falls below CP and this dictates the tolerable duration of intermittent exercise. This is supported by the results presented in Chapter 7 where the capacity to complete $W_{>CP}$ immediately after exhaustive severe-intensity exercise was very limited (39 ± 31 s) when the recovery work rate was set above CP, but was appreciable (all subjects could exercise for at least 10 min) when the recovery work rate was set below CP. Therefore, the CP

concept can also help explain the tolerable duration of intermittent exercise when different recovery exercise intensities are applied.

As well as the exercise intensity of the recovery interval during intermittent exercise, the recovery duration would be expected to influence the rate of W′ reconstitution and, therefore, the tolerable duration of intermittent exercise. This was addressed in Chapter 8 of the thesis. Here, it was shown that W′ increased as the recovery interval was extended and exercise tolerance was enhanced in line with the increase in W′. Taken together, the findings from Chapters 4 and 7-8 indicate that the CP concept can also help explain the tolerable duration of intermittent exercise as exercise tolerance during intermittent appears to be linked to the extent of W′ recharge in the recovery intervals and therefore, the potential to complete $W_{>CP}$ in the work intervals.

9.4 Mechanistic bases for the W′

The results from this thesis have shown that regardless of how work rate is administered, W′ is closely linked to fatigue during high-intensity exercise, consistent with the CP model of bioenergetics. However, while the utilisation of W′ is an important determinant of exercise performance, the mechanistic bases of W′ are not completely understood and it is also unclear whether the physiological corollaries of W′ depletion are similar across fatiguing exercise applying different work rate forcing functions. To explore the mechanistic bases for W′, pulmonary gas exchange was measured to provide a non-invasive measure of muscle $\dot{V}o_2$ (Krustrup et al., 2009), iEMG was measured to provide an indication of muscle activation and ^{31}P-MRS was utilised to assess intramuscular phosphorous metabolites and pH.

In Chapter 5 it was shown that $W_{>CP}$ was not significantly different during a continuous-graded protocol that was self-paced according to a prescribed RPE, a ramp incremental protocol with cadence allowed to fluctuate according to subject preference, a ramp incremental protocol with fixed cadence, a high-intensity constant-power-output test to the limit of tolerance, and a 3-min all-out test during which power output declined precipitously over time. Similarly, $\dot{V}o_{2max}$ was not significantly different across these exercise tests. While this conflicts with the recent findings of Mauger and Sculthorpe (2012) who reported that $\dot{V}o_{2max}$ could be increased during a self-paced perceptually-regulated exercise test, it supports the longstanding view that $\dot{V}o_{2max}$ for a given mode of exercise cannot be exceeded (Hill and Lupton, 1923; Bassett and Howley, 2000; Wagner, 2000; Gonzalez-Alonso and Calbet, 2003; Saltin and Calbet, 2006; Hawkins et al., 2007). As outlined earlier, the fact that test duration was not matched in the self-paced (~10 min) and the conventional incremental (~13 min) test might account for the higher $\dot{V}o_{2max}$ in the self-paced test used by Mauger and Sculthorpe (2012) as the peak power output that can be elicited might be lower as the test duration is extended leading to the attainment of a submaximal rather than a maximal $\dot{V}o_2$ (Astorino, 2004; Yoon, 2007). The $W_{>CP}$ and $\dot{V}o_{2max}$ were also not significantly different during a 3 min all-out cycling test, a constant work rate bout predicted to lead to exhaustion in 3 min and a self-paced 3 min work-trial, as reported in Chapter 6. While it has been reported that W′ depletion and the attainment of the $\dot{V}o_{2max}$ occur simultaneously during severe-intensity CWR exercise (Burnley and Jones, 2007; Ferguson et al., 2007; Jones et al., 2010; Murgatroyd et al., 2011; Vanhatalo et al., 2011), the findings presented in Chapters 5 and 6 extend this by showing that complete depletion of W′ and the attainment of $\dot{V}o_{2max}$ are also concomitant during incremental and self paced exercise.

During the intermittent exercise protocols employed in Chapter 4, end-exercise $\dot{V}o_2$ was not significantly different from the $\dot{V}o_{2max}$ attained a ramp incremental test and the severe-intensity CWR bout continued until the limit of tolerance despite the fact that W′ was increased as exercise intensity in the recovery intervals was lowered. It is likely that total W′ was increased as the intensity of the recovery interval was lowered because W′ recovered at a rate dependent on the intensity of the recovery bouts. In turn, the enhanced W′ resynthesis as the intensity of the recovery interval was lowered delayed the complete depletion of W′ (see Figure 4, Chapter 4). Simultaneously, the $\dot{V}o_2$ slope was progressively lowered as the recovery work rate declined below CP leading to a delayed attainment of $\dot{V}o_{2max}$. Therefore while complete W′ depletion and attainment of $\dot{V}o_{2max}$ is progressively delayed during intermittent exercise as the intensity of the recovery interval is lowered, W′ and $\dot{V}o_2$ reach their capacity concomitantly during intermittent exercise as well as CWR, incremental and self paced exercise.

The blunted rate of W′ depletion and lower $\dot{V}o_2$ slope was accompanied by a blunted increase in muscle activation as evidence by the lower iEMG slope. This suggests that the volume of muscle engaged or the firing frequency of the recruited muscle fibres might be linked to W′ depletion and the attainment of the $\dot{V}o_{2max}$. Previous studies have also shown that iEMG measures are linked to the development of the $\dot{V}o_2$ slow component (Shinohara and Moritani, 1996; Saunders et al., 2000; Borrani et al., 2001; Perry et al., 2001; Burnley et al., 2002; Osborne and Schneider, 2006; DiMenna et al., 2008; Layec et al., 2009; DiMenna et al., 2010) and the degree of muscle metabolic perturbation (Bailey et al., 2010). To further explore the mechanistic bases for W′ depletion we used ^{31}P-MRS in Chapter 7 to investigate the muscle metabolic response when work rate was lowered to the heavy-intensity (<CP) or to a lower work rate in the severe-intensity (>CP) domain immediately following the completion of a severe-

intensity work rate predicted to elicit T_{lim} in 3 min. It was shown that muscle [PCr], pH, [ADP] and [P_i] remained at the levels reached in the initial exhaustive exercise bout and the capacity for $W_{>CP}$ was severely restricted when the recovery work rate was in the severe-intensity exercise domain. However when the recovery work rate was lowered below CP, muscle [PCr], pH, [ADP] and [P_i] returned towards baseline values and exercise could be tolerated for at least 10 min. These results are consistent with the study of Coats et al. (2003). These authors asked subjects to perform severe CWR cycle exercise to T_{lim} (attained in around 6 min) and then reduced the work rate to 80 % GET, 90 % CP, or 110 % CP. The results suggested that replenishment of the W′ following T_{lim} necessitated that work rate be reduced <CP. The data presented in Chapter 7 support the findings of Coats et al. (2003), but extend these results by showing that T_{lim} following exhaustive exercise is critically dependent on whether muscle metabolic homeostasis can be restored or not.

The muscle metabolic responses were investigated during intermittent exercise comprising severe-intensity work bouts interspersed with resting recovery bouts of different durations (18, 30 and 48 s) in Chapter 8. It was shown that T_{lim} and $W_{>CP}$ were progressively increased as the recovery duration was extended in association with a delayed attainment of critical levels of muscle [PCr], pH, [ADP] and [P_i]. Therefore the mechanistic studies conducted in Chapters 7 and 8 revealed that the metabolic milieu within the muscle influences the capacity for $W_{>CP}$ with consistently low [PCr] and pH, and consistently high [ADP] and [P_i] attained at the point of complete W′ depletion and task failure.

When the data across the 5 experimental Chapters are considered together it appears that, irrespective of the work rate forcing function, fatigue during high-intensity

exercise occurs when W′ is completely depleted, $\dot{V}o_{2max}$ is attained and critical levels of intramuscular [PCr], pH, [ADP] and [P_i] are achieved. These findings support previous results regarding the mechanistic bases for the W′ (Jones et al., 2010), but extend these findings by showing that the mechanistic bases for the W′ are consistent across exercise protocols that use different work rate forcing functions. From the results presented in this thesis and published elsewhere, it appears that intense exercise leads to a progressive decline in muscle PCr and a significant increase in muscle ADP and P_i, which are important stimuli for activating oxidative metabolism in the mitochondria (Chance and Williams, 1955; Brown, 1992; Bose et al., 2003). Indeed, the changes in PCr, ADP and P_i are temporally aligned with the development of the $\dot{V}o_2$ slow component during intense exercise (Bailey et al., 2010; DiMenna et al., 2010; Rossiter et al., 2001, 2002). As severe-intensity exercise is continued, further increases in muscle ADP and P_i and a continued decline in PCr amplify the signal for oxidative metabolism augmenting the $\dot{V}o_2$ slow component. Eventually, muscle [ADP], [P_i], [PCr] and pH reach critical levels that might compromise muscle contraction (Allen et al., 2008). At this point, the metabolic stimulus for oxidative metabolism is maximal such that $\dot{V}o_{2max}$ is attained and W′ is completely expended. Therefore, the results presented in this thesis offer important mechanistic insights into the physiological bases for W′ and suggest that W′ depletion and the attainment of $\dot{V}o_{2max}$ and critical levels of muscle [ADP], [P_i], [PCr] and pH are inter-related and occur concomitantly. This sequence of events leads to additional $W_{>CP}$ becoming untenable, consistent with the CP model of bioenergetics, and task failure occurs irrespective of the method of work rate imposition.

9.5 Applications

The original findings from this thesis suggest that the degree of metabolic perturbation and performance across a range of exercise tasks can be explained by the CP model of bioenergetics. Therefore, the CP model might be able to aid the construction of effective interval training programs (i.e. intensity and duration of the work bouts and recovery intervals) for the enhancement of fitness, functional capacity or cardiovascular and metabolic health (Earnest, 2009; Gibala and McGee, 2008; Laursen and Jenkins, 2002). This information might also help athletes to select pacing strategy that optimise the use of W′ and therefore performances during endurance exercise (Skiba et al., 2012). The data presented in this thesis provide strong support for the use of the CP model to predict exercise performance in humans. The ability to use CP and W′ to accurately T_{lim} during different types of exercise is very useful to sports scientist doing laboratory or field studies.

9.6 Topics for Further Research

Elderly and clinical populations

Most studies investigating the CP model have shown it to be an effective predictor of metabolic perturbation and exercise tolerance in healthy adult humans. It is well documented that ageing and various metabolic, cardiovascular and respiratory diseases lead to exercise intolerance, but it is unclear whether these changes in exercise tolerance can be explained by the CP model. This information might be useful for the development of therapeutic interventions to improve exercise tolerance and therefore quality of life in these populations.

Mechanistic bases for W′

While this thesis has extended our understanding of the factors that contribute to W′ by showing that depletion of the W′ is accompanied by attainment of the $\dot{V}_{O2max}$ and critical intramuscular levels of [PCr], pH, [P_i] and [ADP] and coincides with the termination of high-intensity exercise tests when different patterns of work rate imposition are imposed, additional research is required to increase this understanding further. For example, other muscle metabolites including potassium, calcium, ammonia and reactive oxygen species have also been linked to the process of muscle fatigue (Allen et al., 2008; Amann, 2011), but it is unclear whether changes in these metabolites differ <CP compared to >CP.

9.7 Conclusion

The tolerable duration of severe-intensity CWR exercise can be predicted using the CP model of bioenergetics. Based on the CP model, W′ is utilised when exercising at a CWR above CP until W′ is completely depleted and exercise is terminated (e.g., Vanhatalo et al., 2011). Coincident with the depletion of W′, $\dot{V}_{O2max}$ is attained and a critical level of muscle metabolic perturbation is reached (Jones et al., 2008; Poole et al., 1988; Vanhatalo et al., 2010). The findings of this thesis extend our understanding of the CP model by showing that it can help explain metabolic perturbation and performance across a variety of exercise tests using different work rate forcing functions. In addition, the results presented in this thesis support the notion that W′ depletion occurs in concert with the increased $\dot{V}_{O2}$, the depletion of the finite anaerobic reserves and the attainment of critical levels of intramuscular metabolites, as evidenced by the attainment of $\dot{V}_{O2max}$, critically low muscle [PCr] and pH, and critically high [P_i] and [ADP] at T_{lim} during high-intensity exercise, irrespective of the work rate distribution during exercise.

It was shown that a ramp incremental test at a fixed cadence, a ramp incremental test where the subject could self-select cadence and a step incremental test where subjects selected power output according to prescribed increments in ratings of perceived exertion resulted in consistent $\dot{V}_{O2max}$ and $W_{>CP}$ values. Similarly, $\dot{V}_{O2max}$ and $W_{>CP}$ were not significantly different between a 3 min all-out cycling test, a constant work rate bout predicted to lead to exhaustion in 3 min and a self-paced 3 min work-trial. Taken together, these data indicate that a consistent $\dot{V}_{O2max}$ and $W_{>CP}$ are achieved at exhaustion during severe-intensity exercise irrespective of the method of work rate imposition. These findings are consistent with the CP model of bioenergetics and with the reciprocal relationship between W′ and the $\dot{V}_{O2}$ slow component.

While the $W_{>CP}$ was not significantly different from the W′ determined in the 3 min all-out test when the work and recovery components of an intermittent exercise protocol were >CP, $W_{>CP}$ was increased above W′ and T_{lim} was improved in an intensity-dependent manner when the recovery work rate was reduced below CP. The progressive increase in $W_{>CP}$ as recovery intensity was lowered below CP was accompanied by blunted rates of $\dot{V}_{O2}$ and iEMG increase across the intermittent exercise bouts. When the dynamics of muscle metabolic responses were assessed *in vivo* using ^{31}P-MRS, muscle [PCr], pH, [P_i] and [ADP] remained at critical levels and exercise tolerance was significantly impaired following exhaustive exercise when 'recovery' exercise was completed in the severe-intensity domain (>CP); however, muscle [PCr], pH, [P_i] and [ADP] were restored towards baseline values and the tolerable duration of exercise was extended to at least 10 min when the recovery work rate resided in the heavy-intensity domain (<CP). It was also shown that, during severe-intensity intermittent exercise with variable recovery durations, $W_{>CP}$ and T_{lim} were increased as the duration of the recovery interval was extended and this was accompanied by a delay

in the attainment of critical intramuscular metabolites ([PCr], pH, [ADP] and [P_i]). These data suggest that W′ can be recovered at a rate that is dependent on the intensity and duration of the recovery interval during intermittent exercise and these influences the tolerable duration of exercise and the rate at which $\dot{V}_{O2max}$ and critical levels of muscle [PCr], pH, [ADP] and [P_i] are attained.

In summary, this thesis has extended understanding of the CP concept by showing that the CP model can also help explain exercise performance during methods of work rate imposition other than CWR exercise. It has been shown that task failure during incremental, CWR and self-paced exercise occurs at a consistent $W_{>CP}$ (W′) and $\dot{V}_{O2max}$. The $W_{>CP}$ completed during intermittent exercise tests is associated with the tolerable duration of exercise with $W_{>CP}$ increased as the intensity of the recovery interval is lowered or the recovery duration is increased. The rate at which $\dot{V}_{O2max}$ is attained and muscle [PCr], pH, [ADP] and [P_i] reach levels that perturb the muscle milieu is also a function of the intensity and duration of the recovery interval during intermittent exercise. These data support the association between W′ depletion and the degree of metabolic perturbation. Therefore, the findings presented in this thesis suggest that fatigue across a diverse range of exercise tests occurs when W′ is depleted, $\dot{V}_{O2max}$ is attained and critical levels of intramuscular [PCr], pH, [ADP] and [P_i] are reached, consistent with the CP model of human bioenergetics. It is recommended that the CP model be applied more widely in basic and applied studies of exercise physiology and its implications communicated to coaches and athletes for the purposes of informing training prescription.

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