The Influence of Aerobic Fitness on the Recovery of Peak Power Output

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ABSTRACT

PURPOSE: The aims of this study were to evaluate the recovery kinetics of peak power output (PPO) following a maximal sprint, and to evaluate the influence of aerobic fitness on that recovery process. METHODS: On separate occasions 16 well-trained men (age: 21 ± 3 years; height: 1.84 ± 0.05 m; and body mass: 78.8 ± 7.8 kg) performed a 30 s maximal sprint on a cycle ergometer, followed by a predetermined stationary rest period (5, 10, 20, 40, 80, and 160 s) and a subsequent 5 s sprint to determine PPO recovery kinetics. On another occasion, $\dot{V}O_2$ was monitored during recovery from a 30 s sprint to provide a comparison with the recovery of PPO. Finally, subjects completed a $\dot{V}O_{2max}$ test to evaluate the influence of aerobic fitness on the recovery of PPO. RESULTS: Despite following similar time courses ($F = 0.36$, $p = 0.558$), and being well described by double-exponential models, the kinetic parameters of PPO and $\dot{V}O_2$ in recovery were significantly different ($p < 0.05$). There was no significant relationship ($r = 0.15$; $p = 0.578$) between $\dot{V}O_{2max}$ and the time to achieve 50% recovery of PPO. Moreover, there was no difference ($p = 0.61$) between the recovery kinetics of participants classified according to their $\dot{V}O_{2max}$ (59.4 ± 1.3 vs 48.5 ± 2.2 ml·kg^{-1}·min^{-1}). CONCLUSION: Despite similar overall recovery kinetics, $\dot{V}O_2$ and PPO show differences in key model parameters. Moreover, the recovery of PPO does not appear to be affected by aerobic fitness.

Key words: Kinetics, fatigue, maximal oxygen uptake, homeostasis.

Abbreviations

ANOVA: Analysis of variance

GET: Gas exchange threshold

PCr: Phosphocreatine

PPO: Peak power output

$\dot{V}O_2$: Rate of oxygen uptake

$\dot{V}O_{2max}$: Maximal rate of oxygen uptake

INTRODUCTION

The recovery of peak power output (PPO) after exercise has been shown to have similar kinetics to those of phosphocreatine concentration ([PCr]) (Bogdanis et al. 1995). Moreover, the rate of [PCr] resynthesis is reported to follow the same time course as that of oxygen uptake ($\dot{V}O_2$), at least following exercise at intensities below those required to elicit $\dot{V}O_{2max}$ (Barker et al. 2008a; Rossiter et al. 2002). Indeed, the restriction of oxygen availability after exercise (via blood flow occlusion) has been shown to prevent [PCr] resynthesis (Yoshida and Watari 1997). Since there is evidence that the recovery kinetics of $\dot{V}O_2$ and [PCr] are faster after a period of endurance training (Forbes et al. 2008; Fukuoka et al. 2002, 2006; Takahashi et al. 1995; Yoshida 2002), it seems reasonable to assume that the recovery of [PCr], and therefore power output, will be fastest in individuals with higher levels of aerobic conditioning. Indeed, it is generally accepted that trained athletes, regardless of their discipline, possess a greater level of aerobic fitness than sedentary individuals and as a result are able to cope better with the demands of interval training and multiple-sprint sports, where the ability to recover quickly between exercise bouts is important for success (refer to Tomlin and Wenger 2001). However, studies that have investigated the relationship between $\dot{V}O_{2max}$ and fatigue (measured by the ability to maintain performance across repeated sprints) in multiple-sprint tests, have proved equivocal, with reported correlations ranging from $r = -0.16$ to $r = -0.83$ (Aziz et al. 2000; Dupont et al. 2010; Glaister, 2008). Although such discrepancies could be the result of sample homogeneity, studies investigating the effects of endurance training on multiple-sprint performance have failed to show a significant reduction in fatigue, despite inducing significant increases (5−12%) in $\dot{V}O_{2max}$ (Edge et al. 2005; Glaister et al. 2007). Conversely, manipulations of oxygen delivery (via erythropoietin

administration and hypoxia) have been shown to influence physiological and performance responses to multiple-sprint work (Balsom et al. 1994a, 1994b); moreover, endurance-trained athletes have been shown to display less fatigue in multiple-sprint tests than games-sport players (Bishop and Spencer 2004; Hamilton et al. 1991).

One of the main problems when examining the link between aerobic fitness and the recovery of power output is the quantification of the recovery kinetics of power output. Unlike physiological responses, which can be monitored at various time points during recovery from a single exercise bout, the recovery of power output requires a series of trials in which the rest interval between an initial and subsequent exercise bout is manipulated to enable the recovery process to be modelled. Though a few studies have monitored the time course of power output in recovery, none have modelled the kinetics of the process. Therefore, the aims of this study were: first, to compare the recovery kinetics of power output with those of $\dot{V}O_2$; and secondly, to evaluate the relationship between $\dot{V}O_{2max}$ and the recovery of power output.

METHODS

Subjects

16 well-trained men volunteered for the study which was approved by St Mary's University Ethics Committee. Prior to testing, subjects received written and verbal instructions regarding the nature of the investigation and completed a training history questionnaire, which indicated that all had been actively involved in sport for approximately 13 years. Times spent training and competing each week were reported as 9.0 ± 5.0 hours and 5.3 ± 3.8 hours, respectively. Prior to commencement of the

study, all subjects completed a health-screening questionnaire and provided written informed consent. Means ± standard deviation for age, height, body mass, body fat (Durnin and Womersley 1974), and $\dot{V}O_{2max}$ were: 21 ± 3 years, 1.84 ± 0.05 m, 78.8 ± 7.8 kg, 10.8 ± 2.0%, and 54.3 ± 4.5 ml·kg^{-1}·min^{-1} respectively.

Experimental Overview

During the period of investigation, subjects completed eleven trials, at approximately the same time of day, with a minimum of 24 hours between each. All trials were completed in an air-conditioned laboratory maintained at a constant temperature of 18°C. Trial 1 was a baseline trial to establish PPO during a 5 s sprint and to familiarise subjects with the equipment and the demands of a 30 s maximal cycle sprint test. Trials 2 − 7 were performed in a randomised order and involved subjects performing a 30 s maximal sprint followed by a predetermined stationary rest period (5, 10, 20, 40, 80, or 160 s) and a subsequent 5 s sprint to determine the recovery of PPO. Trial 8 consisted of a 30 s sprint followed by a passive recovery period during which subjects were asked to indicate the time point at which they perceived they had recovered sufficiently to perform a 5 s sprint to the best of their ability. Trial 9 was a repeat of the protocol used in trials 2-7, except that the passive recovery period used was the same as that determined in Trial 8. Trial 10 involved the monitoring of $\dot{V}O_2$ during recovery from a 30 s sprint to enable the recovery patterns of $\dot{V}O_2$ and PPO to be compared. Trial 11 was a $\dot{V}O_{2max}$ test to evaluate the influence of this parameter on the kinetics of power output in recovery.

Equipment

All sprints were performed on an electro-magnetically braked cycle ergometer (Lode Excaliber Sport, Groningen, Holland), which was fitted with standard pedals, toe-clips, and straps, and interfaced with a computer to enable high-frequency logging of the flywheel angular velocity. Oxygen uptake was determined from expired air (breath-by-breath) using an on-line gas analyser (Jaeger Oxycon Pro, Hoechberg, Germany). The analyser was calibrated before each test using oxygen and carbon dioxide gases of known concentrations (Cryoservice, Worcester, UK) and the flowmeter was calibrated using a 3-litre syringe (Viasys Healthcare GmbH, Hoechberg, Germany). During the tests subjects breathed room air through a facemask (Hans Rudolph, Kansas City, MO, USA) that was secured in place by a head-cap assembly (Hans Rudolph, Kansas City, MO, USA).

Procedures

Trial 1

On arrival at the laboratory, height, body mass, and body fat (determined from the sum of four skinfolds) were recorded for each subject. Subjects then performed a four-minute warm-up on the cycle ergometer at a power output of 100 W. The same warm-up procedure was used for all trials. The saddle height and handlebar position for each subject were determined before the first trial and remained constant for all subsequent trials. On completion of the warm-up and starting from a stationary position, subjects performed a series of 3×5 s maximal cycle sprints interspersed with stationary rest periods of 3 minutes to determine individual measures of PPO. A torque factor of 0.7 N·m·kg bm^{-1} was used for all trials and subjects were encouraged to give maximal effort. On completion of the third sprint, subjects cycled for a further three minutes at a power output of 100 W before performing a 30 s maximal cycle

sprint for familiarisation purposes. After all trials, subjects completed a cool-down by cycling at 100 W for a minimum of five minutes.

Trials 2 – 7

After the warm-up, and from a rolling starting power output of 100 W, subjects completed a 30 s maximal sprint. On completion of the sprint, subjects were instructed to remain stationary on the ergometer for a period of between 5 s and 160 s before performing a second 5 s maximal sprint. Information on the duration of the recovery period was withheld from the subject in every trial and the computer screen was obscured from view. Since it was anticipated that the recovery of PPO would follow a bi-phasic pattern (Bogdanis et al. 1995; Cherry et al. 1998) the following recovery periods were used: 5 s, 10 s, 20 s, 40 s, 80 s, and 160 s.

Trials 8 and 9

In trials 8 and 9, participants followed the same procedure as in trials 2 – 7 up to the point at which they completed the 30 s sprint. In Trial 8, on completion of the 30 s sprint, participants remained stationary on the ergometer and were asked to indicate at what point in the recovery process they felt they had fully recovered their ability to perform a 5 s sprint. In Trial 9, participants completed the same procedure as in trials 2-7, although the 5 s sprint was performed at the time point in recovery at which they previously perceived (Trial 8) they had fully recovered.

Trial 10

In Trial 10, following the fitting of the face mask and headgear, subjects were asked to remain stationary on the ergometer for a period of three minutes to enable baseline

measurements of $\dot{V}O_2$ to be recorded. After a further four-minute warm-up period, subjects performed a 30 s maximal sprint followed by a five-minute stationary recovery period during which $\dot{V}O_2$ was recorded.

Trial 11

In Trial 11, following the four-minute warm-up, subjects completed a graded exercise test, which commenced at a power output sufficient to achieve a protocol duration of 8 to 15 minutes. Every minute during the test, power output was increased by 20 W until subjects reached volitional exhaustion. During the tests respiratory gases were monitored breath-by-breath using the on-line gas analyser. $\dot{V}O_{2max}$ was determined as the highest 30 s average $\dot{V}O_2$ observed during the test provided that at least two of the following criteria had been met: 1) A plateau in $\dot{V}O_2$; as determined by an increase of less than 2 ml·kg^{-1}·min^{-1} over the previous stage; 2) A respiratory exchange ratio $\geq$ 1.15; 3) A heart rate within 10 b·min^{-1} of age predicted maximum; 4) A blood lactate concentration $\geq$ 8 mmol·L^{-1}.

Data Analysis

Synchronisation of the $\dot{V}O_2$ data between subjects was achieved using linear interpolation at 5 s intervals throughout recovery after eliminating values that were outside four standard deviations of the midpoint of the local mean (attributed to 'noise') (Rossiter et al. 2002). To account for the influence of muscle-lung transit times on recovery kinetics (estimated at 6 s immediately after exercise at 50% of the difference between the gas exchange threshold [GET] and $\dot{V}O_{2max}$, and likely to be appreciably shorter immediately after exercise of the intensity used in the present investigation [Krustrup et al., 2009]), the first 5 s of the $\dot{V}O_2$ response were omitted

from the subsequent analysis. Recovery $\dot{V}O_2$ data were subsequently converted to percentages, with values 5 s after the end of the 30 s sprint used as the reference point for zero recovery, and with mean resting values from the start of Trial 10 used as the reference for full recovery. The recovery of PPO was also determined as percentage data, with the highest PPO from the 5 s sprints in Trial 1 considered as the reference for full recovery. Mono (Eq. 1) and double-exponential (Eq. 2) models were applied to characterise the recovery kinetics of $\dot{V}O_2$ for each individual using a non-linear least-squares fitting procedure (XLfit, IDBS Ltd, Guildford, UK).

$$\text{Recovery (t)} = \text{Recovery}_{(end)} - (A_1 \times \exp^{-(t-td_1/\tau_1)}) \tag{1}$$

$$\text{Recovery (t)} = \text{Recovery}_{(end)} - (A_1 \times \exp^{-(t-td_1/\tau_1)}) + (A_2 \times \exp^{-((t-td_2)/\tau_2)}) \tag{2}$$

Where: 'Recovery$_{(end)}$' is the projected value at the end of recovery and was constrained at $\leq 100\%$; A_1 and A_2 are the amplitudes of the first and second-order responses respectively; τ_1 and τ_2 are the time constants of each exponential; td_1 and td_2 are the time delays for the first and second-order responses respectively. The same modelling approach as above was used for the recovery kinetics of PPO. The time to achieve 50% of full recovery was also determined from each model.

Although the primary purpose of Trial 11 was to determine $\dot{V}O_{2max}$; gas exchange parameters also enabled the GET for each individual to be evaluated from visual inspection of the $\dot{V}CO_2 - \dot{V}O_2$ relationship using the V−slope method (Beaver et al. 1986); thereby potentially providing a better indication of intramuscular oxidative capability.

Statistics

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS for Windows, SPSS Inc., Chicago, IL). Measures of centrality and spread are presented as means ± standard deviation. Goodness of fit of the exponential models was evaluated using F tests. Differences between the recovery kinetics of PPO and $\dot{V}O_2$ were evaluated by using paired t-tests to contrast key modelling parameters. Differences between the recovery of power output and the recovery of $\dot{V}O_2$ were also evaluated using a two-way (variable × time) ANOVA with repeated measures on both factors. Significant effects were followed up using Bonferroni-adjusted *post hoc* analyses. The influence of $\dot{V}O_{2max}$ on the recovery of power output was examined in two ways: first, power output data from the five subjects with the highest $\dot{V}O_{2max}$ were compared with those with the lowest using a two-way (group × time) mixed ANOVA; secondly, the relationship between aerobic fitness parameters ($\dot{V}O_{2max}$ and GET) and the time to achieve 50% recovery of PPO (predicted from the modelling process) was evaluated using Pearson correlations. Unpaired t-tests on key modelling parameters were used to evaluate between-group differences in $\dot{V}O_2$ recovery kinetics. Finally, the potential influence of PPO on subsequent PPO recovery kinetics was evaluated by correlating PPO with the half time of the recovery process. α was set at 0.05 for all analyses.

RESULTS

Peak power output of the subjects (determined from the 5 s sprints in Trial 1) was 1075 ± 177 W. Despite subject perceptions of achieving full recovery of PPO in the trials, only 6 of the participants came within 95% of that value, and the mean maximum value for the group was 92.1 ± 13.0%. The recovery patterns of PPO and

$\dot{V}O_2$ are presented in Figure 1. Analysis of the data revealed no significant effect of variable (PPO vs $\dot{V}O_2$) ($F_{(1,15)} = 0.36$, $p = 0.56$); there was, however, a significant effect of time ($F_{(5,75)} = 394.16$, $p < 0.001$), and a significant variable × time interaction ($F_{(5,75)} = 2.76$, $p = 0.02$). *Post hoc* tests were unable to identify where those differences lay (Figure 1). The recovery kinetics of $\dot{V}O_2$ were very well described by both single (mean *F*-test score: 1.024 ± 0.008) and double-exponential (mean *F*-test score: 1.023 ± 0.008; Figure 2) models; with a small, but significantly ($p < 0.05$) better fit attributed to the latter. The recovery of PPO was also well described by single (mean *F*-test score: 1.06 ± 0.04) and double-exponential (mean *F*-test score: 1.05 ± 0.04; Figure 3) models, though the goodness-of-fit showed greater between-subject variability. Once again, the double-exponential model provided a small but significantly ($p < 0.05$) better fit to the data. The double-exponential modelling process gave no clear evidence of a second-order delay response; therefore, models were developed without those delays. Predicted times to achieve 50% recovery were not significantly different ($p = 0.70$) between PPO (34.9 ± 15.3 s) and $\dot{V}O_2$ (36.7 ± 8.9 s). There were, however, significant differences between variables in the amplitudes and time constants used to describe the recovery kinetics (Table 1).

Descriptive statistics for participants classified in the 'high' and 'low' $\dot{V}O_{2max}$ groups (all participants having met the criteria required for achieving $\dot{V}O_{2max}$) are presented in Table 2, with corresponding patterns for the recovery of PPO presented in Figure 4. Despite significant differences in $\dot{V}O_{2max}$ and GET, there were no corresponding between-group differences in any of the key kinetic parameters for the recovery of $\dot{V}O_2$ (see Table 3). Analysis of the results revealed a significant effect of time ($F_{(5,40)} = 78.49$, $p < 0.001$), but no significant difference between groups ($F_{(1,8)} = 0.29$, $p = $

0.61), and no group × time interaction ($F_{(5,40)} = 0.34$, $p = 0.89$). The time to achieve 50% recovery of PPO was not significantly correlated with $\dot{V}O_{2max}$ ($r = 0.15$; $p = 0.58$), GET (which occurred at a $\dot{V}O_2$ of 31.1 ± 5.6 ml·kg^{-1}·min^{-1}) ($r = -0.23$; $p = 0.39$), or PPO ($r = 0.27$; $p = 0.31$).

DISCUSSION

The aim of this study was to evaluate the recovery kinetics of PPO following a maximal sprint and to compare the resultant responses with: a) the recovery kinetics of $\dot{V}O_2$; and b) one of the key markers of aerobic fitness, namely $\dot{V}O_{2max}$. The main findings were that the overall recovery kinetics of PPO and $\dot{V}O_2$, though similar, and well described by double-exponential models, displayed significant differences in key model parameters. Moreover, there was no significant relationship between $\dot{V}O_{2max}$ and the recovery of PPO.

The observation that the recovery kinetics of $\dot{V}O_2$ were best described by a double-exponential model is in agreement with previous reports, at least when evaluated after high-intensity exercise (Dupont et al. 2010; Özyenor et al. 2001). However, previous research has evaluated $\dot{V}O_2$ in recovery only after exercise at intensities sufficient to elicit up to approximately 120% of $\dot{V}O_{2max}$ (most at < $\dot{V}O_{2max}$). Since excess post-exercise oxygen consumption displays a curvilinear relationship with exercise intensity (Bahr 1992; Børsheim and Bahr 2003), and since the early phase of that recovery is associated with processes other than just the replenishment of [PCr] (restoration of oxymyoglobin stores, metabolism of lactate, and metabolic effects associated with elevations in core temperature, pulmonary ventilation, and cardiac work; Børsheim and Bahr 2003), it is not surprising that Krustrup et al. (2009)

recently reported a dissociation between muscle and pulmonary $\dot{V}O_2$ recovery kinetics; with slower kinetics of the latter reflecting the demand for oxygen to promote recovery in areas other than the active musculature. Nevertheless, given the close association (r = 0.71-0.86) between [PCr] resynthesis and the recovery of PPO (Bogdanis et al. 1995), these results (Krustrup et al. 2009) appear to be at odds with previous research which has suggested a tight coupling between $\dot{V}O_2$ and [PCr] recovery kinetics (Barker et al. 2008a; Rossiter et al. 2002). Whilst it is possible that the lower exercise intensities ($< \dot{V}O_{2max}$) used by Barker et al. (2008a); and Rossiter et al. (2002) may have been insufficient to induce a level of physiological and metabolic stress to elicit a mismatch between intramuscular and whole-body oxygen demands in recovery, it is difficult to reconcile this with the research of Krustrup et al. (2009), which used a similar exercise intensity. Overall, while the ability to recover quickly is important for athletes participating in multiple-sprint sports or interval training, the difficulties associated with measuring recovery have largely confined investigations to recovery from submaximal ($< \dot{V}O_{2max}$) exercise intensities. Given that post-exercise oxygen consumption increases exponentially at high exercise intensities (Børsheim and Bahr 2003), and since, from an applied perspective, athletes are much more likely to need to recover quickly from those intensities, further research should focus on recovery from the higher end of the exercise intensity spectrum.

The present study is the first to model the recovery of PPO following a maximal sprint. While the number of data points used in the modelling process was small, the ability to model recovery using a relatively large sample size provided an effective way of comparing the recovery kinetics of PPO and $\dot{V}O_2$. Although differences in end-exercise power outputs make direct comparisons difficult, the recovery kinetics

of $\dot{V}O_2$ were similar to those of previous research (Dupont et al. 2010; Krustrup et al., 2009; Özyenor et al. 2001). In contrast, although the recoveries of PPO and [PCr] are suggested to have similar kinetics (Bogdanis et al. 1995), with the latter displaying kinetics similar to those of $\dot{V}O_2$ (Barker et al. 2008a; Rossiter et al. 2002), the results of the present study suggest otherwise, revealing a much smaller first-order amplitude for the recovery of PPO than expected. As with $\dot{V}O_2$, the recovery kinetics of PPO may reflect more than the just resynthesis of [PCr], with various peripheral (e.g. intracellular accumulations of inorganic phosphate, lactate, and H^+) and central (e.g. action potential blockage, and reduced motor neuron firing rates) factors potentially influencing the response (Allen et al. 2008; Ament & Verkerke 2009; Gandevia 2001). Nevertheless, despite differences in kinetic parameters, the recoveries of PPO and $\dot{V}O_2$ displayed similar overall kinetics with similar times to achieve 50% recovery. However, participants tended to underestimate the time to achieve full recovery of PPO. Although the latter point would mitigate against the use of perceptual responses to evaluate recovery, self-determined recoveries based on perceptual responses have been found to be an effective way for individuals to maintain performance in a repeated-sprint protocol (12×30 m; mean recovery ~ 80 s) (Glaister et al. 2009). In effect, it appears that recovery is a phenomenon which is dependent on the variable being used to evaluate it, as reflected by the fact that, in contrast to the relatively short recovery time of PPO, elevations in $\dot{V}O_2$ and blood lactate are often observed for long periods after exercise (Bahr 1992; Børsheim and Bahr 2003; Dodd et al. 1984). To complicate matters further, it appears that the time course of the recovery of PPO is likely to be determined by the duration of the preceding exercise bout, with longer bouts producing slower recovery kinetics, despite similar end-exercise levels of fatigue (Baker et al. 1993). Overall, recovery is

determined by the amount of fatigue associated with a bout of exercise and the extent to which mechanisms of that fatigue influence subsequent performance. Since many of the mechanisms of fatigue remain unresolved (Allen et al. 2008; Ament & Verkerke 2009; Gandevia 2001) it is not surprising that the same is true of recovery.

The second aim of the present study was to evaluate the influence of aerobic fitness on the recovery of PPO. The absence of a significant difference in the recovery of PPO between the 'high' versus 'low' $\dot{V}O_{2max}$ groups, coupled with the non-significant correlation between $\dot{V}O_{2max}$ (or GET) and the time to achieve 50% recovery of PPO, suggests that this component of fitness is not an influential factor, at least in the recovery of PPO. It should be noted, however, that significant differences in $\dot{V}O_{2max}$ between the 'high' and 'low' groups were also accompanied by significant differences in relative PPO. This potential mismatch between the corresponding levels of fatigue experienced by each group at the end of the initial sprint is a common dilemma in studies investigating recovery (Børsheim and Bahr 2003; Dodd et al. 1984). As such, whilst between-group differences in PPO allows for speculation that the absence of a faster recovery of PPO by the 'high' group could, at least in part, be explained by a potentially greater level of metabolic stress experienced by this group; the absence of a significant relationship between PPO and the time to achieve 50% recovery of PPO argues against this.

The suggestion of a causative connection between $\dot{V}O_{2max}$ and the recovery of PPO stems from links between [PCr] and PPO recovery kinetics (Bogdanis et al. 1995), combined with reports that [PCr] recovery kinetics are dependent on oxidative phosphorylation capacity (McCully et al. 1993; Paganini et al. 1997). Indeed,

manipulations of oxygen availability have been shown to influence [PCr] recovery kinetics (Haseler et al. 1999) as well as the ability to maintain power output in repeated-sprint tests (Balsom et al. 1994a, 1994b). Moreover, endurance training has been shown to result in faster [PCr] (Forbes et al. 2008; Takahashi et al. 1995; Yoshida 2002) and pulmonary $\dot{V}O_2$ (Billat et al. 2002; Fukuoka et al. 2002, 2006) recovery kinetics, at least following moderate intensity exercise. In contrast, Marwood et al. (2011) found no effect of endurance training on $\dot{V}O_2$ recovery kinetics in an adolescent population, despite significant between-group differences in $\dot{V}O_{2max}$. Furthermore, endurance training has been found to have no significant effect on the ability to maintain performance in repeated-sprint tests (Edge et al. 2005; Glaister et al. 2007). Correlations between $\dot{V}O_{2max}$ and measures of fatigue in repeated-sprint tests show similarly conflicting results (Glaister 2008), with the strongest reported relationship (Dupont et al. 2010) only explaining approximately 69% of the variability in fatigue scores. The use of relatively homogenous populations, combined with small sample sizes, may explain many of these discrepancies. Nevertheless, the results of the present study suggest that either $\dot{V}O_{2max}$ has no effect on the recovery kinetics of PPO, or that the effects of aerobic fitness on [PCr] recovery kinetics are too small to produce an identifiable effect on PPO.

In summary, despite following similar overall kinetics, the results of the present study show that the kinetic parameters which describe PPO in recovery are different from those of $\dot{V}O_2$. Moreover, the ability to recover PPO does not appear to be affected by aerobic fitness, at least when considering differences of the magnitude observed in the present study. Whilst the same effect may not necessarily be true if considering the ability to recover performance in longer bouts of exercise, the difficulties of getting

participants to perform those bouts without adopting pacing strategies, particularly following extremely short recovery periods, poses a challenge for future research.

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Conflict of Interest

The authors have no conflicts of interest that are relevant to the content of this article.

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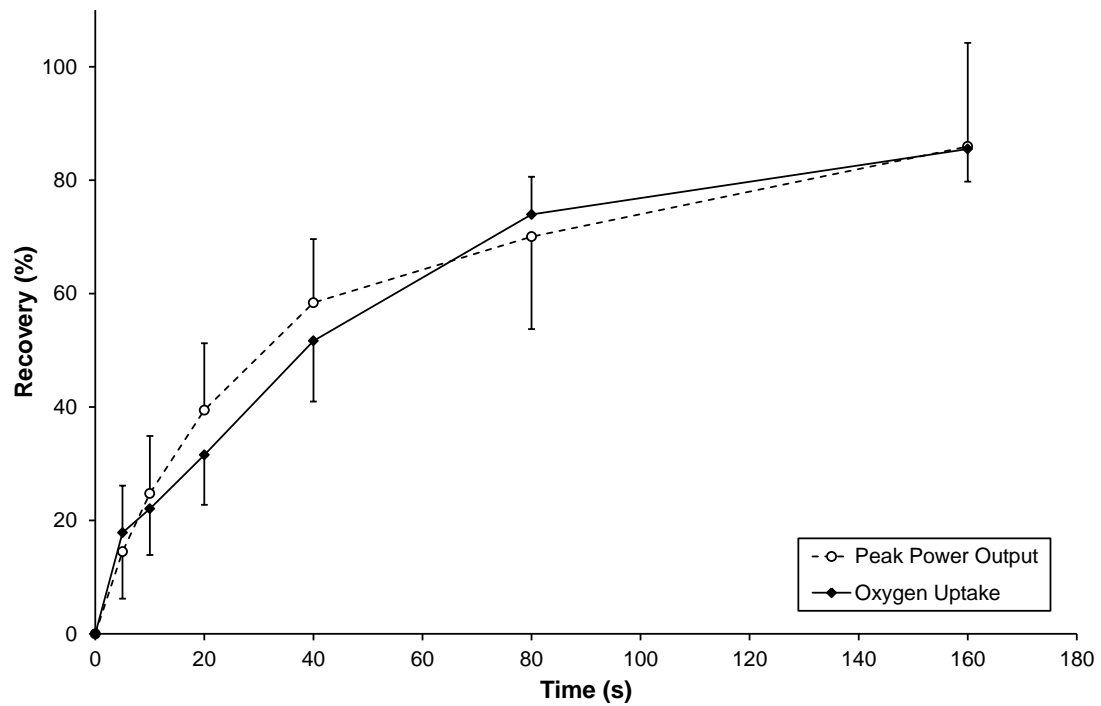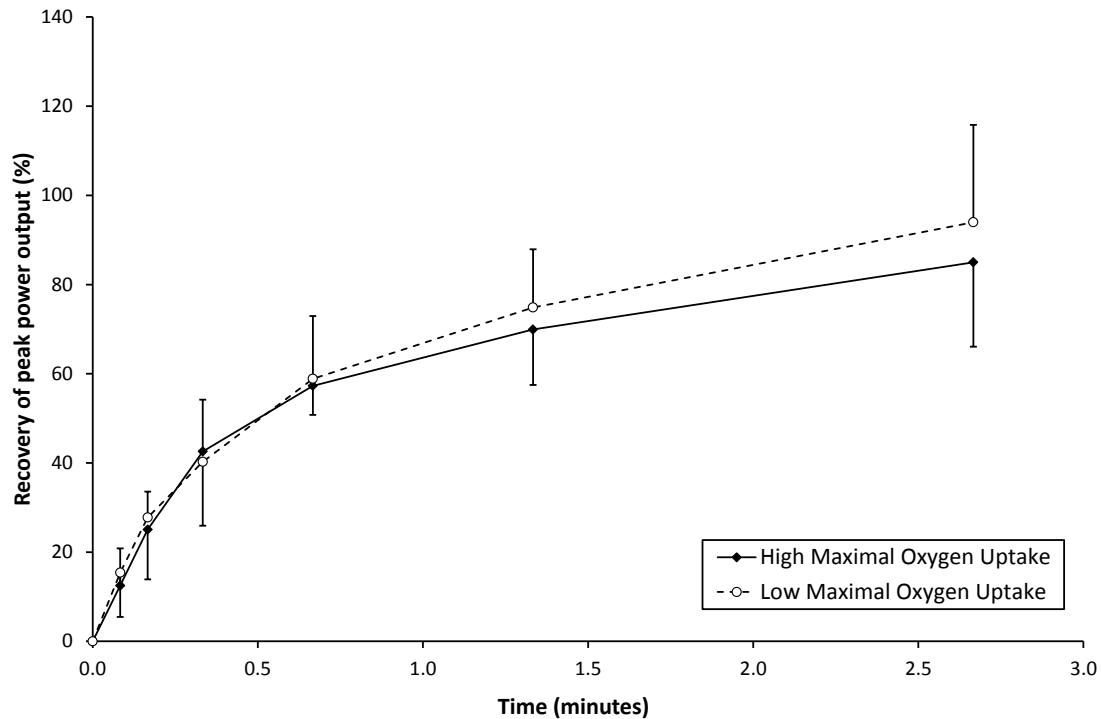
Figure 1. The recovery patterns of peak power output and oxygen uptake following a

maximal 30 s cycle sprint (*n* = 17). Values are means; bars are standard deviations.
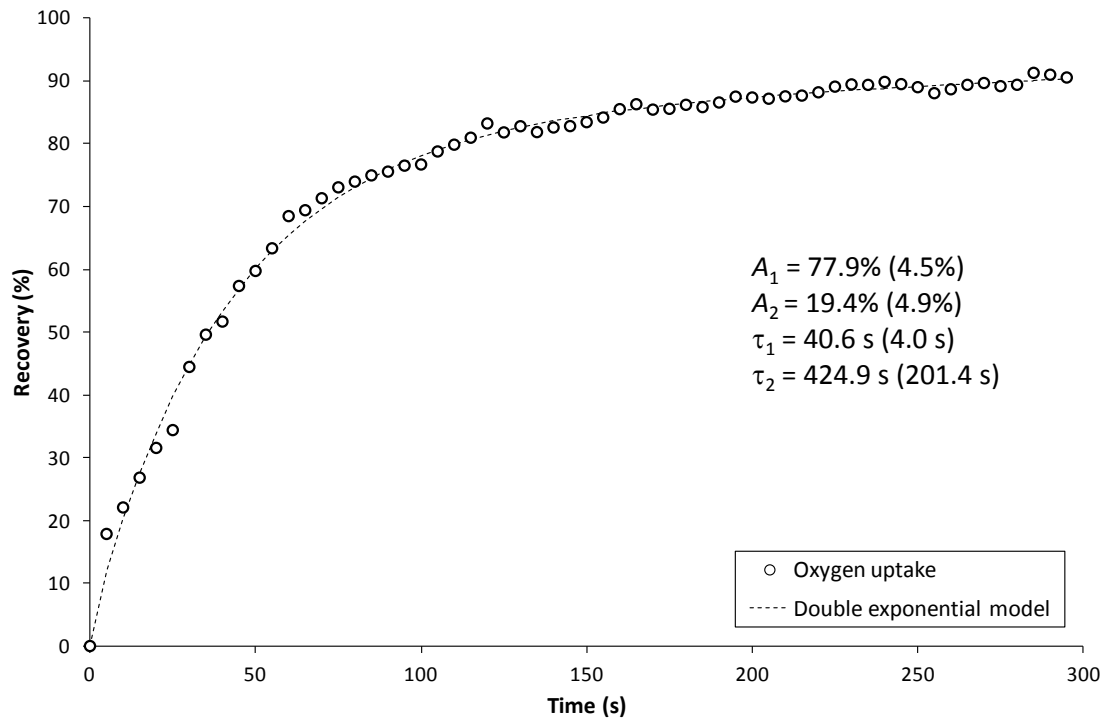
Figure 2. Oxygen uptake as a percentage of recovery from a 30 s bout of maximal sprint cycling and described by a double-exponential model. Values are mean group responses. Note: A_1 and A_2 refer to the first and second-order amplitudes of the responses; τ_1 and τ_2 refer to the first and second-order time constants of the responses. Values in parentheses are 95% confidence intervals.
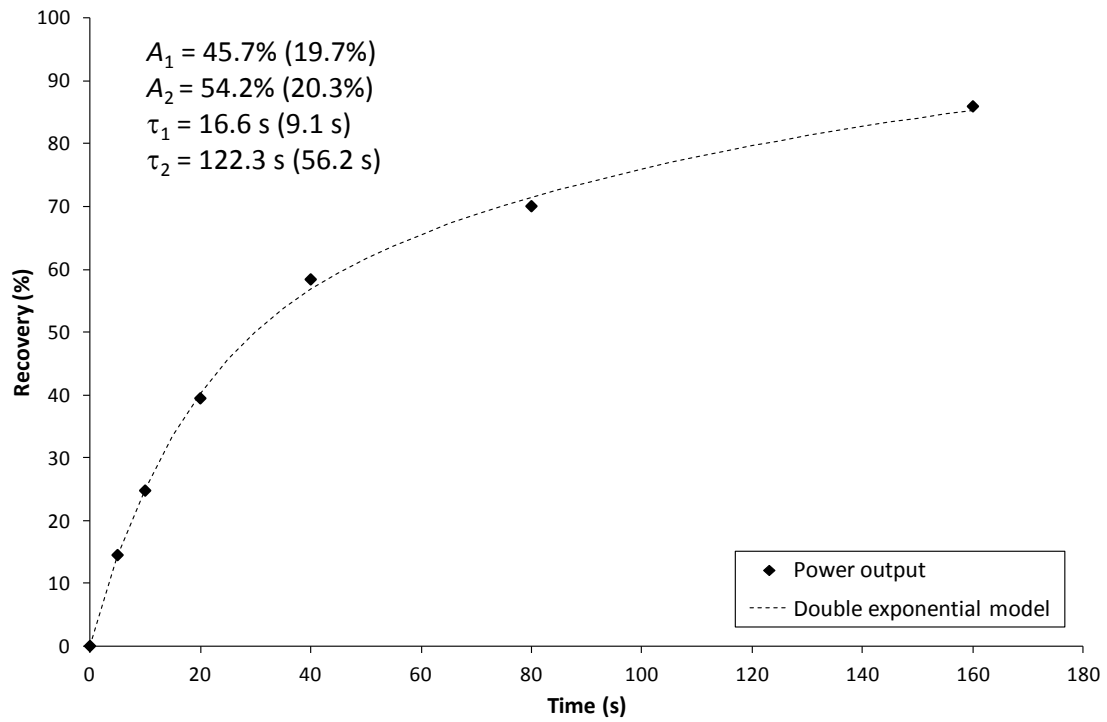
Figure 3. The recovery of peak power output, following a 30 s bout of maximal sprint cycling, modelled using a double-exponential function. Values are mean group responses. Note: A_1 and A_2 refer to the first and second-order amplitudes of the responses; τ_1 and τ_2 refer to the first and second-order time constants of the responses. Values in parentheses are 95% confidence intervals.

Figure 4. The recovery patterns of peak power output following a maximal 30 s cycle sprint for subjects characterised as having 'high' or 'low' rates of maximal oxygen uptake. Values are means; bars are standard deviations.

Table 1. Key variables in double-exponential models of the recoveries of peak power output and oxygen uptake following a maximal 30 s sprint. Values are means ± standard deviation.

	τ_1 (s)	τ_2 (s)	A_1 (%)	A_2 (%)
Peak power output	21.5 ± 13.8	200.3 ± 130.3	53.7 ± 25.8	47.1 ± 24.5
Oxygen uptake	$41.4 \pm 9.8^*$	$317.5 \pm 141.8^*$	$76.6 \pm 11.0^*$	$21.5 \pm 10.1^*$

A_1 and A_2 are the amplitudes of the first and second-order responses respectively; τ_1 and τ_2 are the time constants of each exponential. *significant difference ($p < 0.05$) between peak power output and oxygen uptake responses.

Table 2. Descriptive statistics for participants characterised as having 'high' versus 'low' measures of maximal oxygen uptake. Values are means ± standard deviation.

Group	n	Age (years)	Height (m)	Body mass (kg)	$\dot{V}O_{2max}$ (ml·kg^{-1}·min^{-1})	GET (ml·kg^{-1}·min^{-1})	Peak power outpu (W·kg^{-1})
High	5	22 ± 3	1.85 ± 0.07	78.5 ± 9.2	59.4 ± 1.3	36.1 ± 2.8	14.9 ± 1.6
Low	5	22 ± 3	1.82 ± 0.04	78.7 ± 6.2	$48.5 \pm 2.2^*$	$26.0 \pm 3.1^*$	$13.1 \pm 0.7^*$

GET = gas exchange threshold. *significant difference ($p < 0.05$) between groups

Table 3. Key variables in double-exponential models of the recovery of oxygen uptake for participants characterised as having 'high' versus 'low' measures of maximal oxygen uptake. Values are means ± standard deviation.

Group	τ_1 (s)	τ_2 (s)	A_1 (%)	A_2 (%)	$t_{0.5}$
High	37.4 ± 9.8	298.3 ± 169.5	80.1 ± 6.9	19.2 ± 7.6	33.5 ± 10.6
Low	43.4 ± 12.8	237.4 ± 121.5	70.9 ± 11.7	25.4 ± 8.9	35.7 ± 5.4

A_1 and A_2 are the amplitudes of the first and second-order responses respectively; τ_1 and τ_2 are the time constants of each exponential; $t_{0.5}$ represents the time to achieve 50% of recovery. *significant difference ($p < 0.05$).