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A 350-S Recovery Period Does Not Necessarily Allow Complete Recovery of Peak Power Output during Repeated Cycling Sprints

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Abstract The aim of this study was to determine whether a 350-s recovery period allows recovery of peak power output (PPO) to its initial value under the condition of a blood lactate (La) concentration higher than 10 mmol·L^{-1} during repeated cycling sprints (RCS). RCS (10×10-s cycling sprints) were performed under two conditions. Under one condition, the recovery period of RCS was fixed at 35 s (RCS_{35}), and under the other condition, a 350-s recovery period was set before the 5th and 9th sets, and a 35-s recovery period was set before the other sets (RCS_{comb}). In RCS_{comb}, PPO in the 5th set recovered to that in the 1st set, but PPO in the 9th set did not. Under both conditions, blood La concentration progressively increased and reached approximately 14 mmol·L^{-1} at the end of the RCS. In RCS_{comb}, $\dot{V}O_2$ immediately before the 5th set was not significantly different from that immediately before the 9th set. Mean power frequency (MPF) values estimated by a surface electromyogram from the vastus lateralis in the 5th and 9th sets were significantly higher in RCS_{comb} than in RCS_{35}. In conclusion, a 350-s recovery period does not allow recovery of PPO to its initial value under the condition of a blood La concentration of 14 mmol·L^{-1} during RCS. *J Physiol Anthropol 26(2): 51–57, 2007* http://www.jstage.jst.go.jp/browse/jpa2
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Introduction

Despite the fact that many sporting events are characterized by maximal intermittent sprint exercise in which a short-term sprint is repeated with passive recovery and/or low-intensity exercise, the determinant of performance during maximal intermittent sprint exercise is still not clear. Traditionally, sports scientists have argued that depleted energy substrate and metabolite accumulation reduce performance in such exercise (Gaitanos et al., 1993).

In a single 6-10-s sprint, ATP production resulting from the breakdown of phosphocreatine (PCr) mainly limits peak power output (PPO) (Nevill et al., 1996). Hirvonen et al. (1987) found in a series of maximal sprints (40–100 m) that skeletal muscle PCr store is greatly depleted after 5–7 s, indicating that anaerobic glycolysis must provide the majority of ATP to complete the sprint. Therefore, when short-term sprints are repeated before PCr resynthesis is complete, the energy supply derived from anaerobic glycolysis becomes important for PPO (Dawson et al., 1997), resulting in lactate accumulation (Gaitanos et al., 1993). It is well known that the accumulated lactate increases hydrogen ions (H^+), resulting in reduction of muscle pH. The reduced muscle pH causes muscle fatigue (Fitts, 1994). Thus, when short-term sprints are repeated before PCr resynthesis is complete, both incomplete repletion of the PCr store and subsequent metabolic acidosis reduce PPO (Gaitanos et al., 1993).

However, Sahlin and Ren (1989) have shown that, regardless of the reduced muscle pH, maximal voluntary contraction (MVC) was duplicated when the PCr value had recovered to the resting level. A 3-min recovery period allowed PCr to recover to 90% and 84% of resting values after a single 6-s sprint and repeated sprints (5×6-s sprints) with 24-s recovery periods, respectively (Dawson et al., 1997). In addition, previous studies have shown that the PCr store utilized by high-intensity exercise recovered to almost the resting level in 6 min (Arsac et al., 2004; Harris et al., 1976; McCann et al., 1995). We therefore hypothesize that even if both incomplete recovery of PCr resynthesis and subsequent reduced muscle pH reduce PPO during repeated sprints with short-term recovery periods, a subsequent 6-min recovery period, in which PCr is fully resynthesized, will allow recovery of PPO to its initial value regardless of the reduced muscle pH.

Matsuura et al. (2006) have shown that PPO remained unchanged during repeated cycling sprints (RCS) (10×10-s sprints) with 350-s recovery periods. They found that blood lactate (La) concentration reached approximately 10 mmol·L^{-1} at the end of RCS with 350-s recovery periods. Previous studies have shown that a blood La concentration of

10 mmol·L^{-1} did not reduce PPO during RCS with long (2–5 min) recovery periods (Balsom et al., 1992; Ratel et al., 2002). The results of these studies seemingly support the idea that a relatively longer (2–6 min) recovery period allows recovery of PPO to its initial value regardless of the reduced muscle pH. However, no studies have shown whether a long recovery period allows recovery of PPO to its initial value under the condition of a blood La concentration higher than 10 mmol·L^{-1}.

In the RCS (10×10-s sprints) with 35-s recovery periods in the study by Matsuura et al. (2006), the blood La concentration at the end of RCS (approximately 14 mmol·L^{-1}) was higher than that in the RCS with 350-s recovery periods. Therefore, if 35-s recovery periods in RCS with 35-s recovery periods are periodically replaced with 350-s recovery periods, it would be possible to determine whether a 350-s recovery period, in which PCr seems to be completely resynthesized, allows recovery of PPO to its initial value under the condition of a blood La concentration higher than 10 mmol·L^{-1}.

The aim of this study was to determine whether a 350-s recovery period allows recovery of PPO to its initial value under the condition of a blood La concentration higher than 10 mmol·L^{-1} during RCS.

Methods

Subjects

Eight healthy male undergraduate students participated in this study. The subjects' mean age, height, and body mass were 20.4±1.4 (SD) yr, 171.2±5.9 cm, and 66.7±10.6 kg, respectively. They had no neuromuscular disorders and were competitive, trained swimmers. Their weekly training distance averaged approximately 20000 m. Each subject signed a statement of informed consent following a full explanation regarding the nature of the experiment. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

Design

Each subject attended our laboratory for three tests. The time interval between two consecutive tests was at least 3 days, and all tests were completed within 2 weeks. On the first test day, the subjects' body characteristics were measured, and each subject performed half of the RCS with the 35-s recovery periods described below to become familiar with RCS. Body mass (BM) was used to determine the loads of the cycling sprint. Each subject was instructed to refrain from intense physical exercise, drinking alcohol, and taking caffeine for 24 h prior to each visit. None of the subjects had a smoking habit.

Experimental protocol

Each subject came to the laboratory 30 min before the start of the test. Experimental instruments were then fitted to each subject. After resting for 3 min on the bicycle seat, each subject performed two RCS tests in randomized order on separate days. One test consisted of ten 10-s cycling sprints with 35-s recovery periods (340 s of low-intensity cycling exercise with 0 W at 60 rpm and 10-s passive recovery on the bicycle seat: RCS_{35}). The other test (RCS_{comb}) consisted of ten 10-s cycling sprints with eight 35-s recovery periods (25 s of low-intensity cycling exercise with 0 W at 60 rpm and 10-s passive recovery on the bicycle seat) immediately before the 2nd, 3rd, 4th, 6th, 7th, 8th, and 10th sets, respectively, and two 350-s recovery periods (340-s low-intensity cycling exercise with 0 W at 60 rpm active recovery and 10-s passive recovery) immediately before the 5th and 9th sets, respectively. All cycling sprints were performed with a load (F) [N] of $0.075 \cdot BM \cdot 9.81^{-1}$ (Ayalon et al., 1974) from a stationary start. Subjects were instructed to pedal as many revolutions as possible during the cycling sprints. The number of sets in the RCS was not announced in advance, to prevent the subjects from making an unconscious plan for whole power output.

Peak power output during 10-s cycling sprints

All exercise tests were carried out on a bicycle ergometer (POWERMAX-V_{II}, Combi, Tokyo, Japan). The duration and load were adjusted by a built-in computer. The computer also calculated mean power output and peak rpm (Rpm_{peak}) in a given exercise, and displayed the results. Since the screen on the bicycle ergometer displayed Rpm_{peak}, the screen was covered. Each subject's feet were strapped to the pedals to prevent them from slipping. The seat height was adjusted so that there was a slight bend in the knee joint when the foot pedal was at its lowest position. PPO was calculated by the following equation:

$$PPO\ [W] = Rpm_{peak} \cdot 6 \cdot F \cdot 0.624^{-1},$$

where 6 is the distance calculated by the built-in computer as the flywheel went into a 360-degree roll [m], and 0.624 is the value for transforming Nm units to W units [Nm·min^{-1}·W^{-1}]. To reduce variation due to differences in the body characteristics of subjects, PPO divided by BM ($PPO \cdot BM^{-1}$) was used.

Blood lactate concentration

Blood samples (25 μL) were collected from fingertips using capillary tubes, and analyzed using a lactate analyzer (YSI-1500 sport, YSI, Tokyo, Japan) to measure blood La concentration. The lactate analyzer was calibrated by a standard lactate solution of 5 mmol·L^{-1} before each test. Blood was sampled at rest and immediately after the 5th and 10th cycling sprints.

Oxygen uptake

Data on oxygen uptake ($\dot{V}O2$) were obtained breath by breath using a respiratory gas analyzer (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation ($\dot{V}E$) was measured by a hot-wire flow meter, which was calibrated with

a syringe of known volume (2.0 L). O_2 and CO_2 concentrations were measured by a zirconium sensor and infrared absorption analyzer, respectively. The gas analyzer was calibrated by known standard gas (O_2: 15.17%, CO_2: 4.92%). $\dot{V}O_2$ was measured continuously during rest, exercise, and recovery periods. For each 15-s interval, the averages of $\dot{V}O_2$ were calculated. To reduce variation due to difference in the body characteristics of subjects, $\dot{V}O_2$ divided by BM ($\dot{V}O_2 \cdot BM^{-1}$) was used.

Surface electromyogram

A surface electromyogram (SEMG) was recorded from the left vastus lateralis (VL) and the left rectus femoris (RF) at a rate of 1000 Hz during each of the ten cycling sprints. Before attachment of the surface electrodes, the skin was shaved, abraded, and cleaned with alcohol in order to reduce source impedance. A bipolar SEMG sensor (SX230, Biometrics Ltd, United Kingdom; inter-electrode distance of 20 mm) was placed on the lateral side of the crural area five fingers proximal from the patella of the belly of the VL and on the lower limb midway between the superior surface of the patella and the anterior superior iliac spine of the belly of the RF. The ground electrode was placed over the right lateral malleolus. The SEMG signals were amplified using an amplifier imbedded in the EMG sensor (bandwidth=20–450 Hz; common mode rejection ratio, CMRR>96 dB; input impedance>10 TΩ; gain=1000) and converted into digital signals using an analog-digital converter (MacLab/8s, AD Instruments, Australia). Then SEMG data were processed offline by using analysis software (Acknowledge, BIOPAC Systems, United States). Raw data were filtered using a band pass Finite Impulse Response filter with a cut-off frequency of 10 to 500 Hz. Since a previous study in our laboratory (Matsuura et al. 2005) showed that power output during a 10-s cycling sprint reached a peak at 6.2 s on average, four seconds (4–8 s) of raw data were used for subsequent analysis. The four seconds of raw data were full-wave-rectified and then integrated (IEMG). Furthermore, the raw data were processed with fast Fourier transform to obtain a frequency power spectrum. The frequency spectrum analysis was restricted to frequencies in the range 5–500 Hz (Kay et al., 2001; St Clair Gibson et al., 2001). Mean power frequency (MPF) was defined as the ratio between spectral moments of orders one and zero (Moritani et al., 1982). IEMG and MPF calculated from raw data of an SEMG in a given time period during dynamic exercise such as a cycling sprint are used to determine neuromuscular activation (Hunter et al. 2003; Kay et al. 2001; St Clair Gibson et al. 2001). The positions of the electrodes for SEMG detection were similar under the two conditions because reference points were marked on the skin. The IEMG and MPF were normalized as a percentage of the 1st-set value.

Statistical analysis

Results are presented as means ± standard deviations (SD). A paired t-test was used to examine differences between conditions. One-way ANOVA (for $PPO \cdot BM^{-1}$ and $\dot{V}O_2 \cdot BM^{-1}$) for repeated measures was used to examine the time effect. Two-way ANOVA (for blood La concentration, IEMG, and MPF) for repeated measures on both factors (sprint number and condition) was used. When significant F ratios were found, the means were compared by using Tukey-Kramer's post-hoc test. If significant interaction was indicated, one-way ANOVA for repeated measures was used to examine the time effect, and a paired t-test was used to examine the difference between conditions. A value of $p<0.05$ was regarded as statistically significant.

Results

Peak power output during 10-s cycling sprints

$PPO \cdot BM^{-1}$ values in the 1st set in the two RCS trials were similar (RCS_{35}: 10.8±0.8 $W \cdot kg^{-1}$, RCS_{comb}: 10.7±0.6 $W \cdot kg^{-1}$; $p=0.85$). $PPO \cdot BM^{-1}$ values were significantly lower in the 4th–10th sets than in the 1st set in RCS_{35}, and $PPO \cdot BM^{-1}$ values were significantly lower in the 3rd, 4th, 7th, 8th, 9th, and 10th sets than in the 1st set in RCS_{comb} (Fig. 1).

Blood lactate concentration

Blood La concentration recorded immediately after the 5th and 10th cycling sprints are expressed as 5th- and 10th-set values, respectively (Fig. 2). When two-way ANOVA for repeated measures was used, no significant interaction was found in blood La concentration. No significant effect of the condition on blood La concentration was found. In both RCS_{35} and RCS_{comb}, Blood La concentration was significantly higher in the 5th and 10th sets than at rest and was significantly higher in the 10th set (RCS_{35}: 13.9±3.1 $mmol \cdot L^{-1}$, RCS_{comb}: 14.0±3.1 $mmol \cdot L^{-1}$) than in the 5th set (RCS_{35}: 10.2±1.4 $mmol \cdot L^{-1}$, RCS_{comb}: 11.0±0.9 $mmol \cdot L^{-1}$).

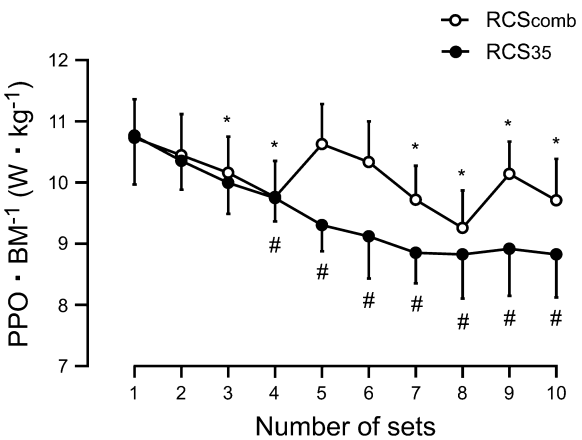

Fig. 1 Changes in peak power output (PPO) divided by body mass (BM) in each of the subjects ($PPO \cdot BM^{-1}$) during each of the ten 10-s cycling sprints in RCS_{comb} (○) and RCS_{35} (●). *: significantly different ($p<0.05$) from the value in the 1st set in RCS_{comb}. #: significantly different ($p<0.05$) from the value in the 1st set in RCS_{35}.

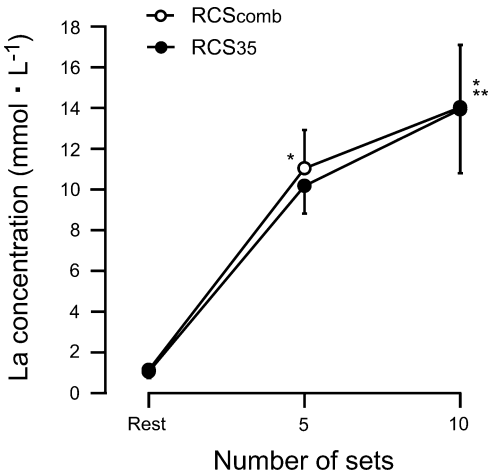

Fig. 2 Changes in blood lactate (La) concentration during RCS_{comb} (○) and RCS_{35} (●). *: significantly different ($p<0.05$) from the value at rest. **: significantly different ($p<0.05$) from the value in the 5th set.

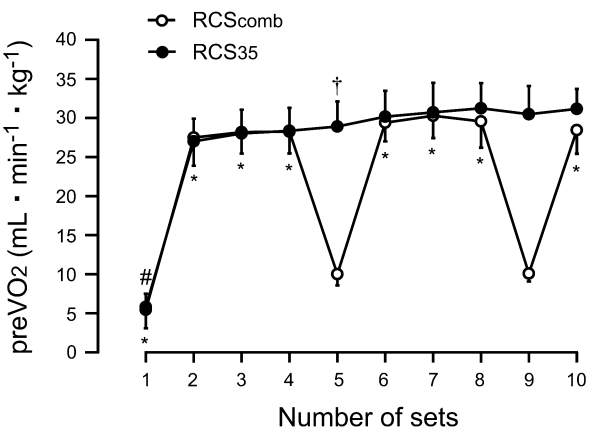

Fig. 3 Changes in $\dot{V}O_2$ divided by body mass (BM) in each of the subjects for 15 s immediately before each of the ten cycling sprints ($pre\dot{V}O_2$) during RCS_{comb} (○) and RCS_{35} (●). *: significantly different ($p<0.05$) from the value in the 5th set in RCS_{comb}. #: significantly different ($p<0.05$) from the value in the 5th set in RCS_{35}. †: significant difference ($p<0.05$) between the value in RCS_{comb} and RCS_{35}.

Oxygen uptake immediately before cycling sprints

The changes in $\dot{V}O_2 \cdot BM^{-1}$ for 15 s immediately before each of the ten cycling sprints ($pre\dot{V}O_2$) in the two RCS tests are shown in Fig. 3. The value of $pre\dot{V}O_2$ recorded immediately before the 1st cycling sprint is expressed as the 1st-set value. The 5th-set value was used as a baseline value since active recovery immediately before the 1st cycling sprint was not performed. In RCS_{35}, $pre\dot{V}O$ was significantly lower in the 1st set than in the 5th set, and no significant differences were found from the 2nd set to the 10th set. In RCS_{comb}, $pre\dot{V}O_2$ was significantly lower in the 1st set than in the 5th set and was significantly higher in the 2nd, 3rd, 4th, 6th, 7th, 8th, and 10th sets than in the 5th set. There was no significant difference between $pre\dot{V}O_2$ value in the 5th set and that in the 9th set. The value of $pre\dot{V}O_2$ in the 5th set during RCS_{35} ($28.9 \pm 3.2\,mL \cdot min^{-1} \cdot kg^{-1}$) was significantly higher than that in the 5th set during RCS_{comb} ($10.0 \pm 1.4\,mL \cdot min^{-1} \cdot kg^{-1}$). Because of technical difficulties, the proportion of low-intensity exercise/passive recovery time intervals contributing to $pre\dot{V}O_2$ was not strictly constant.

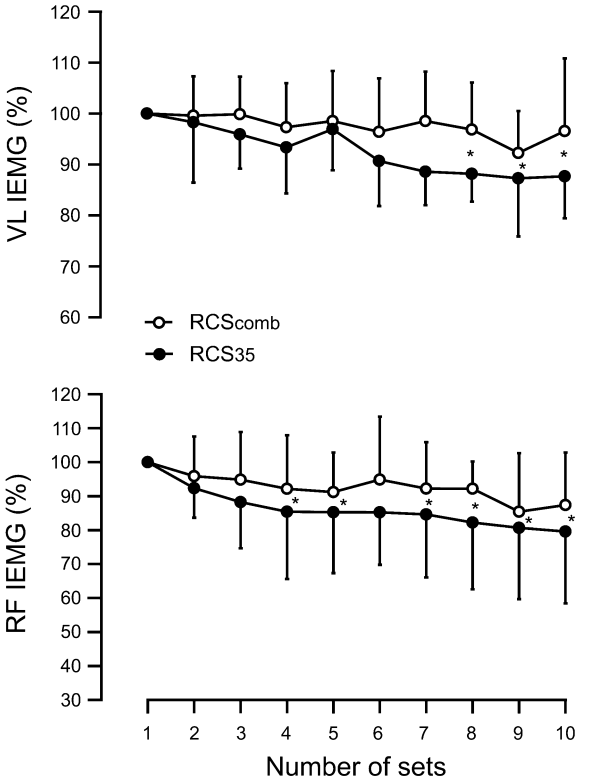

Fig. 4 Changes in integrated EMG (IEMG) from the left vastus lateralis (VL: top) and from the left rectus femoris (RF: bottom) normalized by the 1st set value during RCS_{comb} (○) and RCS_{35} (●). *: significantly different ($p<0.05$) from the value in the 1st set.

IEMG and MPF

There were no differences between the two RCS trials in absolute IEMG value (VL: $p=0.43$, RF: $p=0.81$; data not shown) and absolute MPF value (VL: $p=0.57$, RF: $p=0.15$; data not shown) in the 1st set. Changes in IEMG and MPF in the two RCS trials are shown in Fig. 4 and Fig. 5, respectively. When two-way ANOVA for repeated measures was used, no significant interaction was found in IEMG from both the VL and the RF or in MPF from the RF. In both RCS_{35} and RCS_{comb}, IEMG from the VL significantly decreased from the 8th set compared to that in the 1st set, and IEMG from the RF was significantly lower in the 4th, 5th, 7th, 8th, 9th, and 10th sets than in the 1st set. MPF from the RF was significantly lower in the 3rd set than in the 1st set. A significant interaction was found in MPF from the VL. In RCS_{comb}, MPF from the VL was significantly lower in the 3rd set than in the 1st set and was significantly higher in the 5th set than in the 4th set. MPF values from the VL in the 5th and 9th sets during RCS_{comb}

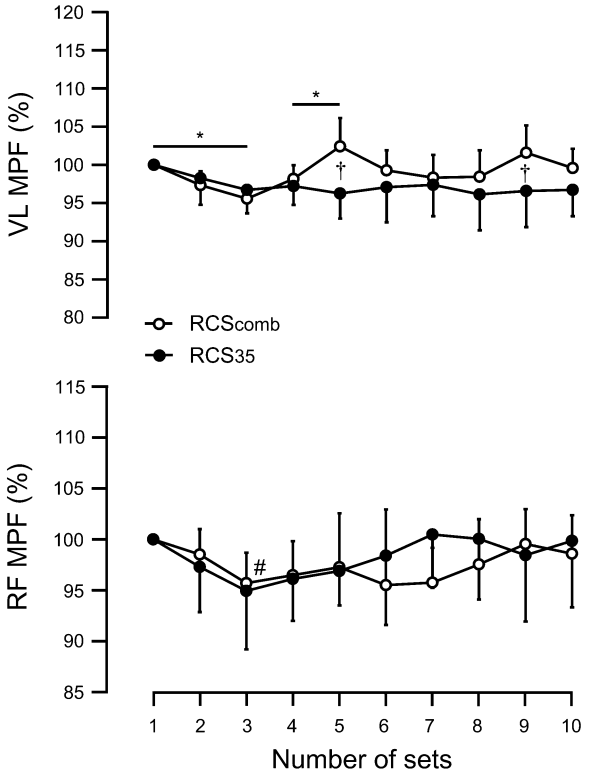

Fig. 5 Changes in mean power frequency (MPF) from the left vastus lateralis (VL: top) and from the left rectus femoris (RF: bottom) normalized by the 1st set value during RCS_{comb} (○) and RCS_{35} (●). *: significantly different ($p<0.05$) in RCS_{comb}. #: significantly different ($p<0.05$) from the value in the 1st set. †: significant difference ($p<0.05$) between the value in RCS_{comb} and RCS_{35}.

were significantly higher than those in the same sets during RCS_{35}.

Discussion

The main finding in the present study was that PPO in the 5th set of RCS_{comb} recovered to that in the 1st set, but that PPO in the 9th set of RCS_{comb} did not recover to that in the 1st set.

In the present study, we expected that a 350-s recovery period would allow PCr to be completely resynthesized. Although PCr content was not directly measured in the present study, the PCr store immediately before each cycling sprint can roughly be estimated from the pre$\dot{V}O_2$ recorded in the present study (Matsuura et al., 2006) since Rossiter et al. (2002) reported that restoration of PCr after high-intensity exercise corresponds to decrease in $\dot{V}O_2$ during the recovery period. Since there was no significant difference between pre$\dot{V}O_2$ in the 5th set and that in the 9th set in RCS_{comb}, it is inferred that in RCS_{comb} the PCr store immediately before the 5th set was very similar to that immediately before the 9th set. In RCS_{comb}, pre$\dot{V}O_2$ values in the 5th and the 9th sets were significantly higher than the value in the 1st set. This higher pre$\dot{V}O_2$ in the 5th and 9th sets may be explained by the fact that active recovery was performed during recovery periods in RCS and was not performed before the 1st set. Recently, some researchers have suggested that accumulation of inorganic phosphate (Pi) due to PCr breakdown induces muscle fatigue (Westerblad et al., 2002). Since there is an almost stoichiometric increase in Pi with PCr breakdown (Sahlin et al. 1998), it is assumed that the magnitude of accumulation of Pi immediately before the 5th set was very similar to that immediately before the 9th set. Therefore, it is inferred that the difference between PPO in the 5th set and that in the 9th set in RCS_{comb} was not associated with PCr store and/or accumulation of Pi.

In RCS_{comb}, blood La concentrations in the 5th and the 10th sets were 11.0 ± 0.9 and 14.0 ± 3.1 mmol·L^{-1}, respectively. The difference between PPO in the 5th set and that in the 9th set may be associated with this difference in blood La concentrations. Reduction in muscle pH induced by lactic acid accumulation results in reductions in Ca^{2+} sensitivity (Fabiato and Fabiato, 1978; Godt and Nosek, 1989), maximal tension (Cooke et al., 1988; Godt and Nosek, 1989), and shortening velocity (Cooke et al., 1988). However, it has recently been shown in studies using mammalian muscle that these direct effects of reduced muscle pH on muscle contractility were absent at physiological temperatures (Pate et al. 1995; Westerblad et al. 1997). Consequently, it is likely that reduced muscle pH indirectly affects muscle contractility via the inhibition of ATP supply derived from anaerobic glycolysis (Sahlin et al., 1998). Therefore, there are the following three possible interpretations for the results of the present study. Firstly, there is a possibility that the reduced PPO in the 9th set during RCS_{comb} reflects this indirect effect of reduced muscle pH. This idea is not in accord with the results of a study by Sahlin and Ren (1989) in which MVC was duplicated despite reduced muscle pH. This discrepancy may be due to the difference between the exercise modality used in the present study and that used in the study by Sahlin and Ren (1989). The duration of MVC used in the study by Sahlin and Ren (1989) was <2 s, but it has been reported that power output during the 10-s cycling sprint used in the present study reached a peak at 6.2 s on average (Matsuura et al., 2005). Indeed, Bogdanis et al. (1998) have shown that approximately 40% of the ATP utilized during a 6–10-s cycling sprint is supplied by anaerobic glycolysis. Thus, the energy supply process utilized during a 10-s cycling sprint may be different from that during MVC of <2 s. PPO during a 10-s cycling sprint would be determined by not only PCr but also glycolysis.

Secondly, the reduced PPO in the 9th set during RCS_{comb} might reflect the alteration of the efferent motor command sent to exercising muscles by the central nervous system (CNS). Recent studies have suggested that the efferent motor command from the CNS to exercising muscle is modified by afferent information about peripheral metabolites to prevent premature fatigue and maintain homeostasis (Lambert et al., 2005; Noakes et al., 2005; St Clair Gibson et al., 2001). In the present study, the values of MPF from the VL in both the 5th and 9th sets were significantly higher in RCS_{comb} than in

RCS_{35}. This result is consistent with that obtained by Matsuura et al. (2006). Matsuura et al. (2006) showed that MPF was lower during RCS with 35-s recovery periods than during RCS with 350-s recovery periods, suggesting that a severer metabolic state induces preferred recruitment of slow twitch motor units. In RCS_{comb}, MPF from the VL in the 5th set was significantly higher than that in the 4th set, but MPF from the VL in the 9th set was not significantly higher than that in the 8th set. Therefore, the reduced PPO in the 9th set during RCS_{comb} might be induced by the efferent motor command altered subconsciously due to the lower muscle pH.

Thirdly, according to Matsuura et al. (2006), there is also a possibility that interaction of the aforementioned two potential effects induced the reduction in PPO in the 9th set in RCS_{comb}. In the 5th set during RCS_{comb}, the negative effect of a blood La concentration of 10 $mmol \cdot L^{-1}$ on PPO may be offset by the positive effect of increase in MPF on PPO. If the increase in MPF in the 9th set during RCS_{comb} were greater, the negative effect of blood La concentration of 14 $mmol \cdot L^{-1}$ on PPO could be offset by the positive effect of the greater increase in MPF on PPO. However, in fact, the CNS might not increase the efferent motor command sent to the exercising muscles to maintain homeostasis (Lambert et al. 2005).

In the present study, trained subjects were used along with previous studies (Balsom et al. 1992; Ratel et al. 2002). Therefore, further research is required to determine whether interpretations of the present study can be applied to non-trained subjects.

In conclusion, a 350-s recovery period does not allow recovery of peak power output to its initial value under the condition of a blood La concentration of 14 $mmol \cdot L^{-1}$ during repeated cycling sprints.

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