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Author(s)	Matsuura, R.; Arimitsu, T.; Yunoki, T.; Yano, T.
Citation	British Journal of Sports Medicine, 45(10), 820-824 <a href="https://doi.org/10.1136/bjism.2009.068007">https://doi.org/10.1136/bjism.2009.068007</a>
Issue Date	2009
Doc URL	<a href="http://hdl.handle.net/2115/51840">http://hdl.handle.net/2115/51840</a>
Type	article (author version)
File Information	Yano-British.pdf



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**Title**

**Effects of resistive load on performance and surface EMG activity during repeated cycling sprints on a non-isokinetic cycle ergometer**

**Running Title**

Resistive load and SEMG activity during cycling sprints

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**Keywords**

Surface electromyogram; Sprint exercise; Fatigue; Resistive load

**Word Count**

2987 words

**Abstract**

**Objectives:** To determine the effects of resistive load on performance and surface electromyogram (SEMG) activity during repeated cycling sprints (RCS) on a non-isokinetic cycle ergometer.

**Methods:** Subjects performed two RCS tests (ten 10-s cycling sprints) interspersed with both 30-s and 360-s recovery periods under light load (RCS<sub>L</sub>) and heavy load conditions (RCS<sub>H</sub>) in a random counterbalanced order. Recovery periods of 360 s were set before the 5th and 9th sprints.

**Results:** In the 9th and 10th sprints, the values of peak power output divided by body mass (PPO·BM<sup>-1</sup>) were significantly higher in RCS<sub>H</sub> than in RCS<sub>L</sub>. Changes in blood lactate concentration were not different between the two conditions. In RCS<sub>L</sub>, root mean square (RMS) calculated from SEMG was significantly lower in the 9th sprint than in the 1st sprint, but there was no differences between RMS in the 1st sprint and that in the 9th sprint in RCS<sub>H</sub>.

**Conclusions:** During RCS on a non-isokinetic cycle ergometer, performance and SEMG activity are influenced by resistive load. It is thought that regulation of skeletal muscle recruitment by the CNS is associated with fatigue during RCS with a light resistive load.

## Introduction

Historically, many researchers have focused on fatigue during exercise. Both static and dynamic exercises have often been used in the studies on fatigue during exercise, but exercise in daily life and sporting events is dynamic exercise. Since speed of movement and exerted force determine performance in dynamic exercise, the effects of frequency of movement on fatigue have been examined in studies on fatigue during dynamic exercise.

Beelen and Sargeant [1-2] showed that greater fatigue of peak power output (PPO) during a cycling sprint on an isokinetic cycle ergometer was generated at a higher pedaling rate. Bogdanis et al. [3] reported that PPO during repeated cycling sprints (RCS) on a friction-loaded cycle ergometer at a light load (higher peak pedaling rate) significantly decreased and that PPO during RCS at a heavy load (lower peak pedaling rate) was unchanged. The greater fatigue of PPO at a higher pedaling rate has been explained by skeletal muscle recruitment and/or the contribution of recruited skeletal muscle fibers to exerted power in studies using an isokinetic cycle ergometer [1, 2, 4] and has been explained by skeletal muscle recruitment, metabolic response, and power-velocity relationship in a study using a non-isokinetic cycle ergometer [3]. However, skeletal muscle recruitment during a cycling sprint on an isokinetic cycle ergometer is considerably different from that during a sporting event or exercise in daily life. Furthermore, it has been reported that intramuscular lactate concentration ( $[La^-]$ ) at the end of a 30-s cycling sprint on a friction-loaded cycle ergometer was not different between a light load condition and a heavy load condition [5]. Therefore, it is thought that the difference in fatigue during RCS at a high pedaling rate and RCS at

a low pedaling rate on a non-isokinetic cycle ergometer would result from something other than metabolic factors.

Some authors [6, 7, 8, 9] have suggested that the central nervous system (CNS) modifies efferent signals to muscle and peripheral organs based on afferent signals from the muscle and peripheral organs to the CNS to prevent the failure of homeostasis and that the modification is influenced by information involving subsequent exercise. If RCS on a non-isokinetic cycle ergometer is performed in a light load condition, a cycling sprint at a pedaling rate close to the limit would be repeated, resulting in risk of severe damage to muscles and joints. Therefore, it is possible that the CNS prevents catastrophic damage to muscles and joints by regulation of skeletal muscle recruitment and that the regulated skeletal muscle recruitment is associated with fatigue during RCS at a light load.

The purpose of this study was to determine the effects of resistive load on performance and surface electromyogram (SEMG) activity during RCS (ten 10-s cycling sprints on a non-isokinetic cycle ergometer). Since it is likely that not only a decline of intramuscular pH but also a decline of phosphocreatine (PCr) act as metabolic stress during RCS [10-11], the RCS used in this study included both 30-s and 360-s recovery periods, and performance and SEMG activity during a cycling sprint immediately after a 360-s recovery period, which enables PCr to be almost completely resynthesized [12-14], were investigated. We hypothesized that SEMG activity and PPO in a cycling sprint immediately after a 360-s recovery period during RCS at a light resistive load would be lower than those at a heavy resistive load despite the fact that there is no difference

in blood  $[La^-]$  between the conditions.

## **Methods**

### *Subjects*

Seven healthy male undergraduate students participated in the present study. The subjects' mean age, height and body weight were  $21.1 \pm 1.9$  (SD) yr,  $172.3 \pm 10.2$  cm and  $64.2 \pm 8.3$  kg, respectively. They had no neuromuscular disorders and were participating in regular training programs. 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 2 days, and all tests were completed within 2 weeks. On the first test day, the subjects' body characteristics were measured and each subject performed the 1st-4th cycling sprints, during which the resistive load [kp] was equal to 3% (in the 1st and 2nd sprints) and 12% (in the 3rd and 4th sprints) of the individual's body mass (BM), of the experimental protocol described below to become familiarized with RCS. Each subject was instructed to refrain from intense physical exercise, drinking, and taking caffeine for 24 h prior to each visit.

### *Experimental protocol*

Each subject performed two RCS tests with the resistive load [N] of either  $0.03 \cdot BM \cdot 9.81$  (light load condition:  $RCS_L$ ) or  $0.12 \cdot BM \cdot 9.81$  (heavy load condition:  $RCS_H$ ) in a randomized, counterbalanced order on separate days. Each subject came to the laboratory 30 min before the start of the test. Then experimental instruments were fitted to each subject. After resting for 3 min on the bicycle seat, each subject started an RCS test. The test consisted of ten 10-s cycling sprints with eight 30-s passive recovery periods immediately before the 2nd, 3rd, 4th, 6th, 7th, 8th, 10th sprints, respectively, and two 360-s passive recovery periods immediately before the 5th and 9th sprints, respectively. Subjects were instructed to pedal as many revolutions as possible during cycling sprints. The number of sprints in RCS was not announced in advance to prevent the subjects from making an unconscious plan for whole peak power output. Subjects were told that the number of sprints in RCS was not necessarily the same in the two conditions ( $RCS_L$  and  $RCS_H$ ). For all tests, subjects were in the seated position during exercise and recovery. All sprints started from a stationary position.

### *Repeated cycling sprints*

All exercise tests were carried out on a cycle ergometer (POWERMAX-VII, Combi, Tokyo, Japan). The duration and resistive load were adjusted by a built-in computer. The computer also calculated peak rpm ( $Rpm_{peak}$ ) for a given exercise and displayed the results. Since the screen on the bicycle ergometer displayed  $Rpm_{peak}$ , the screen was covered. Time series behavior in rpm



during each cycling sprint was recorded by an online computer at a rate of 10 Hz. 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. Peak power output (PPO) and mean power output (MPO) during each cycling sprint were calculated by methods of Lakomy [15]. The results of rpm were averaged over 0.5-s time intervals and acceleration was calculated from the averaged rpm. The "excess load" was calculated from the acceleration. The instantaneous product of average 0.5-s rpm and effective load (resistive load + excess load) was used to determine corrected power output throughout RCS:

$$\text{Power output [W]} = \text{rpm} \cdot 6 \cdot \text{effective load [N]} \cdot 60.04^{-1},$$

where 6 is the distance calculated by the built-in computer as the flywheel went into a 360-degree roll [m], and 60.04 is the value for transforming Nm units to W units [ $\text{Nm} \cdot \text{min}^{-1} \cdot \text{W}^{-1}$ ]. The maximum product of rpm and effective load, during each sprint, is referred to as PPO. The average 0.5-s product of rpm and effective load, over the total time period of each cycling sprint (i.e., 10 s), is referred to as MPO. To reduce variation due to the difference in body characteristics of subjects, PPO divided by BM ( $\text{PPO} \cdot \text{BM}^{-1}$ ) was used. Furthermore, the average MPO, over the 1st-10th cycling sprints, is referred to as an index of total workload.

#### *Blood lactate concentration*

Blood samples (25  $\mu\text{L}$ ) were collected from fingertips using capillary tubes and analyzed using a lactate analyzer (YSI 1500 SPORT, YSI, OH, USA) to measure blood lactate concentration

([La<sup>-</sup>]). The lactate analyzer was calibrated by a standard lactate solution of 5 mmol·L<sup>-1</sup> before each test. Blood was sampled at rest (Rest), 30 s before the 5th and 9th sprints (Pre5 and Pre9, respectively), and immediately after the end of RCS (Post-Ex).

#### *Oxygen uptake*

Data on oxygen uptake ( $\dot{V}O_2$ ) were obtained breath-by-breath using a respiratory gas analyzer (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation was measured by a hot-wire flow meter, and the flow meter was calibrated with a syringe of known volume (2.0 L). O<sub>2</sub> and CO<sub>2</sub> concentrations were measured by a zirconium sensor and infrared absorption analyzer, respectively. The gas analyzer was calibrated by known standard gas (O<sub>2</sub>: 15.17%, CO<sub>2</sub>: 4.92%).  $\dot{V}O_2$  was measured continuously during rest, exercise, and recovery periods. For each 10-s interval, the average of  $\dot{V}O_2$  was calculated.

#### *Surface electromyogram analysis*

An SEMG was recorded from the left vastus lateralis (VL) 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 skin impedance. A bipolar surface EMG sensor (SX230, Biometrics Ltd, Gwent, South Wales, UK; 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 in the main direction of muscle fibers. The ground electrode was placed over the styloid process of the

right wrist. 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 T $\Omega$ ; gain = 1000) and converted into digital signals using an analog-digital converter (MacLab/8s, AD Instruments, Bella Vista, NSW, Australia). Then SEMG data were processed offline by using analysis software (Acknowledge, BIOPAC Systems, Goleta, CA, USA). Raw data were filtered using a band-pass Finite Impulse Response filter with cut-off frequencies of 20 and 450 Hz. The SEMG activity during RCS was determined by measuring the root mean square (RMS) between the onset and the end of the burst [16]. Burst onsets and offsets were determined using a constant electric threshold of  $\pm 0.2$  mV [16-17]. Because the number of pedalings during a cycling sprint varied between sprint numbers or subjects, the number of pedalings was normalized by expressing the number of pedalings of each cycling sprint as percentage of the total number of pedalings. The positions of the electrodes for SEMG detection were similar in the two conditions because reference points were marked on the skin.

#### *Statistical analysis*

Results are presented as means  $\pm$  standard deviations (SD). All data were checked for normality of distribution prior to performing any inferential statistics. Two-way ANOVA for repeated measures on both factors (time  $\times$  condition) was used. When main effects were found, the means were compared by using Tukey-Kramer's post-hoc test. If a significant interactive effect 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 treatment effect. A value of  $P < 0.05$  was regarded as statistically significant.

## Results

### *Performance during repeated cycling sprints*

A significant interaction ( $P < 0.05$ ) was found in  $\text{PPO} \cdot \text{BM}^{-1}$  (Fig. 1, top). In  $\text{RCS}_L$ , the values of  $\text{PPO} \cdot \text{BM}^{-1}$  were significantly lower in the 4th, 6th, 7th, 8th, 9th and 10th sprints than in the 1st sprint, but the values of  $\text{PPO} \cdot \text{BM}^{-1}$  in  $\text{RCS}_H$  did not significantly change. Furthermore, in the 9th and 10th sprints, the values of  $\text{PPO} \cdot \text{BM}^{-1}$  were significantly higher in  $\text{RCS}_H$  than in  $\text{RCS}_L$ . The index of total workload was significantly lower in  $\text{RCS}_L$  ( $392.1 \pm 39.6$  W) than in  $\text{RCS}_H$  ( $606.1 \pm 76.5$  W) (Fig. 1, bottom). The peak pedaling rate achieved during  $\text{RCS}_L$  and  $\text{RCS}_H$  were ranged from 166 to 215 rpm and from 72 to 135 rpm, respectively.

### *Blood lactate concentration*

No significant interaction ( $P > 0.1$ ) was found in blood  $[\text{La}^-]$ , and no significant condition effect ( $P > 0.1$ ) was found in blood  $[\text{La}^-]$ . In both conditions, the values of blood  $[\text{La}^-]$  were significantly higher in Pre5, Pre9 and Post-Ex than in Rest, and the values of blood  $[\text{La}^-]$  in Pre9 and Post-Ex were significantly higher than the value in Pre5 (Fig. 2).

### *Oxygen uptake immediately before each cycling sprint*

Changes in  $\dot{V}O_2$  immediately before each cycling sprint ( $\text{pre}\dot{V}O_2$ ) during the two RCS tests are shown in Fig. 3. In  $\text{pre}\dot{V}O_2$ , no significant interaction ( $P > 0.1$ ) or significant condition effect ( $P > 0.1$ ) was found. In both conditions,  $\text{pre}\dot{V}O_2$  values before the 2nd, 3rd, 4th, 6th, 7th, 8th and 10th sprints were significantly higher than the value before the 1st sprint and there were no significant differences between  $\text{pre}\dot{V}O_2$  values before the 5th and 9th sprints and that before the 1st sprint.

#### *Surface electromyogram*

Since there was a significant difference in  $\text{PPO}\cdot\text{BM}^{-1}$  between conditions in the 9th sprint, the values of RMS in the 9th sprint were compared to those in the 1st sprint (Fig. 4). When RMS was expressed as a percentage of total number of pedalings, a significant interaction ( $P < 0.05$ ) was found in  $\text{RCS}_L$  but not in  $\text{RCS}_H$ . In  $\text{RCS}_L$ , the values of RMS during 20-80% of the total number of pedalings were significantly higher in the 1st sprint than in the 9th sprint.

### **Discussion**

The main findings in the present study were that 1)  $\text{PPO}\cdot\text{BM}^{-1}$  in the 9th sprint significantly decreased in  $\text{RCS}_L$  compared to that in  $\text{RCS}_H$  in spite of the fact that there was no difference in blood  $[\text{La}^-]$  between the conditions and that 2) the amplitude of SEMG activity (i.e., RMS) significantly decreased in the 9th sprint compared to that in the 1st sprint in only  $\text{RCS}_L$ .

In the present study, to eliminate effects of resynthesis of PCr on SEMG activity, a 360-s recovery period was set immediately before the 5th and 9th sprints. Since Rossiter et al. [18]

reported that restoration of PCr after high-intensity exercise is accompanied by a decrease in  $\dot{V}O_2$  during the recovery period,  $\text{pre}\dot{V}O_2$  measured in the present study roughly predicts the degree of resynthesis of PCr. Since there was no difference in  $\text{pre}\dot{V}O_2$  in Pre5 and Pre9 between the conditions, it seems plausible that the degree of PCr resynthesis over the 360-s recovery periods in  $\text{RCS}_L$  was similar to that in  $\text{RCS}_H$ .

In  $\text{RCS}_L$ , RMS was significantly lower in the 9th sprint than in the 1st sprint, but there was no significant difference in RMS between the 1st sprint and the 9th sprint in  $\text{RCS}_H$ . RMS calculated from SEMG is indicative of the number of motor units (MUs) recruited and/or the rate of discharge of the recruited MUs in exercising muscle [19-20]. It has been reported that the amplitude of the SEMG is influenced by factors such as electrode location, thickness of subcutaneous tissues, and distribution of MU conduction velocities [21]. Therefore, there are some limits to interpretation of the results of SEMG activity in the present study. Nevertheless, it is likely that decline of RMS in the present study reflects the decreased excitation level in MUs since the SEMG activity in the present study was determined in the same position in the same muscles in the two conditions. Since there was no significant difference in blood  $[\text{La}^-]$  between the conditions, it is thought that the contribution of glycolysis to ATP resynthesis during  $\text{RCS}_L$  was equivalent to that during  $\text{RCS}_H$ . Therefore, the rate of decline in intramuscular pH in  $\text{RCS}_L$  was similar to that in  $\text{RCS}_H$ . This idea is supported by the results of a study by Cherry et al. [5]. This indicates that the effect of a decline in intramuscular pH on SEMG activity in  $\text{RCS}_L$  is thought to have been similar to that in  $\text{RCS}_H$ , suggesting that the decreased RMS in the 9th sprint during  $\text{RCS}_L$  resulted from reduced skeletal

muscle recruitment by the CNS. It is highly possible that the reduced skeletal muscle recruitment by the CNS was related to the decrease in  $\text{PPO} \cdot \text{BM}^{-1}$  in the 9th sprint during  $\text{RCS}_L$ .

Studies using an isokinetic cycle ergometer [1, 2, 22] have shown that during a cycling sprint at a high pedaling rate, the relative contribution of fatigue-sensitive fast-twitch fibers to produced power was greater, resulting in greater PPO and greater fatigue. During a cycling sprint on a non-isokinetic cycle ergometer at a light resistive load, pedaling rate increases to over 150 rpm and the power produced during a high pedaling rate over 150 rpm is considerably lower than PPO produced at approximately 120 rpm [3-4]. Since maximal velocity of the shortening of slow-twitch fibers will be exceeded at a pedaling rate of 165 rpm [22], the lower power during a high pedaling rate over 150 rpm would be exerted by fast-twitch fibers. Therefore, it is thought that the majority of total work during  $\text{RCS}_L$  was induced by the contribution of fast-twitch fibers. This greater contribution of fast-twitch fibers during  $\text{RCS}_L$  may be the mechanism underlying the reduction of skeletal muscle recruitment. In the present study, intramuscular pH was not directly measured and index of total work was remarkably different between the conditions. Thus, it is possible that the decline of intramuscular pH was greater during  $\text{RCS}_H$  than during  $\text{RCS}_L$ . However, this idea raises the possibility that the reduced  $\text{PPO} \cdot \text{BM}^{-1}$  and reduced RMS in the 9th sprint during  $\text{RCS}_L$  were not directly associated with metabolic stress, suggesting that the CNS prevents severe metabolic stress or catastrophic damage to muscles and joints by anticipatory regulation of skeletal muscle recruitment. Recovered  $\text{PPO} \cdot \text{BM}^{-1}$  in the 5th sprint may result from lower afferent signals derived from peripheral muscles and joints compared to those in the 9th sprint.

In the present study, SEMG activity was recorded from only the left VL. A recent study has shown an increase in activity for the gluteus maximus and long head of the biceps femoris during a submaximal pedaling exercise performed until exhaustion [23]. Accordingly, the reduced muscle recruitment in the left VL by the CNS may be a part of the muscle coordination.

In conclusion, performance and SEMG activity during RCS on a non-isokinetic cycle ergometer are influenced by resistive load. It is thought that the regulation of skeletal muscle by the CNS is associated with fatigue during RCS with a light resistive load.

Competing Interest: None to declare.

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**Information box**

“What is already known on this topic”

Previous work has suggested that fatigue during repeated cycling sprints is due to peripheral fatigue so that there is no attention paid to the role of central regulation.

“What this study adds”

This study shows that central command falls during repeated cycling sprints with a light resistive load, and that this central regulation is associated with fatigue during repeated cycling sprints.

Figure

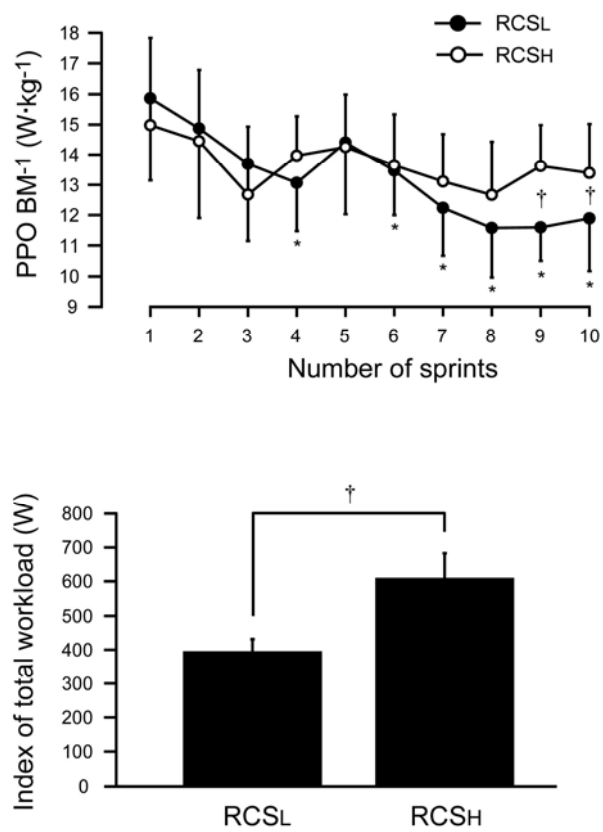
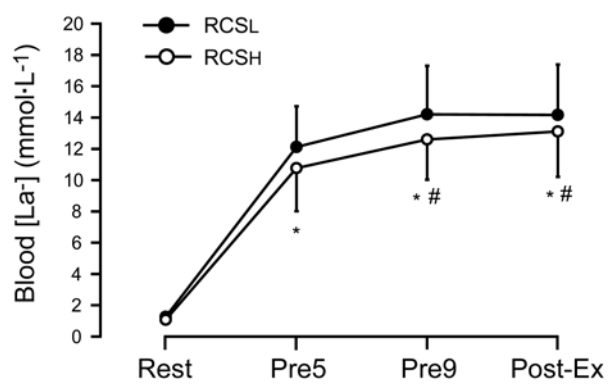
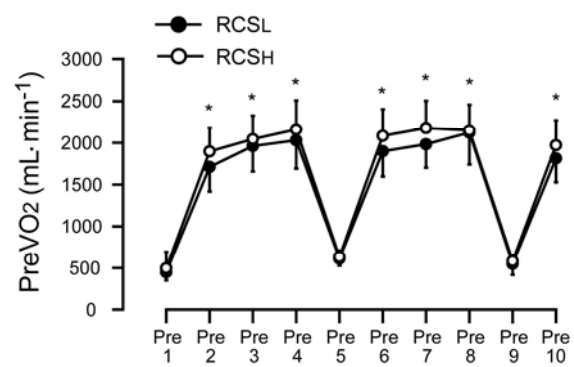


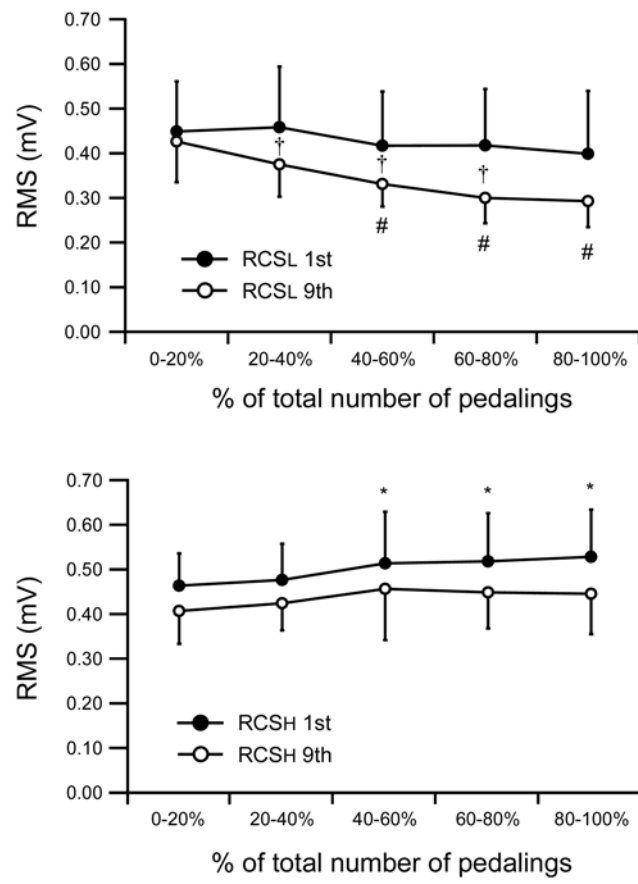
Figure 1



**Figure 2**



**Figure 3**

**Figure 4**

### Figure legends

Fig. 1. Changes in peak power output (PPO) divided by body mass (BM) in each of the subjects ( $\text{PPO} \cdot \text{BM}^{-1}$ ) (top) and the average mean power output (MPO) over the 1st-10th cycling sprints (an index of total workload) (bottom) during two repeated cycling sprints (RCS) with either light load ( $\text{RCS}_L$ : ●) or heavy load ( $\text{RCS}_H$ : ○). \*: significantly different ( $P < 0.05$ ) from the value in the 1st sprint in  $\text{RCS}_L$ . †: significant difference ( $P < 0.05$ ) between the value in  $\text{RCS}_L$  and that in  $\text{RCS}_H$ .

Fig. 2. Changes in blood lactate concentration ( $[\text{La}^-]$ ) at rest (Rest), 30 s before the 5th and 9th sprints (Pre5 and Pre9, respectively), and immediately after the end of RCS (Post-Ex) during two repeated cycling sprints (RCS) with either light load ( $\text{RCS}_L$ : ●) or heavy load ( $\text{RCS}_H$ : ○). \*: significantly different ( $P < 0.05$ ) from Rest in both  $\text{RCS}_L$  and  $\text{RCS}_H$ . #: significantly different ( $P < 0.05$ ) from Pre5 in both  $\text{RCS}_L$  and  $\text{RCS}_H$ .

Fig. 3. Changes in oxygen uptake ( $\dot{V}\text{O}_2$ ) in each of the subjects for 10 s immediately before each of the ten cycling sprints ( $\text{pre}\dot{V}\text{O}_2$ ) during two repeated cycling sprints (RCS) with either light load ( $\text{RCS}_L$ : ●) or heavy load ( $\text{RCS}_H$ : ○). \*: significantly different ( $P < 0.05$ ) from the value immediately before the 1st sprint (Pre1) in both  $\text{RCS}_L$  and  $\text{RCS}_H$ .

Fig. 4. Changes in RMS from the left vastus lateralis (VL) expressed as a percentage of total

number of pedalings in the 1st (●) and 9th (○) sprints during two repeated cycling sprints (RCS) with either light load (RCS<sub>L</sub>: top) or heavy load (RCS<sub>H</sub>: bottom). #: significantly different ( $P < 0.05$ ) from the value during 0-20% of total numbers of pedalings in the 9th sprint in RCS<sub>L</sub>. †: significant difference ( $P < 0.05$ ) between the value in the 1st sprint and that in the 9th sprint during RCS<sub>L</sub>. \*: significantly different ( $P < 0.05$ ) from the value during 0-20% of total numbers of pedalings in both the 1st and the 9th sprints in RCS<sub>H</sub>.

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