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Title

Effects of deception for intensity on SEMG activity and blood lactate concentration during intermittent cycling followed by exhaustive cycling

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Running title

Deception and SEMG activity during intermittent cycling

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Abstract

The purpose of the present study was to determine the effects of deception for exercise intensity on surface electromyogram (SEMG) activity and blood lactate concentration during intermittent cycling exercise (ICE) tests. Sixteen healthy male were randomly assigned to two groups who completed two ICE [three 4-min cycling at 80% peak power output (PPO) with 3-min passive recovery periods followed by exhaustive cycling] tests (ICE-1 and ICE-2). The experimental group (ICE_D) was deceived of the actual cycling intensity, while the control group (ICE_C) was informed of the actual protocol in ICE-2. In ICE-1, both groups were informed of the actual protocol. In ICE-2, root mean square (RMS) calculated from SEMG during submaximal cycling was significantly higher in the ICE_C than in the ICE_D and blood lactate concentration ([La⁻]) was significantly higher in the ICE_C than in the ICE_D. In particular, the difference in RMS between the groups was also observed during the first 4-min cycling, in which there was no difference in [La⁻] between the groups. These results suggest that the CNS modulates skeletal muscle recruitment due to the prior deception for exercise intensity.

Keywords

surface electromyogram, intermittent cycling exercise, deception, exercise intensity, central nervous system, anticipation

Introduction

It is well known that performance in sporting events depends on muscle fatigue. Therefore, interest has been shown in muscle fatigue for more than a century by many sports scientists and exercise physiologists. Muscle fatigue has traditionally been attributable to disability of peripheral or central organs. However, some authors have argued that muscle fatigue results from a protective mechanism rather than the disability. They (5, 7, 9, 18, 19) have suggested that the central nervous system (CNS) modifies efferent signals to skeletal muscle and peripheral organs based on afferent signals from the skeletal muscle and peripheral organs to the CNS to prevent the failure of homeostasis and that the modification is influenced by information involving subsequent exercise. Recently, it has been reported that performance and surface electromyogram (SEMG) activity varied between conditions despite no difference in metabolic state in peripheral skeletal muscle when repeated cycling sprints were performed under light load and heavy load conditions, suggesting that efferent signals from the CNS during repeated cycling sprints were modified by not only afferent feedback from peripheral organs but also information (i.e., frequency of movement) involving subsequent exercise (8). Therefore, it is possible that efferent signals from the CNS to the skeletal muscles during exercise are influenced by prior information involving subsequent exercise.

When consecutive runs were performed at the same intensity but with subjects being told that the runs would be of increasing intensity, subjects who were expecting consecutive runs of increasing work intensity did not report any increase in the sense of fatigue, which was evaluated from ratings of perceived exertion (RPE), of the leg regions with subsequent runs, but during consecutive runs at the same intensity, subjects who were told that the runs would be of the same intensity reported increased RPE with subsequent runs (2). Since the sense of fatigue is believed to arise centrally as a result of a corollary discharge of efferent signals into the sensorium (1, 10), efferent signals from the CNS to peripheral organs during exercise may be modified by prior deception for exercise intensity. Furthermore, Mihevic (11) argued that a number of afferent signals from peripheral organs to the CNS mediate sense of fatigue induced by exercise. Therefore, it is possible that the deception for exercise intensity used in the study of Hampson et

al. (2) influenced efferent signals from the CNS to skeletal muscles and that in turn influenced the metabolic state in skeletal muscles.

Efferent signals from the CNS to skeletal muscles are generally estimated by SEMG recordings. An increase in efferent signals induces increases in the number of motor units (MUs) that are active and/or the discharge rate of the active MUs, resulting in increase in the amplitude of SEMG (6, 13). The increase in the amplitude of SEMG was reported to be associated with an increase in the sense of fatigue (4). Therefore, the purpose of the present study was to determine effects of deception for exercise intensity on SEMG activity and blood lactate concentration ($[La^-]$) during intermittent submaximal cycling followed by exhaustive supramaximal cycling. If the deception for exercise intensity during intermittent submaximal cycling influences efferent signals from the CNS during exercise, there will be differences in SEMG activity and blood $[La^-]$ between conditions.

In the present study, intermittent submaximal cycling was followed by exhaustive supramaximal cycling. Some authors (5, 8, 19) have suggested that efferent signals are modified on the basis of afferent signals and information involving subsequent exercise in order to prevent premature fatigue and excessive damage. Thus, it is likely that exhaustive supramaximal cycling used in the present study will highlight this modification.

Materials and Methods

Sixteen healthy males participated in the present study. The participants' mean age, height, weight, and body mass index (BMI) were 22.7 ± 2.0 (S.D.) yr, 171.8 ± 5.8 cm, 66.4 ± 7.4 kg, and 22.5 ± 1.7 kg/m², respectively. They had no neuromuscular or metabolic disorders and were participating in regular training programs. The participants were all non-smokers. Each participant signed a statement of informed consent prior to any testing procedure. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study. Participants were instructed to refrain from intense physical exercise, drinking, and taking caffeine and not to change their dietary habits for 24 h prior to each visit.

Each participant attended our laboratory for four tests. The time interval between two consecutive tests was at least 48 hours, and all tests were completed within 3 weeks. On the first test day, the participants' body characteristics were measured and each participant performed two 3-min cycling tests at 40–70 W at 60 and 100 rpm on a cycle ergometer in which the power output could be adjusted by a computer (232CXL, Combi, Tokyo, Japan) to become familiarized with cycling at 60 and 100 rpm. On the second test day, each participant completed an incremental cycling test on the cycle ergometer to volitional exhaustion to determine the peak oxygen uptake ($\text{VO}_{2\text{peak}}$) and peak power output (PPO) and to establish the low and high rating anchors of RPE. Participants began cycling at 60 W for 2 min. Power output was increased in incremental steps of 30 W every 2 min until exhaustion. Participants were constrained to maintain pedalling rate at 60 rpm during the warming-up and incremental cycling test with the aid of a metronome. $\text{VO}_{2\text{peak}}$ was defined as the peak value of oxygen uptake (VO_2) during the incremental test. PPO was calculated as follows:

$$\text{PPO} = W_{\text{completed}} + [(t/120) \times 30],$$

where $W_{\text{completed}}$ is the power output (W) in the final completed stage, and t is the time (s) spent in the final noncompleted stage.

All participants performed two intermittent cycling exercise (ICE) tests (ICE-1 and ICE-2) using the cycle ergometer on separate days. Each participant'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. Each participant rested for 3 min on the cycle ergometer and then warmed up for 4 min at 40% PPO (60 rpm). ICE began 3 min after the warming-up. ICE consisted of three 4-min submaximal cycling exercises at 80% PPO (Ex-1, Ex-2 and Ex-3, 60 rpm) with 3-min passive recovery periods followed by exhaustive supramaximal cycling exercise at 120% PPO (SMC, 100 rpm). The recovery period between Ex-3 and SMC was 15 s. The total participant pool was randomly divided into a control group (ICE_C , $n = 8$) and deception group (ICE_D , $n = 8$). The ICE_C included participants who were correctly informed that all 4-min cycling exercises would be completed at 80% PPO (206 ± 29 W) on both the third and fourth test days (i.e., ICE_{C-1} and ICE_{C-2} , respectively). Participants in the ICE_D were informed that all 4-min cycling exercises would be completed at 80% PPO (206 ± 25 W) on the third test

day (ICE_D-1), and on the fourth test day (ICE_D-2) they were told that they would perform at 80%, 83% (214 ± 26 W), and 86% (222 ± 26 W) PPO, while they actually performed all 4-min cycling exercises at 80% PPO. Since the screen on the cycle ergometer displayed the power output, the screen was covered so as not to show data to the participants.

Blood samples (25 μ L) were collected from fingertips using capillary tubes and analyzed using a lactate analyzer (YSI 1500 SPORT, YSI, Yellow Springs, OH, USA) to measure $[La^-]$. The lactate analyzer was calibrated by a standard lactate solution of 5 mmol/L before each test. Blood was sampled at rest, 30 s before Ex-1, Ex-2 and Ex-3, and immediately after the end of Ex-3.

Data on VO_2 were obtained breath-by-breath using a respiratory gas analyzer (AE-280S, Minato Medical Science Co., Ltd., Osaka, Japan). Ventilation (VE) was measured by a hot-wire flow meter, and the flow meter 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%). VO_2 was measured continuously during rest, exercise, and recovery periods. For each 15-s interval, the average of VO_2 was calculated.

An SEMG was recorded from the right vastus lateralis (VL) at a rate of 1000 Hz during the ICE. Before attachment of the surface electrodes, the skin was shaved, abraded, and cleaned with alcohol in order to reduce skin impedance. Bipolar surface EMG sensors (SX230, Biometrics Ltd., Gwent, Wales, UK; inter-electrode distance of 20 mm) were 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 left wrist. The SEMG signals were amplified using an amplifier imbedded in the EMG sensor (bandwidth = 20–450 Hz; CMRR >96 dB; input impedance $>10^{13} \Omega$; gain = 1000) and converted into digital signals using an analog-digital converter (MacLab/8s, ADInstruments, Bella Vista, NSW, Australia). Then SEMG data were processed offline by using analysis software (LabChart v7.1 for Windows, ADInstruments, Bella Vista, NSW, Australia). The SEMG activity during the ICE was determined by measuring the root mean square (RMS) of the signal for 20 s every 1 min (i.e., 40–60 s, 100–120 s, 160–180 s and 220–240 s) during the cycling exercise. The RMS data

were normalized to the value obtained from 10 to 30 s in Ex-1. Electrode placement was marked on the skin surface to facilitate further recording from the same site during the subsequent visit.

Deep tissue temperature of the left VL was measured at a rate of 0.5 Hz using a deep-tissue temperature monitor (CM-210, Terumo Corp., Tokyo, Japan). The probe (CM-210PD1, Terumo Corp., Tokyo, Japan; diameter of 45 mm) was placed on the lateral side of the crural area five-fingers proximal from the patella of the belly of the VL. The depth of measurement was 10 mm from the skin. For each 10-s interval, the average of deep tissue temperature was calculated.

Each participant was asked to assess the sense of fatigue for overall body and legs immediately before and after each 4-min submaximal cycling exercise (warming-up, Ex-1, Ex-2, and Ex-3) during the ICE utilizing Borg's 15-point RPE scale (6–20) and the Borg category-ratio scale (0–10), respectively. During recovery, the sense of fatigue was defined as the intensity of effort, strain, discomfort, and/or fatigue that remained after exercise (21).

Results are presented as means $\pm$ standard deviation (S.D.). For RMS, $[La^-]$, VO_2 , deep tissue temperature and RPE, two-way analysis of variance (ANOVA) with repeated measures was performed with time and group in each day. All variables were examined using Mendoza's multisample sphericity test. Whenever the data violated the assumption of sphericity, P values based on the Greenhouse-Geisser correction were reported. After ANOVA, Shaffer's modified sequentially rejective Bonferroni procedure was performed for multiple comparisons. In order to compare the characteristics of participants and the time to exhaustion in SMC, t -test was performed. $P < 0.05$ was regarded as statistically significant.

Results

Descriptive characteristics of participants are shown in Table I. There were no significant differences between the groups for age, height, weight, BMI, PPO, 80% PPO, 120% PPO, and VO_{2peak} .

****Table I near here****

Fig. 1 shows changes in RMS calculated from SEMG activity in the right VL in the two groups in each day. In each 4-min cycling (i.e., Ex-1, Ex-2, and Ex-3) during ICE-1, no significant interaction ($P > 0.05$) or significant group effect ($P > 0.05$) was found in RMS, and RMS significantly increased over time in the two groups ($P < 0.05$). In Ex-1 and Ex-2 during ICE-2, a significant interaction ($P < 0.05$) was found in RMS, and RMS in the ICE_C was significantly higher than that in the ICE_D at 13, 14, 20, and 21 min in the ICE ($P < 0.05$). In Ex-3 during ICE-2, no significant interaction ($P > 0.05$) or significant group effect ($P > 0.05$) was found in RMS, and RMS significantly increased over time in the two groups ($P < 0.05$).

****Figure 1 near here****

Fig. 2 shows changes in blood [La⁻] in the two groups in each day. During ICE-1, no significant interaction ($P > 0.05$) or significant group effect ($P > 0.05$) was found in blood [La⁻]. Blood [La⁻] significantly increased over time in the two groups during ICE-1 ($P < 0.05$). During ICE-2, a significant interaction ($P < 0.05$) was found in [La⁻], and blood [La⁻] in the ICE_C was significantly higher than that in the ICE_D ($P < 0.05$) immediately before Ex-2 and Ex-3. Blood [La⁻] significantly increased over time in the two groups during ICE-2 ($P < 0.05$).

****Figure 2 near here****

There was no significant difference in VO₂ (Fig. 3), deep tissue temperature of the VL (Fig. 3), and RPE for overall body (Fig. 4) and legs (Fig. 4) during both ICE-1 and ICE-2 between the two groups. The time to exhaustion in SMC was similar in the ICE_D and ICE_C

during both ICE-1 (ICE_D-1: 33.8 ± 12.2 s, ICE_C-1: 33.9 ± 12.8 s; $P > 0.05$) and ICE-2 (ICE_D-2: 39.5 ± 16.0 s, ICE_C-2: 38.4 ± 12.4 s; $P > 0.05$).

****Figure 3 near here****

****Figure 4 near here****

Discussion

The main findings in the present study were that 1) SEMG activity in the VL and blood [La⁻] were significantly lower in ICE_D-2, in which participants expected that they would perform at 80%, 83%, and 86% of PPO, than in ICE_C-2 and that 2) the lower SEMG activity preceded the lower blood [La⁻] during ICE_D-2.

In the present study, VO₂ measured in ICE-1 and ICE-2 was not different between the deception group and the control group. Therefore, it is estimated from the similar VO_{2peak} and PPO in the two groups that not only physical but also physiological exercise intensity was the same in the two groups. In Ex-1 and Ex-2 during ICE-2, RMS was significantly lower in the deception group than in the control group. 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 (6, 13). Furthermore, peripheral fatigue during submaximal contractions has been shown to increase the number of MUs recruited and/or the rate of discharge of the recruited MUs, resulting in increase in RMS (12). Since there was no difference in blood [La⁻] immediately before Ex-1 between ICE_D-2 and ICE_C-2, suggesting that there was no difference in the rate of metabolic acidosis immediately before Ex-1 between the groups, it is likely that the difference in values of RMS in Ex-1 between ICE_D-2 and ICE_C-2 resulted from a difference in central neural output rather than a difference in effects of metabolic state on neuromuscular functions. Therefore, the lower RMS in Ex-1 during ICE_D-2 resulted from a smaller number of MUs recruited and/or a lower rate of discharge of recruited MUs (fatigue-resistive MUs), suggesting that deception for exercise intensity preserved fatigable MUs during ICE. This

modification of skeletal muscle recruitment by deception for exercise intensity may be associated with modification of efferent signals induced by information involving subsequent exercise (8). In the present study, three 4-min submaximal cycling exercises were followed by exhaustive supramaximal cycling exercise at 120% PPO (i.e., SMC). Accordingly, the CNS might have intended to delay severe metabolic stress to optimize performance during the SMC in ICE_D-2, in which the participants expected that progressive increase in exercise intensity during the three 4-min cycling exercises would result in severer metabolic stress. The possibility that muscle temperature influences RMS calculated from SEMG has been reported (16, 17, 20). However, since deep tissue temperature of the left VL was the same in the two groups, it is likely that the influence of muscle temperature on RMS was negligible in the present study.

Nevertheless, the significant difference in RMS between the two groups was diminished in Ex-3 during ICE-2, and blood [La⁻] at the end of Ex-3 in ICE-2 was the same in the two groups. Since blood [La⁻] increased up to very high level (approximately 8 mmol/L), it is possible that the modification of skeletal muscle recruitment by deception for exercise intensity could not maintain the power output (80% PPO) during Ex-3 in ICE-2 and that the number of fatigable MUs active was increased during Ex-3 in ICE_D-2, resulting in no difference in blood [La⁻] at the end of Ex-3 in ICE-2 between the two groups. Although we cannot rule out the possibility that the difference in [La⁻] was due to differences in participant characteristics (e.g., muscle fiber composition, blood capillary density, and enzyme activity), the results of RMS in Ex-1 during ICE-2 highlight the modulation of skeletal muscle recruitment by deception for exercise intensity.

Despite the fact that there were significant differences in both RMS in Ex-1 and Ex-2 and blood [La⁻] immediately before Ex-2 and Ex-3, RPE for overall body and legs immediately before Ex-2 and Ex-3 and immediately after Ex-1 and Ex-2 were the same between the two groups. Although it has been reported that a number of afferent signals from peripheral organs to the CNS mediate sense of fatigue (11) and sense of effort is thought to arise centrally as a result of a corollary discharge of a motor command signal (14), RPE for overall body and legs did not follow changes in RMS and blood [La⁻]. This inconsistency may be explained by an idea proposed by Lambert and colleagues (5). They suggested that afferent signals from skeletal

muscle and other organ systems are interpreted by the CNS against expected outcomes, resulting in the conscious perception of effort. In the present study, although skeletal muscle recruitment was modified to preserve fatigable MUs by deception for exercise intensity, blood $[La^-]$ increased to a very high level during ICE_D-2. Therefore, RPE for overall body and legs measured in the present study might be determined by an interaction between afferent signals from skeletal muscle and efferent signals from the CNS.

Care must be taken in interpreting the results for SEMG activity. Hunter and Enoka (3) have shown that practice of a sustained submaximal isometric contraction with the elbow flexor muscles enabled some men and women (subjects classified as responders) to prolong the endurance time to exhaustion due to a reduced rate of increase in SEMG activity in the biceps brachii muscle. These results seem comparable to SEMG activity during ICE_D in the present study. Thus, it is possible that the reduced rate of increase in RMS calculated by SEMG activity in the VL was associated with a practice effect. Since one difference between the two studies was that the present study applied dynamic contractions rather than static contractions, it is difficult that the results for SEMG activity in the present study are fully explained for the interpretation in Hunter and Enoka (3). Furthermore, the interpretation that deception for exercise intensity preserved fatigable MUs during ICE raises a significant question. How the ICE_D group could bring the same effort (80% PPO) with MUs that fire at a lower rate compared to the ICE_C group? A decreased inhibitory reflex from the branch of the radial nerve that innervates brachioradialis to biceps brachii motor neurons contributed to the reduced rate of increase in SEMG activity during a sustained submaximal isometric contraction (15). It remains unclear whether there is an inhibitory reflex pathway from the branch of nerve innervates knee extensor muscles to the VL motor neurons. However, a cycling exercise needs alternate activations of knee extensor and flexor muscles. Since activation of knee flexor muscles simultaneously inhibits knee extensor motor neurons (i.e., reciprocal inhibition), deception for exercise intensity may modulate coordination of muscle activation during cycling, resulting in prevention of excessive recruitment of the VL motor neurons. A major limitation of the present study was failure to record SEMG activity in other (biceps femoris, gastrocnemius, tibialis anterior and gluteus medius) muscles that contribute to cycling.

In conclusion, deception for exercise intensity partially reduces the amplitude of SEMG activity and blood lactate concentration during intermittent submaximal cycling followed by exhaustive supramaximal cycling. It is thought that the CNS modulates skeletal muscle recruitment due to the prior deception for exercise intensity.

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Table

Table I. Participant characteristics

Group	Age (yr)	Height (cm)	Weight (kg)	BMI (kg/m ²)	PPO (W)	80% PPO (W)	120% PPO (W)	VO _{2peak} (mL/kg/min)
Control (<i>n</i> = 8)	22.9 (1.6)	171.2 (6.3)	65.7 (5.7)	22.4 (1.4)	257.2 (36.8)	205.8 (29.4)	308.6 (44.1)	46.6 (4.5)
Deception (<i>n</i> = 8)	22.5 (2.4)	172.5 (5.6)	67.2 (9.2)	22.5 (2.0)	257.8 (30.8)	206.3 (24.6)	309.4 (36.9)	45.9 (6.3)

Values are expressed as means (SD). Abbreviations: BMI, body mass index; PPO, peak power output; VO_{2peak}, peak oxygen uptake

Figure legends

Fig. 1. Changes in RMS calculated from SEMG of the right VL normalized by the value obtained from 10 to 30 s in first 4-min cycling (Ex-1) during the two ICE tests (ICE-1 and ICE-2) in the two groups (ICE_C: filled circles, ICE_D: open circles). *: significant time effect ($P < 0.05$). †: significant group effect ($P < 0.05$).

Fig. 2. Changes in blood [La⁻] during the two ICE tests (ICE-1 and ICE-2) in the two groups (ICE_C: filled circles, ICE_D: open circles). †: significant group effect ($P < 0.05$).

Fig. 3. Changes in oxygen (O₂) uptake and deep tissue temperature of the left VL during the two ICE tests (ICE-1 and ICE-2) in the two groups (ICE_C: filled circles, ICE_D: open circles).

Fig. 4. Changes in RPE for overall body and legs during the two ICE tests (ICE-1 and ICE-2) in the two groups (ICE_C: filled circles, ICE_D: open circles).

Figures

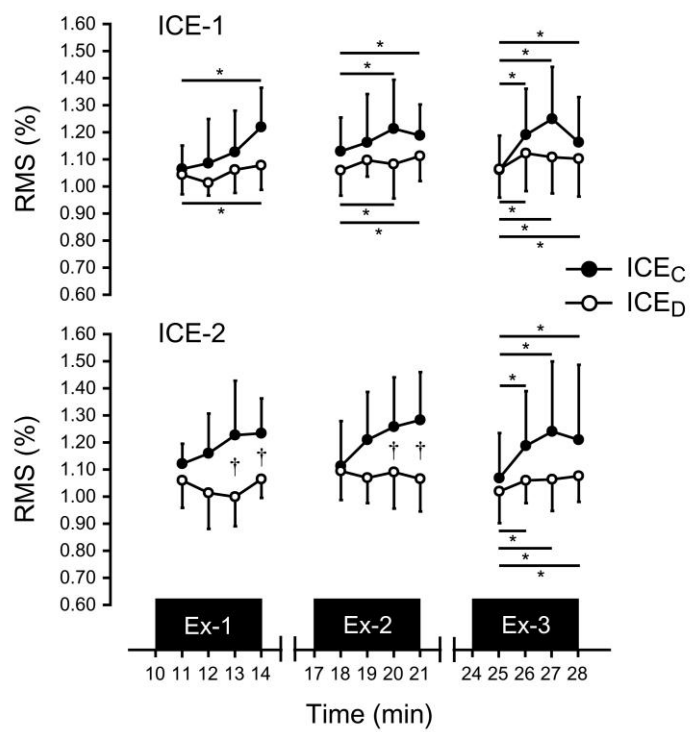


Figure 1

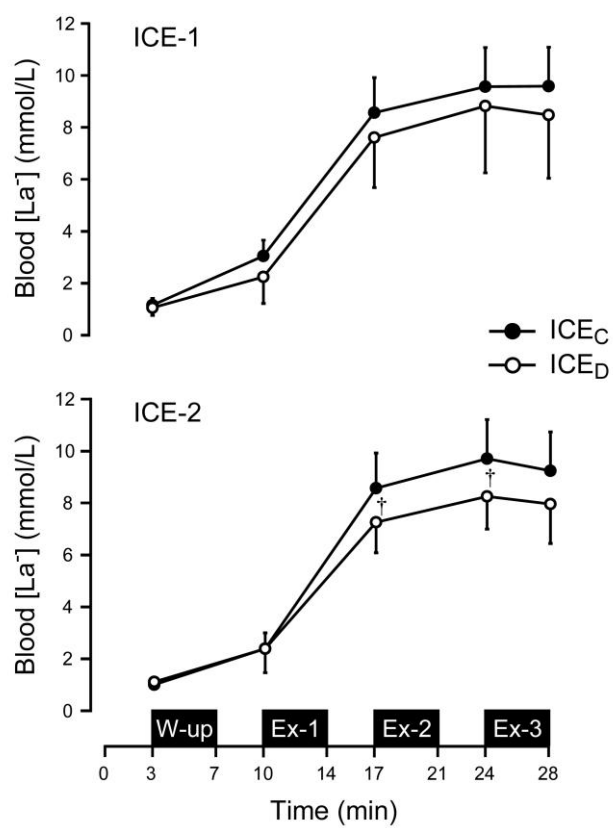


Figure 2

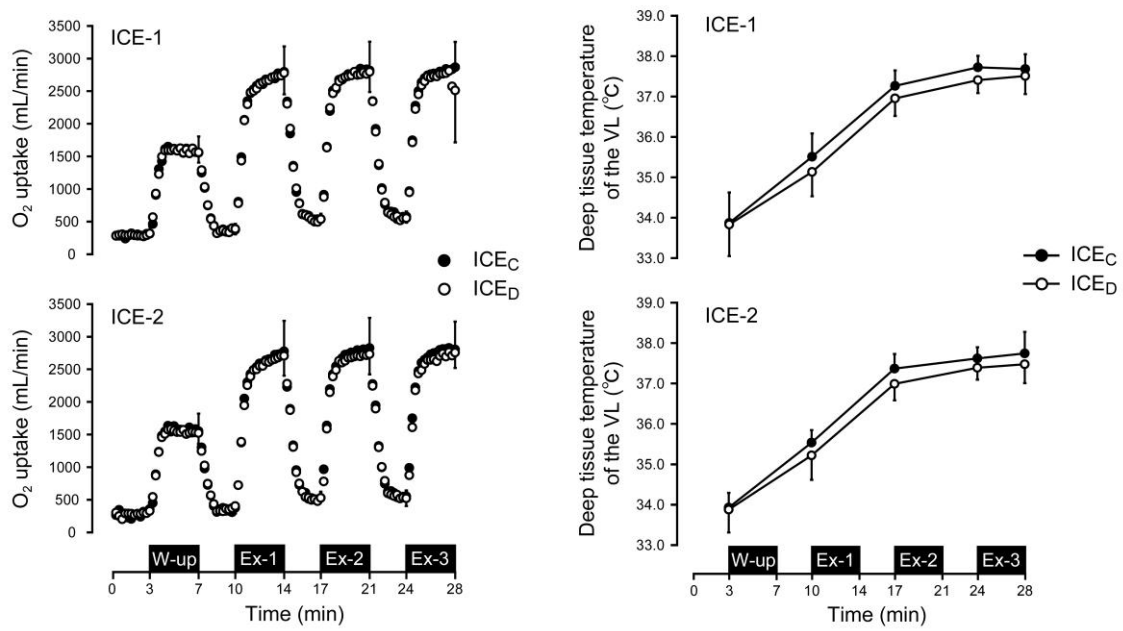


Figure 3

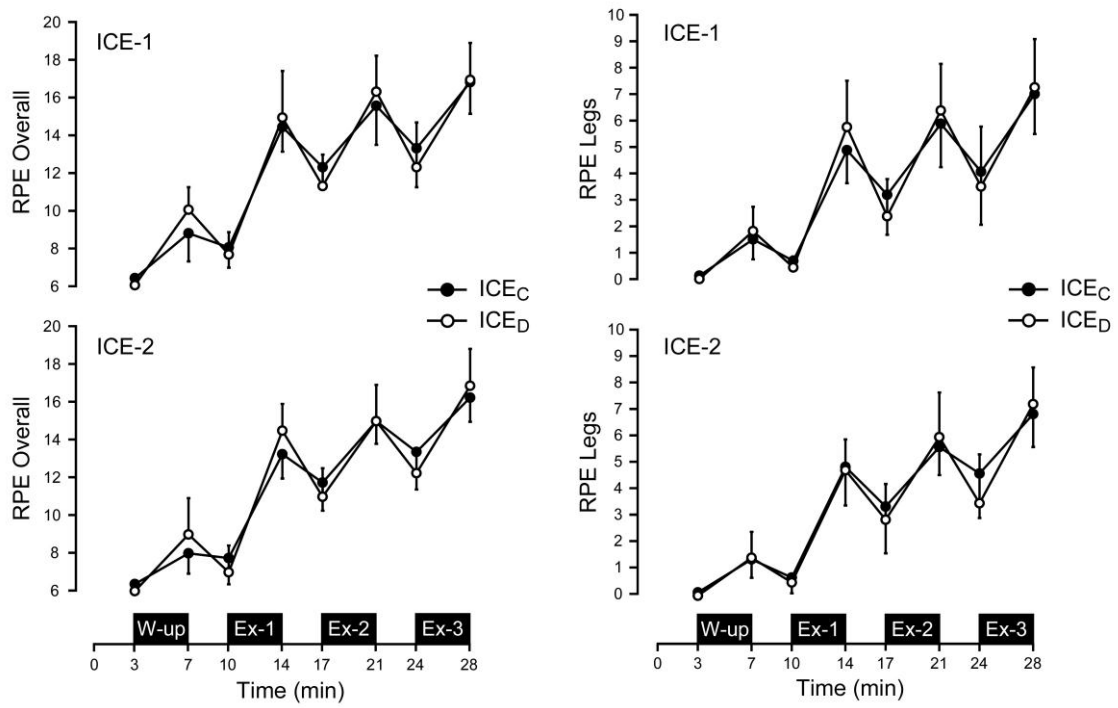


Figure 4