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Effects of heat exposure in the absence of hyperthermia on power output during repeated cycling sprints

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ABSTRACT: The aim of this study was to investigate the effects of heat exposure in the absence of hyperthermia on power output during repeated cycling sprints. Seven males performed four 10-s cycling sprints interspersed by 30 s of active recovery on a cycle ergometer in hot-dry and thermoneutral environments. Changes in rectal temperature were similar under the two ambient conditions. The mean 2-s power output over the 1st–4th sprints was significantly lower under the hot-dry condition than under the thermoneutral condition. The amplitude of the electromyogram was lower under the hot-dry condition than under the thermoneutral condition during the early phase (0–3 s) of each cycling sprint. No significant difference was observed for blood lactate concentration between the two ambient conditions. Power output at the onset of a cycling sprint during repeated cycling sprints is decreased due to heat exposure in the absence of hyperthermia.

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INTRODUCTION

During repeated short-duration sprints, peak power output decreases when the recovery periods are short (approximately 30 s) [1,2,3]. Additionally, in the case of repeated sprints with short-recovery periods (15 or 20 s [4,5]), the decrease of peak power output is greater in a hot environment than in a thermoneutral environment. Larger decreases in peak power output have been observed in hot environments when the core temperature of participants reaches hyperthermic levels (approximately 39°C). When the core temperature fails to reach hyperthermic levels (approximately 38°C) during repeated sprints with long recovery periods (5 or 30 min [6,7]), peak power output does not differ between hot and thermoneutral environments. It has been inferred from these results that the effect of heat exposure on peak power output during repeated sprints depends on the core temperature. However, the effect of heat exposure in the absence of hyperthermia on peak power output during repeated sprints with short recovery periods has not been investigated despite the fact that peak power output during repeated sprints is also influenced by duration of the recovery period [1,8]. Since it is well known that heat exposure can lead to cardiovascular strain in the absence of hyperthermia [6], cardiovascular strain induced by heat exposure

may impair peak power output during repeated sprints only if the recovery periods are short. If the duration of the recovery period is related to the effect of heat exposure on peak power output, peak power output during repeated cycling sprints (RCS) with short recovery periods (e.g., 30 s) will be lower under hot conditions than under thermoneutral conditions, even in the absence of hyperthermia. In addition, cognitive functions are immediately impaired during excessive heat exposure [9]. It is possible that impairment in cognitive functions slows the optimal response to the onset of sprinting, resulting in suboptimal motor command. Therefore, heat exposure in the absence of hyperthermia may impair optimal muscle activities at the onset of sprinting. It is likely that this suboptimal muscle activity influences power output profiles during RCS under hot ambient conditions.

The aim of this study was to investigate the effects of heat exposure in the absence of hyperthermia on peak power output and power output profiles during RCS with short recovery periods. We hypothesized that peak power output and power output profiles during RCS would be influenced by heat exposure in the absence of hyperthermia.

MATERIALS AND METHODS

Subjects. Seven healthy male volunteers (age, 22.7 ± 3.5 years; height, 170.4 ± 5.7 cm; body mass, 65.4 ± 5.5 kg) participated in the study. The subjects were participating in regular training programmes (2 h swimming or cycling 2–3 times a week). All subjects gave written informed consent, and the experimental procedures were carried out in accordance with the Declaration of Helsinki. The ethics committee of Hokkaido University Graduate School of Education approved this study.

Design

Following preliminary tests (familiarization with RCS), each participant completed two RCS under either hot-dry conditions (40°C , 20% relative humidity; HOT) or thermoneutral conditions (20°C , 40% relative humidity; CON). All tests were conducted at the same time of day, with at least a 3-day interval between tests, in a randomized order. All subjects were instructed to avoid intense physical exercise, alcohol drinking, and caffeine for 24 h before each test.

Experimental protocol

Each subject arrived at the laboratory 1 h before the start of the test. Experimental instructions were given to each participant before entering the climatic chamber. Immediately upon entering the climatic chamber, subjects were instructed to rest for 3 min on the cycle ergometer (POWERMAX-V_{II}; Combi, Tokyo, Japan). Next, each subject completed a 5-min warm-up (0 W , 100 min^{-1}) followed by the RCS test. The RCS test consisted of four 10-s cycling sprints (resistive load (N): body mass (kg) $\cdot 0.075 \cdot 9.81$) with 30-s active recovery periods (0 W , 100 min^{-1}) between each sprint. Subjects' 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. For all tests, subjects were in the seated position during exercise and recovery. All sprints started from 100 min^{-1} . Subjects were instructed to pedal as many revolutions as possible during each cycling sprint.

Cycling sprint

Pedal cadence during each cycling sprint was recorded every 0.1 s using data collection software (Combi, Tokyo, Japan) that connected the ergometer to a personal computer. Power output during each cycling sprint was calculated from the effective load of the cycle ergometer using the methods described by Lakomy [10]. Briefly, the effective load is the load required to prevent acceleration of the flywheel and consists of not only the resistive load that is applied to the flywheel but also the excess load that is required to stop the subject from accelerating the flywheel at any instant. Excess load was calculated from the acceleration of cadence, which was calculated from the mean cadence measured over 1-s intervals. The instantaneous product of the mean 1-s cadence and effective load was used to determine power output throughout the RCS test as follows:

$$\text{Power output (W)} = 6 \cdot \text{cadence (s}^{-1}\text{)} \cdot \text{effective load (N)},$$

where 6 is the distance the flywheel moves over a 360-degree roll (m). To reduce variation due to differences in the physical characteristics of subjects, power output per unit body mass was used as the dependent variable. The maximum power output during each sprint is referred to as the peak power output.

Surface electromyogram

A surface electromyogram (SEMG) was recorded from the left vastus lateralis (VL) at a rate of 1000 Hz during each of the four cycling sprints. Before attaching the surface electrodes, the skin was shaved, abraded, and cleaned with alcohol to reduce skin impedance. A bipolar SEMG sensor (SX230; inter-electrode distance of 20 mm; Biometrics Ltd., Gwent, Wales, UK) 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 fibres. The ground electrode was placed over the styloid process of the right wrist. SEMG signals were amplified using an amplifier imbedded in the EMG sensor (bandwidth: 20–450 Hz; common mode rejection ratio: $>96\text{ dB}$; input impedance: $>10^{13}\ \Omega$; gain: 1000) and converted into digital signals using an analogue-digital converter (MacLab/8s; ADInstruments, Bella Vista, Australia). Next, SEMG data were processed offline using analysis software (LabChart v7.2.2 for Windows; ADInstruments, Bella Vista, Australia). SEMG activity during RCS was determined by measuring the root mean square (RMS) of the signals between the onset and end of each burst [11]. In the present study, the mean values of RMS for 1–7 pedal revolutions (corresponding to 0–3 s) during each cycling sprint were analysed. The ratio between power output (in absolute values W) at 2 s and RMS activity was calculated as the index of neuromuscular efficiency. The positions of the electrodes for SEMG detection were similar under both conditions because reference points were marked on the skin.

Blood lactate concentration

Blood samples ($25\ \mu\text{L}$) were collected from the fingertips using capillary tubes and analysed using a lactate analyser (YSI 1500 SPORT; YSI, Yellow Springs, OH, USA) to determine the blood lactate concentration (La). The lactate analyser was calibrated using a $5\text{-mmol} \cdot \text{L}^{-1}$ standard lactate solution before each test. Blood was sampled at rest (Rest), 30 s before the 1st sprint (Pre), and 5 min after the end of RCS (Post).

Rectal temperature

Each subject inserted a rectal probe (LT-ST08-11, accuracy: 0.01°C) approximately 10 cm beyond the anal sphincter to allow continuous measurement of core body temperature 10 min before entering the climatic chamber. Rectal temperature (T_{rec}) was recorded continuously during rest before and after entering the climatic chamber, exercise, and recovery periods using a data logger (LT-8; Gram Corporation, Saitama, Japan) at a rate of 1 Hz. For each 10-s interval, mean T_{rec} was calculated. The values at 1 min before entering the

climatic chamber (Rest) and immediately before each sprint were used for further analysis.

Heart rate

Heart rate (HR) was recorded using an HR monitor installed in a respiratory gas analyser (AE-280S; Minato Medical Science, Osaka, Japan). HR was measured continuously during rest, exercise, and recovery periods. For each 10-s interval, mean HR was calculated. The values from 30 s before the 1st sprint to 10 s after the 4th sprint were used for further analysis.

Rate of perceived exertion

Each subject's rate of perceived exertion (RPE) was recorded 10 s before each sprint (pre-sprint RPE) using the 6–20 Borg scale.

Statistical analysis

Results are presented as means $\pm$ standard deviations (SD). Three-way within-subjects ANOVA for power output profiles was performed to study the effects of condition, number of sprints, and elapsed time. Two-way within-subjects ANOVA for peak power output, RMS, neuromuscular efficiency, pre-sprint RPE, La, HR, and T_{rec} was performed to study the effects of condition and time. 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. The level of significance was set at $p < 0.05$.

RESULTS

Peak power output decreased with repeated sprints ($F_{(1.35,8.09)} = 12.68$, $p < 0.01$; Figure 1) but displayed neither a condition effect ($F_{(1,6)} = 0.02$, $p = 0.88$) nor any significant relationship between time and environment ($F_{(3,18)} = 0.43$, $p = 0.74$). With regard to power output profiles, no significant three-way interaction was found ($F_{(27,162)} = 1.04$, $p = 0.42$), but significant two-way interactions were observed (condition and elapsed time during a sprint: $F_{(2.34,14.05)} = 4.96$, $p = 0.02$; number of sprints and elapsed time during a sprint: $F_{(27,162)} = 2.61$, $p < 0.01$). At 2 s, the mean power output over the 1st–4th sprints was lower under HOT ($12.2 \pm 1.5 \text{ W} \cdot \text{kg}^{-1}$) than under CON ($12.6 \pm 1.4 \text{ W} \cdot \text{kg}^{-1}$) ($F_{(1,6)} = 9.23$, $p = 0.02$), but at 5 and 6 s, the mean power output over the 1st–4th sprints was higher under HOT (5 s: $11.1 \pm 0.7 \text{ W} \cdot \text{kg}^{-1}$, 6 s: $10.5 \pm 0.7 \text{ W} \cdot \text{kg}^{-1}$) than under CON (5 s: $10.8 \pm 0.5 \text{ W} \cdot \text{kg}^{-1}$, 6 s: $10.0 \pm 0.7 \text{ W} \cdot \text{kg}^{-1}$) (5 s: $F_{(1,6)} = 7.00$, $p = 0.04$; 6 s: $F_{(1,6)} = 9.44$, $p = 0.02$; Figure 1).

With regard to RMS, there was neither a time effect ($F_{(1.88,11.27)} = 0.51$, $p = 0.60$) nor any significant relationship between time and environment ($F_{(3,18)} = 0.19$, $p = 0.90$). RMS displayed a condition effect (HOT < CON: $F_{(1,6)} = 36.43$, $p < 0.01$; Figure 2). Neuromuscular efficiency decreased over time ($F_{(3,18)} = 7.83$, $p < 0.01$), with significantly higher values recorded under HOT than under CON ($F_{(1,6)} = 27.84$, $p < 0.01$; Figure 2).

There was no significant relationship between time and condition ($F_{(2.59,15.53)} = 1.65$, $p = 0.22$; Figure 3) for HR. HR displayed a time effect ($F_{(3.19,19.13)} = 87.96$, $p < 0.01$) but did not display a condition effect ($F_{(1,6)} = 0.30$, $p = 0.61$). T_{rec} gradually increased under both conditions but the increase did not reach a significant level ($F_{(1.1,6.63)} = 4.12$, $p = 0.08$; Figure 3). Pre-sprint RPE ($F_{(1.14,6.82)} = 60.32$, $p < 0.01$) and La ($F_{(1.04,6.25)} = 617.29$, $p < 0.01$) increased under both conditions (Figure 3).

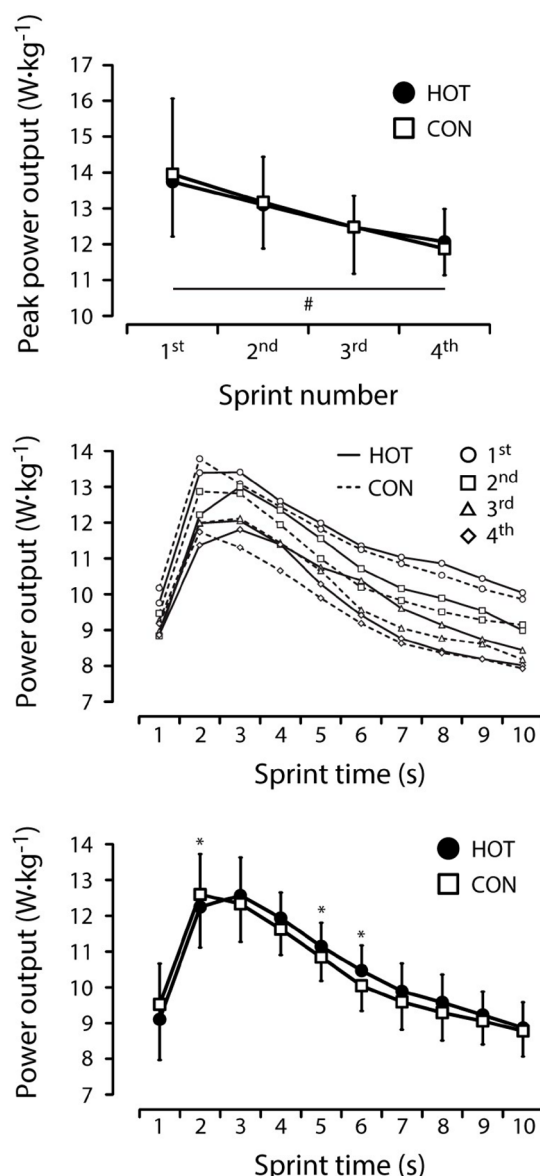


FIG. 1. Top: Peak power output (PPO) divided by body mass (BM) of each subject ($\text{PPO} \cdot \text{BM}^{-1}$) was significantly decreased during repeated cycling sprints (RCS) under hot-dry (filled circles) and thermoneutral (open squares) conditions (HOT and CON, respectively). Middle: Profiles of power output divided BM during the 1st (circles), 2nd (squares), 3rd (triangles), and 4th (diamonds) cycling sprints in RCS under HOT (solid lines) and CON (dashed lines). Bottom: Mean power output profiles over the 1st–4th sprints in RCS under HOT (filled circles) and CON (open squares). Note: *: significant difference at a given time point between the two conditions ($p < 0.05$). #: significant time effect ($p < 0.01$)

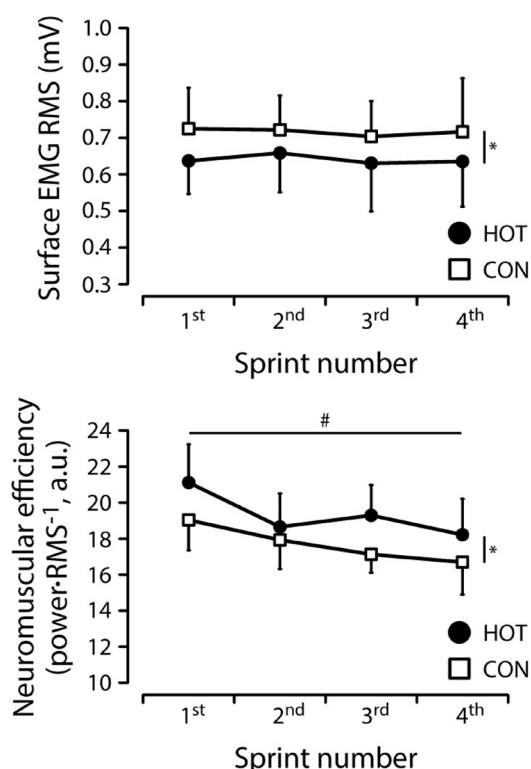


FIG. 2. Top: Root mean square (RMS) calculated from surface electromyogram (SEMG) activity during repeated cycling sprints (RCS) under hot-dry (filled circles) and thermoneutral (open squares) conditions (HOT and CON, respectively). Bottom: Neuromuscular efficiency (power·RMS⁻¹) during RCS under HOT (filled circles) and CON (open squares).

*: significant condition effect ($p < 0.01$)

#: significant time effect ($p < 0.01$)

DISCUSSION

The main findings of the present study were that power output and RMS at the onset of a sprint during RCS were lower under HOT than under CON.

As expected, power output at the onset of a sprint was lower under HOT. In parallel, RMS during the early phase (0–3 s) of RCS was lower under HOT than under CON. Since the amplitude of SEMG activity (i.e., RMS) was interpreted as an indication of central motor command [12,13], it is inferred that neural drive to the VL was suboptimal during the early phase of RCS under HOT. This suboptimal drive may be due to impaired cognitive functions induced by heat exposure [9]. However, the amplitude of SEMG activity is also influenced by peripheral factors [14]. In the present study, the motor cortical activity, excitation-contraction coupling, and neuromuscular junction during or after RCS were not measured using transcranial magnetic and peripheral nerve stimulation [15]. Therefore, there are some limits to the interpretation of the results of SEMG activity in the present study. Nevertheless, it is likely that lower RMS under HOT indicates suboptimal neural drive.

Since there was no significant difference in L_a between the two conditions, it is thought that the contribution of glycolysis to adenosine triphosphate resynthesis was similar under the two conditions, suggesting that the rate of decrease in intramuscular pH was similar in the two conditions. In addition, neuromuscular efficiency decreases in proportion to the development of peripheral fatigue [16]. The rate of decrease in neuromuscular efficiency in the present study

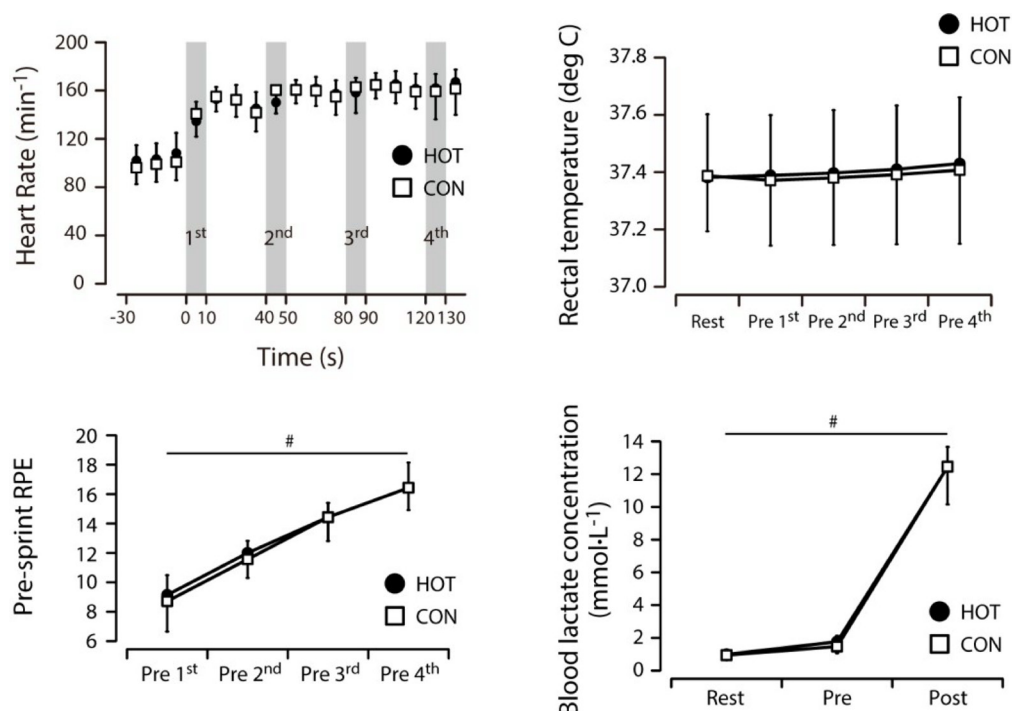


FIG. 3. Heart rate (top left) was similar between the hot-dry condition (filled circles) and the thermoneutral condition (open squares) (HOT and CON, respectively). Rectal temperature (top right) tended to increase during repeated cycling sprints (RCS) under HOT (filled circles) and CON (open squares). Rate of perceived exertion before each sprint (pre-sprint RPE; bottom left) and blood lactate concentration (bottom right) were significantly increased during RCS under HOT (filled circles) and CON (open squares).

Note: #: significant time effect ($p < 0.01$).

did not differ between the two conditions. The results for La and neuromuscular efficiency in the present study, which suggest that peripheral factors were similar in the conditions, support the idea that lower RMS under HOT indicates a suboptimal neural drive. The suboptimal neural drive during the early phase of RCS did not allow optimized sprint performance. However, this suboptimal neural drive might prevent rapid degradation of energy substrate in active muscle during the early phase of each sprint under HOT and lead to greater utilization of energy substrate during the middle phase (i.e., 5 and 6 s) of each sprint compared with CON.

Neuromuscular efficiency during the early phase of each sprint was higher in HOT than in CON. Some studies have shown that increased muscle temperature in a hot environment enhances exercise performance [17,18,19]. This enhancement has been shown to be attributed to increased neuromuscular efficiency (force divided by amplitude of SEMG) [20]. Although we did not measure muscle temperature in the present study, we cannot exclude the possibility that enhancement due to increased muscle temperature contributed to power output under HOT. Power output produced by suboptimal muscle recruitment at the onset of each sprint might be in part augmented by higher efficiency due to increased muscle temperature under HOT. Furthermore, higher power output during the middle phase of each sprint might also result from the augmentation due to increased muscle temperature. Nevertheless, the extent to which the higher neuromuscular efficiency contributed to power output remains unclear since muscle temperature was not measured in the present study. Further studies are needed to clarify the relationship between neuromuscular efficiency and power output profiles during RCS in a hot environment.

The decreases in peak power output and cardiovascular strain were not different between the two ambient conditions. The increase in T_{rec} under HOT and CON was much less than 0.1°C . In the study by Almudehki et al. [6], eight maximal 6-s sprints interspersed by 5-min recovery periods were performed in hot (40°C , 40% relative humidity) and thermoneutral (24°C , 24% relative humidity) environments. This study started repeated sprints immediately after entering an environmental chamber, as in the present study. Core temperature assessed by thermometer pill increased from 37.2 to approximately 37.5°C in the 3rd sprint (approximately 10 min after the start of repeated sprints) in the two conditions. This increase in core temperature is greater than that in the present study. This difference may be explained by a difference in relative humidity [21]. However, both studies found no differences in core temperature between the two conditions. This consistency ensures validity of no differences in core temperature between HOT and CON in the present study. Note that core temperature during exercise is evaluated by not only rectal temperature but also oesophageal temperature. It has been reported that oesophageal temperatures during exercise were similar in hot (40°C , 17% relative humidity) and thermoneutral (20°C , 23% rela-

tive humidity) conditions in the first 10 min when 40-min intermittent cycling (15-s cycling at approximately 300 W, 15-s recovery period) was performed [4]. Since the duration of the experimental protocol in the present study was approximately 10 min, it is inferred that there was no difference in oesophageal temperature between HOT and CON. Therefore, the experimental protocol used in the present study might not have been sufficient to stress the cardiovascular system, resulting in no difference in peak power output between the two conditions. This indicates that we did not clarify whether peak power output during RCS with short recovery periods is impaired due to higher cardiovascular strain in the absence of hyperthermia in a hot environment. Excess elevation in core temperature (i.e., hyperthermia) impaired performance during repeated sprints with short recovery periods (5×15 s sprints with 15-s recovery periods) [4]. In the present study, T_{rec} was not different between the two ambient conditions and did not reach hyperthermic levels. These results support the idea that hyperthermia itself is responsible for impaired peak power output during repeated sprints because the failure to reach hyperthermic temperature in a hot-dry environment resulted in no condition effect on peak power output.

Recently, some studies have suggested that an increased rate of heat storage produces higher RPE, and this increased RPE results in an anticipatory decrease in performance [22,23]. In the present study, pre-sprint RPE was similar in the two ambient conditions. Since T_{rec} was similar between the two conditions and the degree of increase in T_{rec} was much less than 0.1°C , it is possible that the rate of heat storage had a remarkable effect on RPE in the present study.

In the present study, the difference in power output at 2 s between the two conditions was approximately 30 W. This difference is small but may be important for sporting events in which both instantaneous judgments and repeated explosive movements are required.

CONCLUSIONS

Heat exposure in the absence of hyperthermia does not impair peak power output during RCS with short recovery periods. However, power output at the onset of a sprint during RCS with short recovery periods is decreased due to heat exposure. This decrease in power output may be associated with suboptimal muscle activity. Furthermore, heat exposure has a tendency to delay the time to reach maximum power output during RCS with short recovery periods.

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Conflict of interest: The authors declare that they have no conflict of interest.

REFERENCES

1. Matsuura R, Ogata H, Yunoki T, Arimitsu T, Yano T. Effect of blood lactate concentration and the level of oxygen uptake immediately before a cycling sprint on neuromuscular activation during repeated cycling sprints. *J Physiol Anthropol* 2006;25:267–273.
2. Matsuura R, Arimitsu T, Kimura T, Yunoki T, Yano T. Effect of oral administration of sodium bicarbonate on surface EMG activity during repeated cycling sprints. *Eur J Appl Physiol* 2007;101:409–417.
3. Girard O, Bishop DJ, Racinais S. Hot conditions improve power output during repeated cycling sprints without modifying neuromuscular fatigue characteristics. *Eur J Appl Physiol* 2013;113:359–369.
4. Drust B, Rasmussen P, Mohr M, Nielsen B, Nybo L. Elevations in core and muscle temperature impairs repeated sprint performance. *Acta Physiol Scand* 2005;183:181–190.
5. Bishop D, Maxwell NS. Effects of active warm up on thermoregulation and intermittent-sprint performance in hot conditions. *J Sci Med Sport* 2009;12:196–204.
6. Almudehki F, Girard O, Grantham J, Racinais S. Hot ambient conditions do not alter intermittent cycling sprint performance. *J Sci Med Sport* 2012;15:148–152.
7. Cheuvront SN, Carter R 3rd, Haymes EM, Sawka MN. No effect of moderate hypohydration or hyperthermia on anaerobic exercise performance. *Med Sci Sports Exerc* 2006;38:1093–1097.
8. Balsom PD, Seger JY, Sjödin B, Ekblom B. Maximal-intensity intermittent exercise: effect of recovery duration. *Int J Sports Med* 1992;13:528–533.
9. Gaoua N, Grantham J, El Massioui F, Girard O, Racinais S. Cognitive decrements do not follow neuromuscular alterations during passive heat exposure. *Int J Hyperthermia* 2011;27:10–19.
10. Lakomy HKA. Measurement of work and power output using friction-loaded cycle ergometers. *Ergonomics* 1986;29:509–517.
11. Racinais S, Bishop D, Denis R, Lattier G, Mendez-Villaneuva A, Perrey S. Muscle deoxygenation and neural drive to the muscle during repeated sprint cycling. *Med Sci Sports Exerc* 2007;39:268–274.
12. Amann M, Dempsey JA. Locomotor muscle fatigue modifies central motor drive in healthy humans and imposes a limitation to exercise performance. *J Physiol* 2008;586:161–173.
13. Amann M, Eldridge MW, Lovering AT, Stickland MK, Pegelow DF, Dempsey JA. Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *J Physiol* 2006;575:937–952.
14. Farina D, Merletti R, Enoka RM. The extraction of neural strategies from the surface EMG. *J Appl Physiol* 2004;96:1486–1495.
15. Girard O, Bishop DJ, Racinais S. Neuromuscular adjustments of the quadriceps muscle after repeated cycling sprints. *PLoS One* 2013;8:e61793.
16. Hautier CA, Arsac LM, Deghdegh K, Souquet J, Belli A, Lacour JR. Influence of fatigue on EMG/force ratio and cocontraction in cycling. *Med Sci Sports Exerc* 2000;32:839–843.
17. Asmussen E, Boje O. Body temperature and capacity for work. *Acta Physiol Scand* 1945;10:1–22.
18. Ball D, Burrows C, Sargeant AJ. Human output during repeated sprint cycle exercise: the influence of thermal stress. *Eur J Appl Physiol* 1999;79:360–366.
19. Sargeant AJ. Effect of muscle temperature on leg extension force and short-term power output in humans. *Eur J Appl Physiol* 1987;56:693–698.
20. Stewart D, Macaluso A, De Vito G. The effect of an active warm-up on surface EMG and muscle performance in healthy humans. *Eur J Appl Physiol* 2003;89:509–513.
21. Hayes M, Castle PC, Ross EZ, Maxwell NS. The influence of hot humid and hot dry environments on intermittent-sprint exercise performance. *Int J Sports Physiol Perform* 2014;9:387–396.
22. Tucker R, Rauch L, Harley YXR, Noakes TD. Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment. *Pflügers Arch* 2004;448:422–430.
23. Tucker R, Marle T, Lambert EV, Noakes TD. The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. *J Physiol* 2006;574:905–915.