Effect of carbohydrate ingestion and ambient temperature on muscle fatigue development in endurance-trained male cyclists

Chris R. Abbiss,1 Jeremiah J. Peiffer,1 Jonathan M. Peake,2 Kazunori Nosaka,1 Katsuhiko Suzuki,3 David T. Martin,4 and Paul B. Laursen1

1School of Exercise, Biomedical and Health Sciences, Edith Cowan University, Joondalup, Western Australia; 2School of Human Movement Studies, University of Queensland, Brisbane, Queensland, Australia; 3Consolidated Research Institute for Advanced Science and Medical Care, Waseda University, Tokyo, Japan; and 4Department of Physiology, Australian Institute of Sport, Canberra, Australian Capital Territory, Australia

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Abbiss CR, Peiffer JJ, Peake JM, Nosaka K, Suzuki K, Martin DT, Laursen PB. Effect of carbohydrate ingestion and ambient temperature on muscle fatigue development in endurance-trained male cyclists. J Appl Physiol 104: 1021–1028, 2008. First published January 24, 2008; doi:10.1152/japplphysiol.00683.2007.—The aim of the present study was to determine the effect of carbohydrate (CHO; sucrose) ingestion and environmental heat on the development of fatigue and the distribution of power output during a 16.1-km cycling time trial. Ten male cyclists (V˙O2max = 61.7 ± 5.0 ml·kg−1·min−1, mean ± SD) performed four 90-min constant-pace cycling trials at 80% of second ventilatory threshold (220 ± 12 W). Trials were conducted in temperate (18.1 ± 0.4°C) or hot (32.2 ± 0.7°C) conditions during which subjects ingested either CHO (0.96 g·kg−1·h−1) or placebo (PLA) gels. All trials were followed by a 16.1-km time trial. Before and immediately after exercise, percent muscle activation was determined using superimposed electrical stimulation. Power output, integrated electromyography (iEMG) of vastus lateralis, rectal temperature, and skin temperature were recorded throughout the trial. Percent muscle activation significantly declined during the CHO and PLA trials in hot (6.0 and 6.9%, respectively) but not temperate conditions (1.9 and 2.2%, respectively). The decline in power output during the first 6 km was significantly greater during exercise in the heat. iEMG correlated significantly with power output during the CHO trials in hot and temperate conditions (r = 0.93 and 0.73; P < 0.05) but not during either PLA trial. In conclusion, cyclists tended to self-select an aggressive pacing strategy (initial high intensity) in the heat.

power; integrated electromyography; heat; thermoregulation

HIGH AMBIENT TEMPERATURE (>27°C) (24, 30, 34, 41) and the depletion of endogenous carbohydrate stores (8, 23) significantly reduce the capacity to generate power during prolonged exercise. Under these conditions, cycling time to exhaustion at 60% of maximal oxygen uptake (V˙O2max) coincides with reaching a critical core body temperature in the range of 39.5–40.5°C (21, 33). During self-paced exercise, however, the brain may regulate power output by means of a feed-forward anticipatory mechanism that reduces the likelihood of reaching such high core body temperatures (30, 40, 41). In support of this concept, Tucker et al. (40) recently showed that reductions in power output during prolonged cycling in hot (35°C), neutral (25°C), and temperate conditions (15°C) strongly correlated (r = 0.92) with preceding increases in whole body heat content. Furthermore, they also reported that power output during the first 10 min of exercise was similar between hot (261 ± 33 W), neutral (250 ± 43 W), and temperate conditions (245 ± 35 W). Tucker et al. (40) hypothesized that the similarities in starting power output may relate to the low level of thermal stress present in the early stages of exercise. To our knowledge, no research has examined the relationship between markers of thermal stress and self-selected power output following prolonged exercise in hot and temperate conditions. Because of the influence that hyperthermia has on exercise performance, an increase in thermal stress induced during constant-pace cycling in the heat may attenuate starting power output during a subsequent self-paced bout of exercise.

Carbohydrate ingestion improves prolonged exercise performance and may do so by maintaining blood glucose levels (7), by attenuating muscle and liver glycogenolysis (8, 36), or through its influence on higher centers (i.e., the brain) (5, 9, 10). Other research has addressed the possible relationship between carbohydrate depletion and central and peripheral fatigue (1, 31). However, few studies have examined the effects of carbohydrate ingestion on markers of central fatigue and the distribution of power output during self-paced exercise. The progressive shift in substrate utilization that occurs during prolonged endurance exercise increases plasma free fatty acid concentration (12), leading to displacement of tryptophan from albumin. In turn, albumin crosses the blood-brain barrier and stimulates serotonin synthesis (9, 31). Central serotonergic and dopaminergic transmitter systems are associated with arousal, lethargy, motivation, motor control, and mood and are therefore implicated in central fatigue both at rest and during exercise (10, 11, 28). Carbohydrate ingestion may delay the increase in free fatty acid release, possibly resulting in reduced serotonin synthesis and reduced central fatigue. Furthermore, since hyperthermia is known to increase ATP utilization (17, 18), it is possible that carbohydrate supplementation may be more beneficial during exercise in hot compared with temperate conditions. However, the combined influence of both carbohydrate ingestion and hyperthermia on serotoninergic and dopaminergic activity during prolonged self-paced exercise is unknown. In addition, no studies have examined the influence of both carbohydrate ingestion and environmental heat on the self-selected distribution of work during an exercise task. The purpose of this study, therefore, was to determine the independent and additive effects of carbohydrate ingestion and envi-
Environmental heat stress on muscle fatigue development and the power output distribution during a 16.1-km cycling time trial following a prolonged period of moderate-intensity cycling (90 min at 60 ± 3% \( \text{VO}_{2\text{max}} \)).

**MATERIALS AND METHODS**

**Subjects.** Ten endurance-trained male cyclists (means ± SD: age, 27 ± 7 yr; height, 1.81 ± 0.06 m; mass, 77.9 ± 6.6 kg; sum of 7 skin folds, 66.7 ± 12.0 mm; \( \text{VO}_{2\text{max}} \), 61.7 ± 5.0 ml/kg⁻¹·min⁻¹; maximal aerobic power, 343 ± 24 W) with a minimum of 2 yr of competitive cycling experience were recruited to perform in this study. Before the study, all subjects provided written, informed consent, and the experimental procedure was approved by the Central Human Research Ethics Committee at Edith Cowan University.

Subjects were requested to maintain regular training commitments throughout the duration of the study and to refrain from any exercise on the day before each test. At the time of testing, subjects were cycling between 250 and 350 km/wk (325 ± 53 km/wk). Trials were separated by at least 4 days and commenced in the morning between the hours of 0800 and 0930 to minimize the effects of diurnal variation. Subjects consumed the same diet on the day before and the day of each trial. All tests were performed on a Velotron cycle ergometer (RacerMate; Seattle, WA), which was adjusted to the dimensions of each subject’s own bicycle and fitted with each cyclist’s own cycling pedals. All cycle tests were performed in a climate chamber (2.9 × 6.8 × 2.7 m) and were conducted between the months of October and December, where daily ambient temperatures ranged from between 12.1 ± 3.3 and 23.1 ± 3.9°C.

**Incremental cycling test.** Under controlled environmental conditions [16–18°C and 40–50% relative humidity (rh)], \( \text{VO}_{2\text{max}} \) was measured during an incremental cycling test to exhaustion. Starting at a power output of 100 W for 5 min, the workload was increased by 50 W every 5 min. During the test, subjects were allowed to cycle at their preferred pedaling rate, since the Velotron ergometer controls power output irrespective of cadence. The test was terminated either voluntarily by the cyclist or when pedal rate dropped below 60 rpm (38).

Gas exchange was measured throughout the entire test using a ParvoMedics TrueOne 2400 diagnostic system (Sandy, UT). Immediately before all tests, the gas analyzer was calibrated using alpha gases of known concentrations, and the ventilometer was calibrated using a 3-liter syringe (Hans Rudolph, Kansas, MO). The metabolic system was verified using the Laboratory Standards Assistance Scheme (22). \( \text{VO}_{2\text{max}} \) was defined as the highest \( \text{VO}_{2} \) value recorded over a 30-s average. The workload (W and W/kg) corresponding to the second ventilation threshold (VT₂) was determined using the methods of Lucia et al. (29). To become familiar with the 16.1-km trial distance following a fatiguing bout of exercise, subjects performed a 16.1-km familiarization performance time trial (described below) within 3 min of completing the \( \text{VO}_{2\text{max}} \) test.

**Experimental trials.** Subjects completed a total of four experimental trials, which were performed in a randomized crossover fashion. Two experimental trials were performed in a balanced order in both temperate (18.1 ± 0.4°C, 58 ± 8% rh) and hot conditions (32.2 ± 0.7°C, 55 ± 2% rh), during which subjects ingested either CHO or placebo (PLA) gel/solutions. Cyclists began all trials with a standard 6 g carbohydrate (CHO)/kg body wt on the day before each trial and were allowed 3.3 min of rest before starting a 16.1-km time trial. A venous blood sample was taken during the rest period (see below).

Throughout the time trials, subjects were given instantaneous feedback regarding their power output, cadence, speed, and distance cycled. Subjects were able to adjust their power output by altering their gear ratio and pedaling cadence as required. Power output and cycling speed were automatically recorded every second (Velotron Coaching Software) and averaged over each 2 km. Because of variations in starting gear ratio, the first 500 m of each time trial were removed from the analysis, because we believed that this masked any real differences between the trials. The average power output measured over each 2-km segment was compared with the mean power output recorded for each individual time trial. Cyclists were instructed to complete all time trials in the shortest possible time. Perceived exertion and thermal sensation were recorded at 4, 8, and 12 km throughout the time trials. Water consumption was ad libitum during both the 90-min cycling phase and 16.1-km time trial.

**Blood analysis.** A venous blood sample was drawn preexercise, directly following the 90-min cycling phase, and immediately following the time trial by standard venipuncture technique from the antecubital vein. During each blood draw, two separate Vacutainers were collected, using a serum separation tube and a Vacutainer containing K₂-EDTA. Serum separation tubes were left at room temperature to clot. All blood samples were centrifuged at 4°C for 10 min, with the supernatant frozen and stored at −80°C until the day of analysis. Plasma was analyzed for lactate using an enzymatic assay (Determiner LA; Kyowa Medics, Tokyo, Japan) and an automated analyzer (JCA-BM12; JEOL, Tokyo, Japan). Plasma glucose concentration was determined spectrophotometrically on an automated analyzer (Hitachi model 7170; Tokyo, Japan) using an enzymatic reaction involving hexokinase [Glu-HK (M); Shinotest, Tokyo, Japan]. Serum free fatty acid concentration was also measured on an automated analyzer (Hitachi model 7170) using an enzymatic reaction involving acyl-coenzyme A synthetase and acyl-coenzyme A oxidase (Wako NEFA HR-II; Osaka, Japan). Enzyme-linked immunosorbent assays (ELISAs) were used to measure the concentrations of serum prolactin, serum serotonin (IBL, Gunma, Japan), and plasma dopamine (Labor Diagnostika Nord, Nordhorn, Germany). ELISA measurements were performed on a microplate reader (VERSAmax; Molecular Devices, Sunnyvale, CA). The intra-assay coefficient of variation was <6% for all three assays. Data for all plasma and serum variables were corrected for changes in plasma volume according to the methods of Dill and Costill (13).

**Skin, rectal, and mean body temperature.** Skin temperature (\( T_{\text{skin}} \)) was measured using four flat-top copper skin thermists (YTS Temperature, 400 Series; Dayton, OH) attached on the chest, arm, thigh, and calf (27, 38, 41), and mean skin temperature was determined using Ramanathan’s formula (6):

\[
T_{\text{skin}} = (0.3 \times T_{\text{chest}}) + (0.3 \times T_{\text{bicep}}) + (0.2 \times T_{\text{thigh}}) + (0.2 \times T_{\text{calf}})
\]

Rectal temperature (\( T_{\text{rect}} \)) was measured using a disposable rectal thermometer (Monatherm Thermistor, 400 Series; Mallinckrodt Medical, St. Louis, MO), which was self-inserted to a depth of 12 cm from the anal sphincter. Mean body temperature (\( T_{\text{body}} \)) was determined using Burton’s formula (6):

\[
T_{\text{body}} = (0.3 \times T_{\text{chest}}) + (0.3 \times T_{\text{bicep}}) + (0.2 \times T_{\text{thigh}}) + (0.2 \times T_{\text{calf}})
\]
The heat content \(Q_{\text{content}}\) was calculated every kilometer during the time trial and was determined using the following equation (40):

\[
Q_{\text{content}} = T_{\text{body}} \times \text{body mass (kg)} \times 3.47 \text{kJ}^\circ\text{C}^{-1}\text{kg}^{-1}
\]

Heat storage \(Q_{\text{storage}}\) was determined by comparing the heat content calculated during each kilometer (i.e., \(D_2\)) with that of the preceding kilometer (i.e., \(D_1\)) and was determined using the following equation (40):

\[
Q_{\text{storage}} = Q_{\text{content}}(D_1) - Q_{\text{content}}(D_2)
\]

Skin, rectal, and environmental temperatures were recorded every second (SquirrelView 2020; Grant, Shephreth, UK) and averaged over each kilometer of the time trials. For all trials, a fan providing a wind speed of \(\sim 32\) km/h was placed directly in front (\(\sim 1\) m) of the cyclist and used during the warm-up, 90-min cycling phase, and time trial.

**Electromyography and electrical stimulation.** Muscle activation of vastus lateralis was assessed via surface electromyography (EMG) at the end of each kilometer of the time trial. Two silver-silver chloride surface electrodes were positioned as suggested by the European Recommendations for Surface EMG (25) before subjects began exercise. Before electrode placement, the site was shaved and cleaned with 70% isopropyl alcohol. EMG was recorded for 10 s (1,000 Hz) using a MegaWin muscle tester (ME3000P8; Mega Electronics, Kuopio, Finland). With the use of customized software written in LabVIEW (version 6.1; National Instruments, Austin, TX), raw EMG data were demeaned, full-wave rectified, and smoothed using a low-pass fourth-order Butterworth filter (cut-off frequency of 5 Hz) to produce a linear envelope (or integrated EMG, iEMG) (41). To reduce within-subject variability, we generated an ensemble average from five crank revolutions, which were time-normalized using a cubic spline (0–1,000 points). EMG data were amplitude-normalized using maximum voluntary isometric contractions as described below.

Before the 90-min constant-pace cycling phase and immediately following the 16.1-km time trial (within 60 s), subjects performed four 5-s maximum voluntary isometric contractions while seated on a Biodynamic isokinetic dynamometer (System 3 Pro; Shirley, NY). All contractions were performed with the quadriceps femoris of the right leg with trunk-thigh angle set at 85°. Isolation of the leg extensors was achieved via two crossover shoulder harnesses and belt straps across both the abdomen and thigh of the exercising leg. The knee flexion-extension axis was aligned with the dynamometer axis. Knee extension strength was measured at 60° of flexion. The first two of the four contractions were performed to obtain a maximum voluntary contraction for EMG normalization purposes. The EMG value corresponding with the maximum voluntary isometric contraction was determined as the greatest value recorded for an averaged 200-ms window of the linear envelope during either of the first two contractions.

During the two last maximal voluntary contractions, a train of stimulation of 1-s duration was superimposed over the isometric plateau of the maximal voluntary torque to distinguish the contributions of central and peripheral fatigue (27, 34). Square wave pulses with uniform characteristics (frequency, 100 Hz; width, 0.3 ms) were delivered to the quadriceps femoris using a Vital Stim EMS-4000 stimulator (Vitalityweb.com, San Diego, CA) and two electrodes (5 × 9 cm; Axelgaard, Fallbrook, CA). The cathode was carefully placed posterior to the greater trochanter, and the anode was positioned superior to the patella. The stimulation intensity for each subject was set by progressively increasing the stimulus intensity 25% beyond the intensity that elicited maximal peak twitch torque.

Peak torque of the superimposed train of stimulation was used to calculate the percent muscle activation using the following formula (34):

\[
\text{percent muscle activation} (\%) = \frac{\text{maximal voluntary isometric torque (maximal voluntary isometric torque + superimposed train torque)}}{\times 100}\%
\]

In the present study, we defined central fatigue as the reduction in percent muscle activation (34).
Rectal temperature was higher during the CHO trial compared with the PLA trial in hot (P < 0.05) but not in temperate conditions (Fig. 5). Heat storage, thermal sensation, ratings of perceived exertion, and mean body and mean skin temperature (Table 2) did not differ significantly between CHO and PLA trials in either hot or temperate conditions.

CHO ingestion did not significantly affect the concentrations of plasma dopamine, serum serotonin, or serum prolactin (Fig. 6, A–C). However, CHO ingestion significantly depressed the rise in serum free fatty acid concentration seen in the PLA trials (P < 0.05; Fig. 6D). Plasma glucose concentration was significantly higher in the CHO trial compared with the PLA trial at the end of the 90-min constant-pace cycling phase in both the hot and temperate conditions. It was also significantly higher after the CHO time trial completed in hot but not temperate conditions (Fig. 6E). Plasma lactate concentration was significantly higher following the CHO time trial compared with the PLA time trial performed in hot but not temperate conditions (P < 0.05; Fig. 6F).

**DISCUSSION**

The aims of this study were to examine the effects of environmental heat stress and carbohydrate consumption on the distribution of power output during a self-paced cycling time trial performed immediately following prolonged moderate-intensity cycling. Furthermore, by monitoring blood hormones, iEMG, and percent muscle activation, we have attempted to evaluate the influence of environmental heat and carbohydrate consumption on the development of muscle fatigue and task failure during self-paced exercise in endurance-trained male cyclists. The important findings of this study are as follows: 1) carbohydrate ingestion attenuated the rise in serum free fatty acid concentration but did not influence percent muscle activation following exercise; 2) carbohydrate ingestion improved time trial performance in hot but not temperate conditions; 3) exercise in high ambient temperature resulted in an increase in serum prolactin concentration and a reduction in percent activation of the quadriceps; and 4) performance in a high ambient temperature was associated with a relatively high power output at the beginning of the time trial, whereas carbohydrate ingestion resulted in a greater increase in power output during the final 2 km of the time trial.

The relative contribution that peripheral and central factors make toward the development of exercise-induced fatigue has been a focus of attention for many years (3, 34). Despite this, few studies have yet to manipulate exercise conditions to understand the task dependency nature of fatigue and thus determine the dominant mechanisms responsible for the decrease in power output and performance time during 16.1-km time trials.

### Table 1. Power output and performance time during 16.1-km time trials

<table>
<thead>
<tr>
<th>Power, W</th>
<th>Time, min</th>
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<tbody>
<tr>
<td><strong>CHO</strong></td>
<td><strong>PLA</strong></td>
</tr>
<tr>
<td>Hot</td>
<td>262±40*</td>
</tr>
<tr>
<td>Temperate</td>
<td>298±38</td>
</tr>
</tbody>
</table>

Values are means ± SD of average power output and performance time during 16.1-km time trials in hot (32°C) and temperate (18°C) conditions in carbohydrate (CHO) and placebo (PLA) trials. †P < 0.05 vs. CHO trial. *P < 0.05 vs. temperate trial.
cline in performance witnessed during self-paced exercise. Indeed, fatigue has been suggested to be a multidimensional aspect, and the specific mechanisms responsible for performance reductions appear to be task dependent (1, 2). Previous hypotheses suggest that the ingestion of carbohydrate during exercise may improve exercise performance by preventing an increase in serotonin synthesis, which contributes to central fatigue (9, 31). In the present study, however, carbohydrate ingestion did not influence percent muscle activation of the quadriceps or serum serotonin concentration, despite a significant reduction in serum free fatty acid concentration. Similarities in percent muscle activation of the quadriceps between the placebo and carbohydrate trials in this study indicate that the reductions in isometric torque observed following exercise were not simply the result of a reduction in central drive. Furthermore, results of the present study also provide evidence to suggest that when subjects consumed a placebo, reductions in time trial performance (i.e., task failure) were not solely dictated by muscle activation. iEMG of vastus lateralis correlated significantly with, and paralleled changes in, power output during both carbohydrate trials, but not during placebo trials (Figs. 3 and 4). More specifically, iEMG remained constant throughout the entire placebo trial in the heat despite a gradual decline in power output (Fig. 2A). It is important to note, however, that many factors may influence the relationship between muscle force and iEMG (16, 32). Furthermore, the lack of correlation observed between power output and iEMG during the placebo trials may simply be due to the lower variation in power output observed in these trials (Fig. 3A). Indeed, carbohydrate ingestion increased power output during the final 2 km of the time trial, whereas placebo ingestion did not (Fig. 3A). The significant increase in sprint power output observed in the latter stages of prolonged carbohydrate-supplemented exercise may be due to an attenuation of muscular fatigue associated with greater blood glucose availability (7) or, possibly, an inhibition of metabolite-mediated stimulation of group III and IV afferents (37). Carbohydrate ingestion in the present study significantly increased plasma glucose concentration and depressed the rise in serum free fatty acid concentration. Increases in power output toward the end of a time trial, despite significantly high core body temperatures (Fig. 5), may result from a voluntary increase in central neural drive (5, 41). In support of this, iEMG and power output both increased markedly during the final 2 km of both carbohydrate trials in the present study (Fig. 3).

One of the novel findings from the present study was that carbohydrate consumption improved exercise performance to a greater extent in hot compared with temperate environmental conditions. The postponement of fatigue in response to carbohydrate ingestion during prolonged exercise (14, 39) would suggest that power output and performance declines in response to placebo ingestion during exercise. However, in the present study, we only noted this effect during exercise in the heat. Elevated environmental (18) and muscle temperature (17, 18) increase muscle glycogenolysis, anaerobic glycolysis, and creatine phosphate hydrolysis during exercise. Consequently, the augmented glycogenolytic ATP resynthesis required during exercise in hot conditions likely promoted greater intramuscular glycogen depletion in the subjects of the present study (19). This effect may explain why carbohydrate consumption in the present study enhanced performance times in hot but not temperate conditions. Interestingly, although carbohydrate ingestion was found to improve time trial performance during exercise in the heat, maximal voluntary isometric torque de-

**Table 2. Skin temperature, body temperature, thermal sensation, and rating of perceived exertion at end of 90-min constant-pace cycling phase and during the 16.1-km time trial**

<table>
<thead>
<tr>
<th></th>
<th>End of 90-min Constant-Pace Cycling Phase</th>
<th>16.1-km Time Trial</th>
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<tbody>
<tr>
<td></td>
<td>iPLA</td>
<td>tCHO</td>
</tr>
<tr>
<td>Mean skin temperature</td>
<td>19.5±0.6</td>
<td>19.8±0.8</td>
</tr>
<tr>
<td>Mean body temperature</td>
<td>31.6±0.2</td>
<td>31.6±0.3</td>
</tr>
<tr>
<td>Thermal sensation</td>
<td>4±0.5</td>
<td>4±1</td>
</tr>
<tr>
<td>Perceived exertion</td>
<td>14±1</td>
<td>13±2</td>
</tr>
</tbody>
</table>

Values are means ± SD of mean skin temperature, mean body temperature, thermal sensation, and rating of perceived exertion at completion of the 90-min constant-pace cycling phase and averaged during the 16.1-km time trial in hot (h; 32°C) and temperate (t; 18°C) CHO and PLA trials. *P < 0.05 v.s temperate trial.
termined directly following exercise was similar among all trials (Fig. 1A). Such results highlight the task-specific nature of fatigue and indicate that the specific mechanisms responsible for muscle fatigue (i.e., a reduction in voluntary isometric torque) do not necessarily dictate task failure (i.e., time trial performance) (2). The greater power output observed during the carbohydrate trial in the heat also may have contributed to the higher rectal temperature observed in this trial (Fig. 5). When subjects consumed the placebo, rectal temperature was not significantly different from that in either of the temperate trials (Fig. 5). These findings indicate that performance during the placebo trial in the heat may be limited by carbohydrate availability more so than increases in core body temperature.

Exercise in the heat increased serum prolactin concentration (Fig. 6C) and reduced percentage of maximal muscle activation of the quadriceps (Fig. 1B). From these results it may be suggested that exercise in high ambient temperatures promoted central fatigue. Such an increase in markers of central fatigue during exercise in hyperthermic conditions is not a novel finding (34). Self-selected exercise intensity may be centrally regulated in an anticipatory feed-forward manner necessary to control the rate of heat storage and avoid the development of “catastrophic” hyperthermia (30, 40, 41). In the present study, power output during the time trial decreased at a greater rate during the first 6 km in the heat compared with that during exercise in temperate conditions (Fig. 2), after which heat storage was not significantly different between trials. The initial negative heat storage observed in this study is most likely associated with the rapid reduction in skin temperature resulting from the commencement of the cooling fan. However, the constant rate of heat storage that occurred beyond 6 km is consistent with the findings of Tucker et al. (40), who found that reductions in power output correlated strongly \((r = 0.92)\) with preceding (10-min period) increases in heat storage. The greater decline in power output that occurred during exercise in the heat may have been related to the fact that power output during the first 2 km of the time trial was not significantly different between hot and temperate conditions (Fig. 2). This occurred despite greater thermal sensation, perceived exertion, serum prolactin, and core, skin, and mean body temperatures before commencement of the time trials in the heat. We suggest that the similarities in initial power output shown between the trials (Fig. 2A), irrespective of differences in thermal stress, may have been due to an overriding conscious effort. Supporting this, ratings of perceived exertion were not significantly different between hot and tem-
perate conditions throughout the time trials. Such high initial self-selected power output in the heat should be of interest to coaches and athletes, since performance might improve by adopting a lower initial power output and a more evenly distributed pace (15, 20, 35). We did not examine the influence of specific pacing strategies on overall time trial performance. As such, we cannot draw conclusions as to whether the pacing strategies chosen by the cyclists in the present study were optimal or not. The present study has nevertheless shown that environmental heat and the consumption of carbohydrates during exercise influenced the pacing strategy chosen by endurance-trained male cyclists. Further research is needed to determine whether ambient temperature or carbohydrate ingestion interferes with the ability of endurance athletes to judge appropriate pacing strategies.

In conclusion, the results of the present study indicate that both carbohydrate ingestion and environmental temperature influenced the development of muscle fatigue during exercise. Such alterations impacted on the regulation of pace during high-intensity self-paced cycling following prolonged moderate-intensity exercise. Carbohydrate ingestion did not affect percent muscle activation, improved final sprint performance, and was more beneficial to performance in hot compared with temperate conditions. In contrast, power output was high at the beginning of the time trial in hot conditions and decreased rapidly thereafter for the first 6 km. Central fatigue was likely a factor contributing to this decline. Because performance during prolonged exercise may be improved when power output is distributed more evenly, the practical implication of these combined findings is that increases in environmental heat stress as well as the consumption of carbohydrate during exercise may interfere with the ability of endurance athletes to judge appropriate pacing strategies during competition.

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