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Water and carbohydrate ingestion during prolonged exercise increase maximal neuromuscular power

RICARDO G. FRITZSCHE, THOMAS W. SWITZER, BRADLEY J. HODGKINSON, SUK-HO LEE, JAMES C. MARTIN, AND EDWARD F. COYLE

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Fritzsche, Ricardo G., Thomas W. Switzer, Bradley J. Hodgkinson, Suk-Ho Lee, James C. Martin, and Edward F. Coyle. Water and carbohydrate ingestion during prolonged exercise increase maximal neuromuscular power (Pmax). J. Appl. Physiol. 88: 730–737, 2000.—This study investigated the individual and combined effects of water and carbohydrate ingestion during prolonged cycling on maximal neuromuscular power (Pmax), thermoregulation, cardiovascular function, and metabolism. Eight endurance-trained cyclists exercised for 122 min at 62% maximal oxygen uptake in a 35°C environment (50% relative humidity, 2 m/s fan speed). Pmax was measured in triplicate during 6-min periods beginning at 26, 56, 86, and 116 min. On four different occasions, immediately before and during exercise, subjects ingested 1) 3.28 ± 0.21 liters of water with no carbohydrate (W); 2) 3.39 ± 0.23 liters of a solution containing 204 ± 14 g of carbohydrate (W+C); 3) 204 ± 14 g of carbohydrate in only 0.49 ± 0.03 liter of solution (C); and 4) 0.37 ± 0.02 liter of water with no carbohydrate (placebo; Pl). These treatments were randomized, disguised, and presented double blind. At 26 min of exercise, Pmax was similar in all trials. From 26 to 116 min, Pmax declined 15.2 ± 3.3 and 14.5 ± 2.1% during C and Pl, respectively; 10.4 ± 1.9% during W (W > C, W > Pl; P < 0.05); and 7.4 ± 2.2% during W+C (W+C > W, W+C > C, and W+C > Pl; P < 0.05). As an interesting secondary findings, we also observed that carbohydrate ingestion increased heat production, final core temperature, and whole body sweating rate. We conclude that, during prolonged moderate-intensity exercise in a warm environment, ingestion of W attenuates the decline in Pmax. Furthermore, ingestion of W+C attenuates the decline in maximal power more than does W alone, and ingestion of C alone does not attenuate the decline in Pmax compared with Pl.

Carbohydrate ingestion during prolonged exercise also improves endurance performance, sometimes by preventing hypoglycemia (8, 10, 11) and sometimes by other factors that are still unclear (3, 22, 23, 34). The potential benefits of water and/or carbohydrate ingestion on maximal anaerobic power performance (4-s sprints), performed throughout prolonged exercise to simulate sport, has not been systematically investigated. However, Montain et al. (33) have recently observed that moderate hypohydration reduces muscle endurance during intense exercise lasting ~4 min, although the mechanism for this effect is unclear.

Therefore, the main purposes of this study were to determine, after 110 min of exercise, the effects of water and carbohydrate ingestion on maximal power performance (Pmax). As a secondary purpose, this study also described the separate and combined effects of water and carbohydrate ingestion during prolonged exercise lasting 120 min on thermoregulatory, cardiovascular, and metabolic functions.

METHODS

Subjects. Eight male endurance-trained cyclists gave written informed consent to participate in this study, which was approved by the Institutional Review Board of the University of Texas at Austin. The mean maximal oxygen uptake (V\textsubscript{O\textsubscript{2max}}) stature, body mass, and age of the subjects were 63.5 ± 1.0 (SE) m\textsuperscript{2} kg\textsuperscript{-1} min\textsuperscript{-1}, 1.85 ± 0.01 m, 74.5 ± 3.0 kg, and 22.1 ± 1.0 yr, respectively.

Preexperimental sessions. The preexperimental and experimental sessions were conducted during the summer and early fall in Austin, Texas. V\textsubscript{O\textsubscript{2max}} was determined by using a continuous incremental protocol on a cycle ergometer. Subjects were required to meet criteria of 1) a plateau in oxygen uptake (V\textsubscript{O\textsubscript{2}}) despite an increase in work rate and 2) respiratory exchange ratio higher than 1.1. The subjects were familiarized with the experimental measures during three heat-acclimation sessions and also during two P\textsubscript{max} control sessions. Heat acclimation was confirmed by a difference of no more than 0.2°C in the final rectal temperature between the second and third acclimation sessions. All but one subject demonstrated heat acclimation in three sessions. One subject became ill after the second acclimation (influenza), restarted the acclimation process after recovering and underwent two wk of training before successfully reaching the acclimation criteria. The heat-acclimation sessions reproduced the experimental protocol described in Experimental protocol and were separated by 48–96 h. The cyclists performed two P\textsubscript{max} control sessions on separate days other than the acclimation days, after the second and third heat-acclimation bouts, respectively, by using a protocol previously described (26) with the goal of establishing a P\textsubscript{max} control value in ideal conditions.
(i.e., without the stress of the experimental protocol). A total of 72 sprints (i.e., \( P_{\text{max}} \) tests) were completed during the familiarization sessions.

Experimental protocol. On the day before the experimental trials, the subjects were instructed not to exercise, to drink liberally, and to consume a standard diet with a carbohydrate content of at least 6 g/kg body mass (e.g., 450 g for a 75-kg person). On the morning of the experimental trials, subjects reported to the laboratory after an overnight fast. After an instrumentation period of ~45 min, subjects cycled for 122 min at a work rate previously demonstrated to elicit 62% \( V\dot{O}_2_{\text{max}} \) during the first 20 min of exercise.

\( P_{\text{max}} \) was measured during 6-min periods (3 sprints per period) starting at 26, 56, 86, and 116 min. Within a set, sprints were performed every 2 min (i.e., at 26, 28, and 30 min during the first set). Ten seconds before the sprints, the subjects stopped pedaling, positioned their dominant foot at a 30° crank angle (above the horizontal plane), gripped the drop (lower) portion of the handlebars, and anchored their body by using their arms. Then, on a verbal command, they accelerated maximally for \(~4 \text{s}\). Standardized instructions and encouragement were given by an experienced investigator blinded to the experimental treatment. After the sprints, and for a little less than 2 min, subjects cycled at a work rate that, when combined with the sprints, elicited an average of ~62% \( V\dot{O}_2_{\text{max}} \) during the 6-min sprint periods (\( V\dot{O}_2 \) during the 6-min sprint periods was measured during the acclimation session only). During the experimental protocol, dry and wet bulb temperatures and front fan speed were controlled at 34.7 ± 0.2°C, 24.5 ± 0.3°C (relative humidity: 43.3%), and ~2 m/s, respectively.

Experimental design. On 4 different days, separated by 48–72 h, the subjects received the following treatments: 1) water and carbohydrate (\( W + C \)), a large volume (3.39 ± 0.23 liters) of a flavored 6% carbohydrate solution with a formulation equal to a commercially available sports drink (Gatorade; 4% sucrose + 2% glucose); 2) water only (\( W \)), a large volume (3.28 ± 0.21 liters) of flavored, sweetened water with no carbohydrate (water volume equal to that in \( W + C \)); 3) carbohydrate only (\( C \)), a small volume (0.49 ± 0.03 liter) of a flavored, 42% carbohydrate solution (all maltodextrins, with a colorant equal to that in \( W + C \)); 4) placebo (\( Pl \)), a small volume (0.37 ± 0.02 liter) of flavored water with no carbohydrate, along with placebo gelatin capsules filled with electrolytes (water volume equal to that in \( C \)). The fluid volume ingested during \( W + C \) was determined to be the fluid volume that was ingested without discomfort on the last acclimation session. Until the time of ingestion, all treatments were kept in a water bath regulated to match core temperature. The fluid volumes ingested during \( W, C \), and \( Pl \) were derived from \( W + C \) and the electrolyte contents in \( W, C \) and \( Pl \) were equal to the one in \( W + C \). These four treatments were randomly counterbalanced, presented to the subjects as equally beneficial ergogenic aids, and administered in a double-blind fashion. Fluid treatments (\( W + C \) and \( W \)) were presented as “regular” Gatorade and a new formulation of Gatorade, respectively. \( C \) was presented as a new carbohydrate supplement that would be marketed in a gel form, and \( Pl \) was presented as capsules of sodium citrate, an alleged ergogenic aid.

Experimental procedures. On arrival at the laboratory, the subjects dressed with cycling shoes and shorts, inserted their thermistor, voided their bladder, and recorded their nude body mass on a platform scale (FW 150 KAI, Acme). After they lay down, a heart rate monitor (Polar Vantage) was placed on the chest, a Teflon catheter was inserted into an antecubital vein for blood sampling, and electrocardiograph electrodes (used by the blood pressure monitor) were placed on the chest. Immediately after, they walked into the environmental chamber where they mounted the cycle ergometer and sat quietly while the skin temperature probes, the laser-Doppler probe, and the arm blood pressure cuff were placed in position. After the subjects sat for 15 min, a blood sample was withdrawn and temperature was recorded. Then, they ingested one-third of their treatment volume and started the 122-min exercise bout.

Drinking schedule. During \( W \) and \( W + C \), the cyclists ingested one-third of the total volume just before exercise to maximize gastric emptying (36) and thus fluid intake. During exercise, they ingested the remainder of the volume in four equal doses (i.e., \( \frac{1}{3} \) of the total volume) at 8, 31, 61, and 91 min. The same timing and proportions were used for \( C \) and \( Pl \). Drinks were ingested at body temperature (i.e., ~38–39°C).

Cycle ergometer, power measures, and perceived exertion. Exercise was performed on a Monark 818 mechanical cycle ergometer modified in sit position, handlebars, crank set, and pedals to simulate a track bicycle (see Ref. 26 for further details). The endurance work rate was calculated from continuous recordings of belt tension (analog potentiometer recording pendulum position) and pedaling velocity (Cateye Micro). \( P_{\text{max}} \) was measured during a short (~4-s) cycling sprint as previously described (26). This method has a coefficient of variation of ~3% (26). Briefly, subjects started from rest and accelerated maximally for ~4 s. Data were recorded for ~6.5 pedal revolutions, and the increase in kinetic energy resulting from the acceleration of the ergometer flywheel was averaged over every pedal revolution and used to calculate power. The highest power recorded during a pedal revolution was defined as \( P_{\text{max}} \). Perceived exertion (4) was recorded before and after each set of power measurements.

\( V\dot{O}_2 \) and heart rate. During the periods of continuous exercise, \( V\dot{O}_2 \) and \( CO_2 \) production were measured for 4 min beginning at 4, 35, 65, and 95 min, by using open-circuit spirometry. Briefly, subjects breathed through a one-way Daniel’s valve connected to a dry-gas volume meter and to a mixing chamber. Expired air was continuously sampled from the mixing chamber and analyzed for \( O_2 \) (model 5-SAI, Ametek) and \( CO_2 \) (model CD-3A, Ametek) concentrations. Both analyzers and the dry-gas meter were interfaced to a laboratory computer. Heart rate was recorded by using a Polar Vantage XL Heartwatch.

Body temperatures and cutaneous blood flow. Rectal temperature was recorded at rest and every 30 min during exercise by using a thermistor (YSI 401) inserted 12 cm past the anal sphincter. Mean skin temperature was calculated at rest and every 30 min during exercise, by using weighted-average recordings (20) from six skin thermostors (YSI 409A). Cutaneous blood flow was measured continuously by using a laser-Doppler flowmeter (AlF 1) (24). The skin probe was taped to the ventral side of the right forearm. Because the effect of movement on recording was observed to be minimal, laser-Doppler recordings were not filtered. Cutaneous blood flow was reported as a percentage of the value recorded during the minute before the exercise onset.

Body mass and sweat loss. Nude body mass before and after the experimental trials was recorded by using a platform scale (FW 150 KAI, Acme). Whole body sweat loss was calculated from the difference in nude body mass before and after exercise, corrected for fluid intake, urine production, and water and carbon respiratory losses, and normalized by surface area (Dubois formula).

Blood analysis. Blood samples (94 ml/trial) were withdrawn while cyclists were sitting on the ergometer immedi-
ate before and at 8, 19, 31, 49, 79, 111, and 121 min during exercise. Part of the blood samples was separated into prechilled plastic tubes containing an EDTA solution (glucose, free fatty acids, glycerol, and lactate), an EDTA-aprotinin solution (insulin), and an EGTA-reduced-glutathione-sodium-heparin solution (catecholamines). The plasma fraction of these samples was stored at −70°C for later analysis. The other part of the blood samples was used for serum samples (osmolality, sodium, chloride, and potassium), hemoglobin (cyanmethemoglobin method), and hematocrit (microhematocrit). Blood and plasma volume changes were calculated from hemoglobin and hematocrit (13).

Statistics. Data were analyzed for two (fluid and carbohydrate) or three (fluid, carbohydrate, and time) main effects by using repeated-measures multiple ANOVA (MANOVA). When a fluid effect but no significant carbohydrate or carbohydrate by fluid interaction effects were observed, W+C and W trials were pooled into a large-fluid-volume treatment (Large F), and C and Pl were pooled into a small-fluid-volume treatment (Small F). Similarly, when MANOVA indicated a carbohydrate effect but no fluid or carbohydrate by fluid interaction effects, W+C and C trials were pooled into a large-carbohydrate-ingestion treatment (Large CHO), and W and Pl trials were pooled into a no-carbohydrate-ingestion treatment (No CHO). After a significant F test, the significance of pairwise comparisons was determined by using the Newman-Keuls post hoc test. The following pairwise comparisons were tested: W+C vs. W, W+C vs. PI, C vs. PI, and W vs. PI. Because Pmax was the main variable, for Pmax only, W+C vs. PI was also tested. The significance level was set at P < 0.05.

RESULTS

Pmax. Pmax for the second control session and during the experimental trial is presented in Fig. 1. The mean coefficient of variation for Pmax during the control session (6 sprints) was 2.4%. At 26 min of exercise (i.e., during the 26- to 32-min sprint period), Pmax was similar in all treatments and also similar to the Pmax control value (i.e., ~99% of control Pmax). From 26 to 116 min, Pmax declined 14% during C and Pl, 10% during W (P < 0.05 vs. PI), and 7% during W+C (P < 0.05 vs. W, C, and Pl). The mean coefficient of variation for Pmax during exercise (3 sprints) was 2.8%.

Body mass. For this variable, treatments were pooled into Large F and Small F treatments. Initial body mass was similar in Small F and Large F (75.4 ± 2.0 vs. 75.3 ± 2.0 kg, respectively). After exercise-induced dehydration (Small F), body mass declined to 72.3 ± 2.0 kg (P < 0.05 vs. initial value). Fluid ingestion (Large F) attenuated this decline in body mass (i.e., 74.6 ± 2.1 kg; P < 0.05 vs. Small F and vs. initial value). The percent body mass lost during exercise was 4.21 ± 0.20 and 1.04 ± 0.21% for Small F and Large F, respectively.

Perceived exertion. For this variable, treatments were pooled into Large F and Small F treatments. At 19 min of exercise, perceived exertion was similar during Large F compared with Small F. From 19 to 109 min, perceived exertion increased during Large F (11.94 ± 0.23 vs. 14.31 ± 0.45 units) and even more during Small F (11.88 ± 0.20 to 16.06 ± 0.37 units; P < 0.05 vs. Large F).

V̇O2, heart rate, and blood volume (Tables 1 and 2). V̇O2 did not demonstrate any treatment effect but tended to increase over time (P = not significant). Blood volume demonstrated a fluid effect and, therefore, was pooled into Large F and Small F treatments. During the first 30 min, fluid ingestion before and during the initial minutes of exercise slightly attenuated the increase in heart rate and the decline in blood volume (Large F compared with Small F, P < 0.05). From the first to the last 0.5 h of exercise (0–30 to 90–120 min), progressive dehydration (i.e., Small F) elicited a 4% decline in blood volume and a 16% increase in heart rate (all P < 0.05). During the same period of time, Large F prevented declines in blood volume and attenuated the increase in heart rate (~11%) (all P < 0.05 vs. Small F).

Body temperature regulation (Fig. 2). Initial resting rectal temperature was similar (36.8–36.9°C) for all treatments. By the end of the exercise bout (120 min), fluid and carbohydrate effects were observed. Rectal temperature was −0.7°C higher after dehydration (i.e., C vs. W+C, and Pl vs. C; both P < 0.05) and −0.3°C higher after carbohydrate ingestion (i.e., W+C vs. W, and C vs. Pl; both P < 0.05). Conversely, mean skin temperature was similar in all treatments throughout the exercise bout. When all subjects and all treatments were averaged, mean skin temperature was 34.4 ± 0.1°C at rest, 33.8 ± 0.1°C at 30 min, 33.3 ± 0.1°C at 60 min, and 33.2 ± 0.1°C at 120 min.

Glucose and insulin (Fig. 3). A large amount of carbohydrate ingested before 11 min of exercise were completed (~100 g for W+C and C) resulted in rapid and large increases in plasma glucose and insulin concentrations (see Fig. 3). Insulin concentration increased more rapidly and to a higher value when a given amount of carbohydrate was ingested in a large compared with a small volume of water (W+C vs. C, P < 0.05). Conversely, the increase in glucose concentration was larger when carbohydrate was ingested with a small volume of water (C vs. W+C, P < 0.05). From the first to the last 0.5 h of exercise, insulin concentration differences were also observed. However, plasma glucose concentrations were similar throughout all treatments.

Fig. 1. Effects of water and carbohydrate ingestion on maximal volitional power (Pmax) during prolonged exercise of moderate intensity. Values are means ± SE for 8 subjects who ingested 1) 3.39 ± 0.23 liters of fluid with 204 ± 14 g of carbohydrate (W+C); 2) 3.28 ± 0.21 liters of fluid, no carbohydrate (W); 3) 204 ± 14 g of carbohydrate, 0.49 ± 0.03 liter of fluid (C); or 4) 0.37 ± 0.02 liter of fluid, no carbohydrate, along with placebo capsules (Pl). *W+C different from W+C and Pl, P < 0.05. †W+C different from C, P < 0.05. ‡W different from Pl, P < 0.05. Values at 56, 86, and 116 min are lower than values at 26 min in all treatments, P < 0.05. Error bars in W and C are omitted for clarity.
Table 1. Effects of fluid ingestion during 2 h of prolonged exercise on oxygen uptake and heart rate

<table>
<thead>
<tr>
<th>Variable</th>
<th>4–19</th>
<th>35–49</th>
<th>65–79</th>
<th>95–109</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂, l/min</td>
<td>2.93 ± 0.05</td>
<td>2.90 ± 0.05</td>
<td>2.93 ± 0.05</td>
<td>3.02 ± 0.05</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>148 ± 2</td>
<td>151 ± 2</td>
<td>156 ± 2</td>
<td>161 ± 2</td>
</tr>
</tbody>
</table>

Values are means ± SE for 8 subjects. VO₂, oxygen uptake; HR, heart rate; Large F, large fluid volume; Small F, small fluid volume. * Small F different from Large F, P < 0.05. † Different from initial exercise value (i.e., 4–19 min), P < 0.05.

decreased when no carbohydrate was ingested (during W and PI, P < 0.05). During the same period, glucose concentration was maintained during W and slightly increased during PI (P < 0.05).

Catecholamines. For norepinephrine concentration, treatments were pooled into Large F and Small F treatments. During the first 0.5 h of exercise, mean plasma norepinephrine concentration was similar (P = not significant) in all treatments. From 19 to 109 min of exercise, norepinephrine concentration increased significantly (P < 0.05) in both treatment groups (11.97 ± 1.02 to 18.93 ± 1.39 nM for Large F vs. 12.58 ± 0.85 to 24.52 ± 2.24 nM for Small F; P < 0.05). At 109 min, norepinephrine concentration was lower (P < 0.05) for Large F compared with Small F. Somewhat unexpectedly, mean plasma epinephrine concentration did not demonstrate a significant fluid effect, and treatments were pooled into Large CHO and No CHO treatments for statistical analysis. From 19 to 109 min of exercise, epinephrine concentration increased significantly (P < 0.05) in both treatment groups (1.60 ± 0.15 to 2.35 ± 0.26 nM for Large CHO vs. 2.16 ± 0.20 to 3.52 ± 0.47 nM for No CHO; P < 0.05). Epinephrine concentration was lower for the Large CHO compared with No CHO at 19 and 109 min (P < 0.05). No increases in epinephrine concentration were detected after compared with before a set of sprints. At 19 min, epinephrine concentration values for the individual treatments were 1.61 ± 0.25, 2.02 ± 0.28, 1.59 ± 0.20, and 2.30 ± 0.29 nM for the W+C, W, C, and PI treatments, respectively (n = 8). At 109 min, epinephrine concentration values for the individual treatments were 2.20 ± 0.36, 3.12 ± 0.47, 2.50 ± 0.40, and 3.93 ± 0.82 nM for the W+C, W, C, and PI treatments, respectively (n = 8).

Carbohydrate and fat oxidation (Fig. 4). At 5 min of exercise, carbohydrate oxidation was similar in all treatments, with values of 2.7 g/min (=200 µmol·kg⁻¹·min⁻¹). During the 5- to 95-min period of exercise, a large decline in carbohydrate oxidation was observed during the No CHO treatments (i.e., 18% during W and 15% during PI). This large decline was not observed during the carbohydrate-ingestion treat-

Table 2. Effects of fluid ingestion during 2 h of prolonged exercise on plasma lactate, serum electrolytes, osmolality, hemoglobin, blood volume, and plasma volume

<table>
<thead>
<tr>
<th>Time, min</th>
<th>Rest</th>
<th>19</th>
<th>49</th>
<th>79</th>
<th>109</th>
<th>121</th>
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<tr>
<td>Plasma lactate, mM</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Large F</td>
<td>1.25 ± 0.05</td>
<td>2.25 ± 0.19</td>
<td>4.11 ± 0.24</td>
<td>2.31 ± 0.17</td>
<td>2.42 ± 0.20</td>
<td>2.69 ± 0.23</td>
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<tr>
<td>Small F</td>
<td>1.17 ± 0.04</td>
<td>2.15 ± 0.15</td>
<td>3.95 ± 0.22</td>
<td>2.39 ± 0.18</td>
<td>2.67 ± 0.20</td>
<td>2.77 ± 0.20</td>
</tr>
<tr>
<td>Na⁺, meq/l</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large F</td>
<td>143 ± 0</td>
<td>143 ± 0</td>
<td>144 ± 0</td>
<td>144 ± 0</td>
<td>144 ± 0</td>
<td>144 ± 1</td>
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<tr>
<td>Small F</td>
<td>143 ± 0</td>
<td>145 ± 0</td>
<td>146 ± 0</td>
<td>147 ± 0</td>
<td>149 ± 0</td>
<td>150 ± 1</td>
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<td>K⁺, meq/l</td>
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<tr>
<td>Large F</td>
<td>4.68 ± 0.07</td>
<td>5.19 ± 0.10</td>
<td>4.79 ± 0.10</td>
<td>5.11 ± 0.07</td>
<td>5.20 ± 0.08</td>
<td>5.23 ± 0.11</td>
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<tr>
<td>Small F</td>
<td>4.72 ± 0.10</td>
<td>5.40 ± 0.10</td>
<td>4.96 ± 0.08</td>
<td>5.34 ± 0.11</td>
<td>5.39 ± 0.07</td>
<td>5.47 ± 0.09</td>
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<tr>
<td>Cl⁻, meq/l</td>
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<tr>
<td>Large F</td>
<td>104 ± 0</td>
<td>106 ± 0</td>
<td>105 ± 0</td>
<td>105 ± 0</td>
<td>105 ± 0</td>
<td>104 ± 0</td>
</tr>
<tr>
<td>Small F</td>
<td>104 ± 0</td>
<td>106 ± 0</td>
<td>107 ± 0</td>
<td>107 ± 1</td>
<td>108 ± 1</td>
<td>110 ± 1</td>
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<tr>
<td>Osmolality, mosmol/kgH₂O</td>
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<td></td>
<td></td>
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<tr>
<td>Large F</td>
<td>281 ± 1</td>
<td>284 ± 1</td>
<td>285 ± 1</td>
<td>284 ± 1</td>
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<td>285 ± 1</td>
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<tr>
<td>Small F</td>
<td>282 ± 1</td>
<td>288 ± 1</td>
<td>291 ± 1</td>
<td>292 ± 1</td>
<td>295 ± 1</td>
<td>299 ± 1</td>
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<td>Hemoglobin, g/dl</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Large F</td>
<td>15.1 ± 0.2</td>
<td>15.5 ± 0.2</td>
<td>15.5 ± 0.2</td>
<td>15.5 ± 0.2</td>
<td>15.6 ± 0.2</td>
<td>15.7 ± 0.2</td>
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<tr>
<td>Small F</td>
<td>15.1 ± 0.2</td>
<td>15.8 ± 0.2</td>
<td>15.8 ± 0.2</td>
<td>16.0 ± 0.2</td>
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<tr>
<td>Decline in blood volume, Δ%</td>
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<td></td>
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<tr>
<td>Large F</td>
<td>2.80 ± 0.47</td>
<td>2.71 ± 0.49</td>
<td>2.97 ± 0.36</td>
<td>3.81 ± 0.36</td>
<td>4.24 ± 0.45</td>
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<td>Small F</td>
<td>4.65 ± 0.42</td>
<td>4.69 ± 0.36</td>
<td>5.98 ± 0.31</td>
<td>7.00 ± 0.54</td>
<td>8.39 ± 0.57</td>
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<tr>
<td>Decline in plasma volume, Δ%</td>
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<tr>
<td>Large F</td>
<td>4.46 ± 0.66</td>
<td>3.97 ± 0.75</td>
<td>4.36 ± 0.49</td>
<td>5.60 ± 0.49</td>
<td>6.28 ± 0.68</td>
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<tr>
<td>Small F</td>
<td>6.97 ± 0.57</td>
<td>6.93 ± 0.43</td>
<td>8.20 ± 0.42</td>
<td>9.89 ± 0.76</td>
<td>11.5 ± 0.78</td>
<td>11.3 ± 0.81</td>
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</tbody>
</table>

Values are means ± SE for 8 subjects. Δ, Change. Values at 19, 49, 79 and 109 min are before sprint periods, whereas values at 31 and 121 min are after first and fourth sprint periods, respectively. * Small F different from Large F, P < 0.05. † Values at 49, 79 or 109 min different from 19-min value, P < 0.05. ‡ Postsprint different from presprint, P < 0.05. § 19 min different from rest, P < 0.05.
ments (W+C and C). After the first measurement (i.e., 5 min), W+C elicited a slightly higher (P < 0.05) carbohydrate oxidation compared with C.

Free fatty acids and glycerol. Because of a significant carbohydrate effect, free fatty acids and glycerol data were pooled into Large CHO and No CHO treatments. When no carbohydrate was ingested (No CHO), large increases in free fatty acids and glycerol concentrations were observed from the first to the last 0.5 h of exercise. In contrast, carbohydrate ingestion (Large CHO) attenuated most of the increase in free fatty acids and glycerol concentrations during exercise. Plasma free fatty acids values were 276 ± 25 vs. 273 ± 29 µM at rest, 206 ± 21 vs. 235 ± 16 µM at 19 min, and 282 ± 30 vs. 740 ± 73 µM at 109 min for the Large CHO compared with the No CHO treatment, respectively. Plasma glycerol values were 43.07 ± 2.49 vs. 45.26 ± 3.25 µM at rest, 61.87 ± 6.66 vs. 75.98 ± 5.54 µM at 19 min, and 131.27 ± 13.22 vs. 309.27 ± 28.00 µM at 109 min for the Large CHO compared with the No CHO treatment, respectively.

Sweat loss. Both fluid and carbohydrate ingestion increased sweat loss (Large F: 1.76 ± 0.07 kg/m² vs. Small F: 1.57 ± 0.07 kg/m², P < 0.05; No CHO: 1.71 ± 0.08 kg/m² vs. No CHO: 1.62 ± 0.07 kg/m², P < 0.05).

Cutaneous blood flow (Fig. 5). Because of a significant fluid effect, cutaneous blood flow data were pooled into Large F and Small F treatments. Cutaneous blood flow increased rapidly soon after exercise started, reaching its maximal value before 10 min of exercise. Dehydration (i.e., Small F) elicited a decline in cutaneous blood flow during exercise from the 10- to 30-min period (average value) to the 100-to 120-min period (average...
value), whereas fluid ingestion (Large F) prevented this decline.

Osmolality, electrolytes, and lactate (Table 2). Because of a significant fluid effect, osmolality and electrolyte data were pooled into Large F and Small F treatments. Throughout exercise, dehydration elicited progressive increases in serum osmolality and in sodium and chloride ions, whereas fluid ingestion prevented these increases (i.e., Small F vs. Large F, \( P < 0.05 \)). Serum potassium concentration was higher during the dehydration treatments throughout exercise (i.e., Small F vs. Large F, \( P < 0.05 \)). However, serum potassium did not increase progressively over time, and it decreased immediately after the sprint periods (\( P < 0.05 \)) in all experimental trials. Plasma lactate concentration was not affected by fluid or carbohydrate ingestion. Lactate concentration increased significantly from 19 to 109 min (all treatments combined; \( P < 0.05 \)) and also after \( P_{\text{max}} \) measurements (i.e., at 31 vs. 19 min, and at 121 vs. 109 min, all treatments combined; \( P < 0.05 \)). Potassium and lactate were the only two measured blood variables affected by the sprints.

DISCUSSION

Dehydration, hyperthermia, and carbohydrate depletion cause fatigue during exercise by mechanisms that compromise neuromuscular, metabolic, cardiovascular, and thermoregulatory function (3, 7, 14, 17, 22, 23, 27, 32, 35, 37–39). However, the exact mechanisms causing fatigue are likely to be complex. We hypothesized that a reduced ability for maximal motor neuronal recruitment might occur with dehydration, hyperthermia, and/or carbohydrate depletion and thus maximal (sprint) cycling power was measured in the present experiment by using the recently reported inertial-load method that has been shown to have a low coefficient of variation (i.e., 3%; see Ref. 26). During 2 h of exercise at ~62% \( V_{\text{O}_2\text{max}} \) in the heat, the ingestion of a small volume of water with or without carbohydrate (C and Pl, respectively) elicited a 14% decline in \( P_{\text{max}} \) during the 30- to 120-min period of exercise (\( P < 0.05 \)). The ingestion of a large volume of water (i.e., W) attenuated this decline in \( P_{\text{max}} \) to 10% (\( P < 0.05 \) vs. Pl and C). The ingestion of a 6% carbohydrate solution (i.e., \( W + C \)) further attenuated the decline in \( P_{\text{max}} \) to 7% (\( P < 0.05 \) vs. \( W, \text{ Pl, and C} \)).

The beneficial effect of water ingestion on \( P_{\text{max}} \) observed in the present study and the well-known beneficial effect of water ingestion on endurance performance (3, 14, 27, 35) could be related. Physiological changes associated with dehydration, such as higher core temperature (3, 32, 35, 38, 39) and higher perceived exertion (3, 35) also occurred in the present experiment, indicating that impairments in endurance and \( P_{\text{max}} \), elicited by exercise-induced dehydration may share a common causality. Another potential explanation for the impaired \( P_{\text{max}} \) observed in the present study is the attenuation in glycogen utilization observed with fluid ingestion (21). To date, the direct mechanisms that might link exercise-induced dehydration to either impaired endurance or \( P_{\text{max}} \) are still unclear (33). A higher perceived exertion, observed with dehydration and hyperthermia (18), suggest that the decrease in \( P_{\text{max}} \) may originate in the central nervous system. Dehydration does reduce cardiac output and muscle blood flow, which are likely to impair performance during aerobic exercise (16–18). The extent to which this would reduce \( P_{\text{max}} \) is unclear.

To our knowledge, this is the first study to demonstrate that, during prolonged exercise in the heat, ingestion of a large volume of a 6% carbohydrate solution is more beneficial for \( P_{\text{max}} \) performance than is a large volume of water alone. The beneficial effect of ingesting water with carbohydrate (\( W + C \)) on \( P_{\text{max}} \) performance was associated with large increases in insulin and glucose concentrations during the first hour of exercise, with a ~33% higher carbohydrate oxidation after 90 min of exercise, and with a lower epinephrine concentration. Previous carbohydrate feeding studies (in which dehydration was not a factor) also reported that a higher carbohydrate oxidation (elicited by the feedings after 90–120 min of exercise) was associated with a better endurance performance (7, 8, 10, 11, 19, 22, 29, 34, 41, 42). Because carbohydrate feedings do not affect muscle glycogen degradation during prolonged exercise of moderate intensity (5, 10), the higher carbohydrate oxidation in the present and previous studies was, most likely, caused by a higher blood glucose uptake and oxidation (5). Therefore, at least when dehydration is not a significant factor, carbohydrate feedings could benefit endurance performance by increasing blood glucose uptake and oxidation. The mechanisms by which a higher blood glucose uptake and oxidation and other factors related to carbohydrate ingestion can improve \( P_{\text{max}} \) or endurance performance during prolonged exercise are unclear at this time. Carbohydrate ingestion during prolonged exercise has been shown to attenuate the increase in muscle IMP levels (40), which is thought to be a marker of a mismatch between ATP resynthesis and degradation. It is, therefore, possible that the benefit of carbohydrate, when added to water, for maintaining \( P_{\text{max}} \) was related to an effect of carbohydrate on maintaining maximal ATP hydrolysis. In addition to a muscular effect, the \( W + C \) treatment could have exerted a beneficial effect on the central nervous system that might have contributed to the higher \( P_{\text{max}} \).

In the present study, carbohydrate ingestion improved performance when it was combined with water ingestion (i.e., \( W + C \) vs. W) but not by itself (i.e., C vs. Pl). These findings might appear at odds with a previous study from our laboratory (3) that reported that carbohydrate ingestion by itself can improve endurance performance of ~1-h duration compared with placebo. Our previous study (3) used continuous exercise of a higher intensity (80% \( V_{\text{O}_2\text{max}} \)), a lower environmental temperature (30°C), and shorter duration (50 min) followed by an ~10-min performance bout. Because the level of dehydration was much larger in the present study, the difference in dehydration levels (and its associated physiological changes) may explain the different effect of carbohydrate alone on performance in
the present compared with the previous study (3). In the present study, exercise-induced dehydration elicited a 14% lower cardiac output (P < 0.05; data not reported), a 0.7°C higher core temperature (i.e., 39.5°C), and a 1.5-point higher perceived exertion. However, in the previous study (3), dehydration did not reduce cardiac output, and it elicited only a 0.3°C higher core temperature (i.e., 38.6°C) and only a 0.8-point higher perceived exertion. The combined results of the previous (3) and present experiments could be explained by an interactive effect of fluid and carbohydrate ingestion on performance. Carbohydrate ingestion appears to enhance performance when the effects of dehydration (and associated physiological changes) are small. However, when the effects of dehydration are large, as in the present experiment, it seems to override the carbohydrate effect.

A noticeable difference of the present compared with previous carbohydrate feeding studies during exercise (e.g., Ref. 10) is that a large amount of the carbohydrate feeding was ingested immediately before exercise (~68 g before exercise and ~34 g at 8, 31, 61, and 91 min of exercise). This feeding schedule elicited a large increase in insulin concentration during the first hour of exercise and a ~30% higher carbohydrate oxidation during the second hour of exercise in the Large CHO compared with the No CHO treatments. Conversely, when Coyle et al. (10) initiated the feeding at 20 min of exercise (~135 g at 20 min and ~27 g every 20 min thereafter), they reported no significant differences in insulin concentration or in carbohydrate oxidation during the first 2 h of exercise in the carbohydrate compared with the no-carbohydrate treatment. When the present and previous (10) observations are combined, they suggest that the timing of carbohydrate ingestion may affect carbohydrate oxidation, and thus it may also affect P<sub>max</sub> performance. As interesting secondary findings, we also observed that carbohydrate ingestion increased heat production by 2% (P < 0.05), final core temperature, and whole body sweating rate. The increased heat production was associated with hyperglycemia, hyperinsulinemia and increased carbohydrate oxidation (e.g., respiratory exchange ratio). The extent to which the increase in carbohydrate oxidation, and thus the concomitant increase in heat production, is needed in order for W+C to display improved P<sub>max</sub> compared with W is not clear at this time.

In agreement with a previous study from our laboratory (18), we observed that exercise-induced dehydration (Small F vs. Large F) elicited a much larger increase in plasma norepinephrine concentration but did not significantly affect epinephrine concentration. In contrast, we observed that carbohydrate ingestion (Large CHO vs. No CHO) blunted the increase in plasma epinephrine concentration but did not affect the increase in norepinephrine concentration. The carbohydrate-induced blunting of the epinephrine response could be related to the large increase in insulin concentration. When carbohydrate ingestion during exercise affects insulin concentration such as in the present and other (12, 15) studies, the epinephrine response appears to be blunted. However, when carbohydrate ingestion during exercise does not affect insulin concentration, the epinephrine response does not seem to be affected (6, 29).

The exercise model used in the present study resembles several game-type sports (e.g., soccer, tennis, basketball, rugby, American football) in which success is linked to sprinting ability (i.e., P<sub>max</sub>) at the end of a game as well as to aerobic endurance throughout the game. For example, the present exercise protocol is similar to soccer in average aerobic intensity (i.e., ~40 ml·kg<sup>-1</sup>·min<sup>-1</sup>; Ref. 1), and in average number of sprints performed (2). Because of the similarities in the exercise model between the present protocol and the above-mentioned sports, it is possible that ingestion of a carbohydrate solution immediately before and throughout exercise may provide an additional benefit (compared with water alone) for these sports. Among the limitations to be considered when the present model is extrapolated to game-type sports are that this model did not reproduce the complex variations in exercise intensity or the movement patterns most common in game-type sports (i.e., running and jumping).

In summary, the effects of water and carbohydrate ingestion on P<sub>max</sub> were studied in endurance-trained cyclists during 2 h of moderate exercise in a warm environment. First, ingestion of 3.28 ± 0.21 liters of water before and during exercise attenuated the decline in P<sub>max</sub> compared with a placebo. Second, the addition of carbohydrate (204 ± 14 g) to water further attenuated the decline in P<sub>max</sub>. Finally, ingestion of carbohydrate (204 ± 14 g) with little water did not attenuate the decline in P<sub>max</sub> compared with placebo.

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REFERENCES