Effect of hypohydration on gastric emptying and intestinal absorption during exercise


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Ryan, A. J., G. P. Lambert, X. Shi, R. T. Chang, R. W. Summers, and C. V. Gisolfi. Effect of hypohydration on gastric emptying and intestinal absorption during exercise. J. Appl. Physiol. 84(5): 1581–1588, 1998.—Dehydration and hyperthermia may impair gastric emptying (GE) during exercise; the effect of these alterations on intestinal water flux (WF) is unknown. Thus the purpose of this study was to determine the effect of hypohydration (−2.7% body weight) on GE and WF of a water placebo (WP) and a 6% (330 mosmol), 8% (400 mosmol), or a 9% (590 mosmol) CES the morning following hypohydration. For comparison, subjects ingested WP after a euhydration protocol. Solutions (−2.0 liters total) were ingested as a large bolus (4.6 ml/kg body wt) 5 min before exercise and as small serial feedings (2.3 ml/kg body wt) every 10 min of exercise. Average GE rates were not different among conditions (P > 0.05). Mean (±SE) values for WF were also similar (P > 0.05) for the euhydration (15.3 ± 1.7 ml·cm⁻¹·h⁻¹) and hypohydration (18.3 ± 2.6 ml·cm⁻¹·h⁻¹) experiments. During exercise after hypohydration, water absorption was greater (P < 0.05) with ingestion of WP (18.3 ± 2.6) and the 6% CES (16.5 ± 3.7) compared with the 8% CES (6.9 ± 1.5) and the 9% CES (1.8 ± 1.7). Mean values for final core temperature (38.6 ± 0.1°C), heart rate (152 ± 1 beats/min), and change in plasma volume (5.7 ± 0.7%) were similar among experimental trials. We conclude that 1) hypohydration to −3% body weight does not impair GE or fluid absorption during moderate exercise when ingesting WP, and 2) hyperosmolality (−400 mosmol) reduced WF in the proximal intestine.

Segmental perfusion; osmolality; plasma volume

Dehydration represents a threat to the health and well-being of individuals engaged in strenuous exercise. During such exercise, dehydration (loss of body water without equilibration of body fluid compartments) or hypohydration (loss of body water with equilibration of body fluid compartments) may increase the risk for heat illness by reducing skin blood flow (9) and/or sweat rate (7, 9), the two primary avenues for dissipation of excess heat generated by muscular activity. Depending on environmental conditions and the metabolic rate sustained during exercise, sweat rates can reach 0.5–1.5 l/h, resulting in water losses exceeding 2–4% of body weight (BW), blood glucose oxidation can exceed 1 g/min (4), and core temperatures can exceed 39°C (2, 11, 23, 24). The effect of hypohydration on heat storage can be large; increments in core temperature during exercise can be elevated by 0.1 to 0.4°C for each 1% decrease in BW (7, 11).

Previous investigations demonstrate that dehydration of 4% BW or hypohydration of 5% BW, when combined with elevated core temperatures (−39°C), impair gastric emptying of ingested fluids [i.e., 7% carbohydrate-electrolyte solution (CES) or water] during moderate-intensity [50–60% maximal oxygen consumption (VO₂max)] treadmill exercise performed in a cool (18°C) or warm (30–35°C) environment (22, 28). Together, these findings suggest that an excessive loss of body water not only can enhance body heat storage during exercise but also can impair an individual’s ability to replenish needed fluids and carbohydrates.

Both gastric emptying and intestinal absorption are important for supplementing endogenous carbohydrate stores and for enhancing fluid homeostasis and thermoregulation during exercise. The pattern of drinking can markedly affect gastric emptying rates of both fluids and carbohydrates (25). Numerous investigations demonstrate that, compared with drinking a single bolus, repeated ingestion of small volumes of dilute (up to 8%) CES can maintain a relatively high gastric volume, thereby increasing delivery rates of both carbohydrates (30–60 g/h) and fluids (15–20 ml/min) to the small intestine (19, 25, 26, 31). Exercise studies showing that gastric emptying is impaired by body fluid deficits (22, 28) have only studied ingestion of a single bolus and did not examine the effect of repeated ingestion. Recent findings also suggest that the act of repetitive drinking, perhaps by repeated stimulation of an oropharyngeal reflex, may attenuate the reductions in skin blood flow and sweat rate associated with thermal dehydration (20, 36). The effect of dehydration on intestinal absorption has not been examined in humans, although studies conducted in experimental animals suggest that it can stimulate intestinal Na⁺ and water absorption by mechanisms involving both the sympathetic and renin-angiotensin-aldosterone systems (16, 17).

Recently, our laboratory described a technique that simultaneously determines gastric emptying and intestinal absorption during repeated ingestion of a dilute CES (14). With the use of this novel technique, this investigation examined the effects of moderate (−3% BW) hypohydration on gastric emptying and intestinal absorption of a water placebo (WP) during prolonged cycling exercise in a cool environment. In addition, we were also interested in studying the efficacy of three different CES in terms of gastric emptying, intestinal absorption, and plasma volume (PV) changes while subjects exercised in a hypohydration state.

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METHODS

Seven healthy volunteers (5 men, 2 women) provided signed informed consent and served as subjects. Physical characteristics were as follows: age 26.7 ± 1.7 (SE) yr, height 183 ± 5 cm, mass 79.3 ± 5.6 kg, and VO$_{2\text{max}}$ 55.9 ± 3.1 ml·kg$^{-1}$·min$^{-1}$ (Table 1). VO$_{2\text{max}}$ and the workload (175 ± 17 W) corresponding to 65% VO$_{2\text{max}}$ were determined 1 wk before experiments by using a graded-exercise protocol on an electronically braked cycle ergometer (Cybex, Ronkonkoma, NY) and a Q-Plex metabolic system (Quinton Instruments, Seattle, WA) to measure expired gases and ventilation. Experiments were conducted during September through January in Iowa City, IA. All procedures were approved by our Institutional Review Board.

Experimental design. A balanced design was used in which treatment order was randomly assigned to subjects and in which each subject completed five experiments. At least 7 days separated each experiment. Subjects completed five 85-min bouts of cycle exercise at 65% VO$_{2\text{max}}$ in a cool (22 ± 2°C) environment while repeatedly ingesting either WP or a 6, 8, or 9% CES (Table 1). Subjects completed a hypohydration (Hypo) protocol on the day before experiments were conducted with either WP or one of the three CES. For comparison, subjects completed a euyhydration (Euhy) protocol on the day before ingestion of WP. Test solutions (NE~2.0 liters total), designed to have similar taste and appearance, were ingested as a large bolus (4.6 ml/kg body wt) 5 min before exercise and as a small serial feedings (2.3 ml/kg body wt) at 5 min of exercise and at every 10-min interval thereafter. In each experiment, gastric emptying and intestinal absorption were determined simultaneously, as previously described (14), by using a nasogastric tube placed in the gastric antrum and via a multilumen tube that spanned the entire duodenum and the first 25 cm of jejunum.

Experimental protocol. Hypohydration was attained, 12–16 h before experiments, by intermittent low-intensity treadmill exercise in a hot (45–50°C), humid (relative humidity 40–50%) environment without fluid replacement. On reporting to the laboratory, subjects were weighed nude, and a baseline rectal temperature ($T_{\text{re}}$, clinical thermometer inserted 5–7 cm past anal sphincter) was obtained. Subjects dressed in running gear (shorts, socks, and shoes) mounted the treadmill and then walked or ran (6–11 km/h) for 30 min while blood samples (10 ml) were drawn at 15, 30, and 45 min of exercise. The subjects then walked to the Exercise Physiology Laboratory, where the exercise experiments were conducted. A urine sample, a nude BW, and $T_{\text{re}}$ were obtained immediately after arrival. Subjects sat quietly for 20 min while ECG electrodes with leads were attached to the skin and fasting contents were aspirated from the stomach. After 20 min of rest, heart rate was determined, a 10-ml blood sample was drawn, and the subject mounted the cycle ergometer. A single bolus (4.6 ml/kg body wt) of chilled (10–15°C) test solution was presented in clear graduated flasks, and subjects were encouraged to consume this drink within 60–90 s. At exactly 5 min after consumption of the initial large bolus (364 ± 27 ml), subjects began the 85-min bout of cycling at 65% VO$_{2\text{max}}$ (175 ± 17 W). Additional small serial feedings (2.3 ml/kg body wt, 182 ± 14 ml) were given at 5 min of exercise and at every 10-min interval thereafter. Heart rates were taken every 10 min while blood samples (10 ml) were drawn at 15, 35, 55, 75, and 85 min of exercise. All exercise bouts were performed in a cool (22 ± 1°C) environment, with a wind velocity of ~2 feet/s produced by a fan placed in front of the subject. A nude BW, $T_{\text{re}}$, and a urine sample were obtained within 5 min after completion of exercise.

Gastric emptying was measured at 10-min intervals by using the double-sampling technique of George (10), as modified by Beckers et al. (3). Briefly, at each time point, a 5-ml sample of stomach contents was aspirated by using a 60-ml syringe. Phenol red (15 ml, 200 mg/l) was then administered via the nasogastric tube and mixed thoroughly with the stomach contents. Mixing was performed with a 60-ml syringe, required ~1 min to complete, and consisted of repeated (~10 times) withdrawal and instillation of 20–50 ml of stomach contents. A second 5-ml sample was collected after mixing. All gastric samples were stored at ~20°C until analysis. Finally, a second estimate of gastric emptying was calculated from total volume consumed, experimental time, and final gastric residual volume. Gastric residual volumes were obtained via aspiration within 5–10 min after completion of exercise.

Net intestinal absorption of fluid and solutes was determined by using techniques and calculations described by Cooper et al. (5) and Gisolfi et al. (12). As described by Lambert et al. (14), calculations of net water and solute flux were conducted by using mean gastric emptying values,
instead of a constant intestinal perfusion rate, and by using only samples obtained after a 35-min equilibration period. Intestinal fluid was collected from the proximal sampling site at a rate of 1 ml/min and from the distal site by syphonage. All test solutions contained 1.0 mg/ml polyethylene glycol 3350 as a nonabsorbable marker.

Analyses and calculations. Phenol red in gastric samples was determined spectrophotometrically at 560 nm after dilution and alkalinization to pH 9.2 with borate buffer (33). Polyethylene glycol in intestinal samples was measured by the turbidometric assay described by Malawer and Powell (18). Glucose, fructose, and sucrose were analyzed by using HPLC (Dionex DX-500, Dionex, Sunnyvale, CA). Briefly, samples (25 μl) were injected into a 250 × 40-mm Dionex CarboPac PA1 column and eluted with 0.2 M NaOH at a flow rate of 1 ml/min at 20°C. Detection was conducted by integrated amperometry with a gold working electrode and a silver-silver chloride reference electrode (8). Samples with maltodextrins were first hydrolyzed with 2 N trifluoroacetic acid at 100°C for 2 h, and the liberated glucose was then measured by HPLC. Osmolality was measured via freezing-point depression (Multi-Osmette, Precision Systems, Natick, MA) and Na⁺ and K⁺ via flame photometry (model IL 943, Instrumentation Laboratory, Lexington, MA). Changes in PV were calculated from changes in hemoglobin and hematocrit according to Dill and Costill (6). Sweat rate was calculated from the change in BW corrected for fluid ingestion, phenol red injection, and stomach, intestinal, and blood samples.

Data were analyzed by using either a one-factor ANOVA or a two-factor ANOVA with repeated measures. Significant differences identified by these analyses were located by using the Scheffé post hoc test. Statistical significance was set at P < 0.05. Values are given as means ± SE.

RESULTS

The Hypo protocol produced similar (P > 0.05) changes in BW loss, plasma osmolality, and urine osmolality for the four Hypo experiments (Table 2). Mean BW loss was 2.20 ± 0.16 kg, representing ~2.7% of initial BW (80.9 ± 2.6 kg). Compared with Euhy values, hypohydration did not alter plasma osmolality but did increase (P < 0.05) urine osmolality.

Subjects ingested mean volumes of 1.83 ± 0.14 liters during the five experimental trials. There was no significant difference in gastric emptying rates among the five experiments, which averaged 19.1 ± 1.7 ml/min (6% CES), 18.1 ± 1.6 ml/min (8% CES), 17.0 ± 1.7 ml/min (9% CES), 19.5 ± 2.0 ml/min (WP-Hypo), and 18.1 ± 1.7 ml/min (WP-Euhy) (Fig. 1). Hypohydration did not influence the gastric emptying rate of WP. The overall mean gastric emptying rate determined via the double-sampling technique was similar to values determined via final gastric residual volumes (18.5 ± 1.0 and 18.2 ± 0.8 ml/min, respectively). Final gastric residue volume was similar among the WP and 6 and 8% CES (mean for all = 162 ± 54 ml); however, for the 9% CES (405 ± 76 ml) the volume was significantly greater (P < 0.05) than for the more dilute solutions. This higher stomach volume maintained by the 9% CES likely allowed the stomach to empty at a comparable rate to the others after the 35-min equilibration period.

Hypohydration did not alter intestinal water absorption (Fig. 1) when WP was ingested. Net water flux values during WP ingestion were 18.3 ± 2.6 and 15.3 ± 1.8 ml·cm⁻¹·h⁻¹ for the hypohydration and euhydration conditions, respectively. During the Hypo experiments, however, intestinal water absorption was greater (P < 0.05) during ingestion of WP (18.3 ± 2.6 ml·cm⁻¹·h⁻¹) and the 6%CES (16.5 ± 3.7 ml·cm⁻¹·h⁻¹), compared with ingestion of the 8% (6.9 ± 1.5 ml·cm⁻¹·h⁻¹) and 9% (1.8 ± 1.7 ml·cm⁻¹·h⁻¹) CES. Also, hypohydration did not alter net intestinal Na⁺ or K⁺ flux; ingestion of WP resulted in similar mean flux values for the hypohydration and euhydration conditions (Fig. 2). During hypohydration, ingestion of the 6% CES resulted in net Na⁺ absorption (negative values indicate absorption), whereas ingestion of the 8% and 9% CES produced Na⁺ secretion. This is likely a reflection of a greater Na⁺ concentration entering the test segment with the 6% CES (34 ± 4 mmol/l) compared with the 8% (16 ± 2 mmol/l) and 9% (19 ± 3 mmol/l) CES (P < 0.05). Net K⁺ flux values were similar for all solutions. During hypohydration, ingestion of the three CES resulted in similar intestinal absorption rates for glucose and fructose (Fig. 3).

Table 2. Effect of hypohydration on body weight loss and plasma and urine osmolalities

<table>
<thead>
<tr>
<th>Condition</th>
<th>Body Weight Loss, %</th>
<th>Plasma Osmolality, mosmol/kgH₂O</th>
<th>Urine Osmolality, mosmol/kgH₂O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypohydration</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>6% CES</td>
<td>2.72 ± 0.15</td>
<td>289 ± 1</td>
<td>865 ± 59</td>
</tr>
<tr>
<td>8% CES</td>
<td>2.70 ± 0.16</td>
<td>290 ± 2</td>
<td>886 ± 79</td>
</tr>
<tr>
<td>9% CES</td>
<td>2.64 ± 0.17</td>
<td>288 ± 2</td>
<td>870 ± 71</td>
</tr>
<tr>
<td>WP</td>
<td>2.69 ± 0.16</td>
<td>289 ± 2</td>
<td>872 ± 68</td>
</tr>
<tr>
<td>Euhydration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WP</td>
<td>0 ± 0</td>
<td>286 ± 1</td>
<td>527 ± 92*</td>
</tr>
</tbody>
</table>

Values are means ± SE. *Significantly different (P > 0.05) from all hypohydration values.
Mean osmolality of the WP in the test segment was not altered by hypohydration but was different (P < 0.05) among the four different solutions studied (Fig. 4). Mean values during ingestion of WP (Euhy), WP (Hypo), 6% CES, 8% CES, and 9% CES were 113 ± 6, 129 ± 13, 284 ± 6, 328 ± 10, and 404 ± 9 mosmol/kgH₂O, respectively. Compared with the original solution osmolality (Table 1), test segment osmolality was increased during ingestion of WP (~115 mosmol/kgH₂O), likely the result of greater water absorption compared with solute absorption. Test segment osmolality was reduced during ingestion of the 6% CES (~43 mosmol/kgH₂O), 8% CES (~79 mosmol/kgH₂O), and 9% CES (~190 mosmol/kgH₂O), likely reflecting water secretion early in the duodenum (to bring the solutions closer to isotonicity), with subsequent fluid absorption more distally as water followed net solute absorption.

PV fell significantly (P < 0.05) within the first 15 min of exercise and remained depressed for the final 70 min (Fig. 5). Reductions in percentage change in PV were similar for all solutions and were not altered by hydration status. Similarly, changes in plasma osmolality, Na⁺, and K⁺ were similar throughout exercise, regard-
less of solution ingested or hydration status (Fig. 6). Ingestion of WP during euhydration tended to elicit smaller increases in plasma osmolality and K⁺ compared with other experimental conditions. Plasma glucose concentrations tended (P > 0.05) to increase during exercise when the three CES were ingested under hypohydration conditions (Fig. 7). In contrast, ingestion of WP during euhydration, but not during hypohydration, resulted in lowered plasma glucose concentrations during the final 30 min of exercise.

After exercise, mean values for BW loss (Table 3) were similar among the four Hypo experiments (~3% of euhydration BW) and similar to values (~2.7%) observed at the start of these experiments (Table 2). Similarly, ingestion of WP during the Euhy protocol produced little BW loss (0.02%), indicating that the drinking pattern was sufficient to offset fluid loss via sweating. Mean values for final heart rate (~152 beats/min), Tre (~38.5°C), and sweat rate (~1.13 kg/h) were also similar for the five experimental trials. Urine production was significantly greater in the WP-Euhy trial (162 ± 44 ml), compared with the hypohydration experiments that were not different from each other (mean for all = 31 ± 12 ml).

DISCUSSION

Hypohydration at levels as low as 1–2% of BW can be associated with impairments in physical work capacity, thirst perception, and cardiovascular and thermoregulatory function (32), the latter three representing important risk factors for the development of heat illnesses such as heat exhaustion and fatal heatstroke. Studies conducted by Rehrer et al. (28) and Neufer et al. (22) indicate that relatively severe body fluid deficits (4–5% BW) can impair gastric emptying during strenuous exercise. These important observations suggest that impairments in gastrointestinal function (i.e., reduced availability of needed fluids and carbohydrates) may constitute an additional mechanism by which hypohydration contributes to the development of heat illness during exercise.

By using a technique to simultaneously measure gastric emptying and intestinal absorption (14), the present investigation showed that moderate (~3% BW)

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**Table 3. Physiological responses to moderate-intensity cycling exercise during the five experimental conditions**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Final Body Weight Loss, %</th>
<th>Final Heart Rate, beats/min</th>
<th>Final Tre, °C</th>
<th>Sweat Rate, kg/h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypohydration</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6% CES</td>
<td>2.96 ± 0.18</td>
<td>150 ± 5</td>
<td>38.5 ± 0.2</td>
<td>1.15 ± 0.11</td>
</tr>
<tr>
<td>8% CES</td>
<td>2.81 ± 0.24</td>
<td>154 ± 3</td>
<td>38.5 ± 0.1</td>
<td>1.06 ± 0.10</td>
</tr>
<tr>
<td>9% CES</td>
<td>3.01 ± 0.10</td>
<td>156 ± 7</td>
<td>38.7 ± 0.2</td>
<td>1.17 ± 0.07</td>
</tr>
<tr>
<td>WP</td>
<td>2.95 ± 0.18</td>
<td>154 ± 4</td>
<td>38.6 ± 0.1</td>
<td>1.24 ± 0.14</td>
</tr>
<tr>
<td>Euhydration</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WP</td>
<td>0.02 ± 0.0*</td>
<td>148 ± 6</td>
<td>38.4 ± 0.2</td>
<td>1.05 ± 0.13</td>
</tr>
</tbody>
</table>

Values are means ± SE (n = 7 subjects). Tre, rectal temperature. *Significantly different (P < 0.05) from all hypohydration values.
hypohydration did not significantly alter gastrointestinal function when small volumes of WP were repeatedly ingested during 85 min of cycling exercise in a cool environment. The major findings were 1) gastric emptying rates of WP and dilute CES were maintained at high values (~18 ml/min) during exercise while the subjects were hypohydrated; 2) hypohydration did not alter gastric emptying or intestinal water absorption during repeated ingestion of WP; and 3) replacement of fluids at rates sufficient to offset fluid lost by sweating, can help maintain thermoregulatory (core temperature) and cardiovascular function (heart rate) during exercise performed with moderate hypohydration.

As noted, Rehrr et al. (28) and Neufert et al. (22) demonstrated that dehydration of 4% BW or hypohydration of 5% BW, respectively, can impair gastric emptying of a single large bolus (8 ml/kg body wt or 400 ml) of either a 7% CES or water during 60 or 15 min of treadmill running (60 or 50% \( \text{V}_{\text{O2max}} \)) in a cool or warm (35°C) environment. In contrast, the present investigation showed that more moderate levels of hypohydration (3% BW) did not alter the relatively high gastric emptying rates (~18 ml/min) of WP when small volumes (2.3 ml/kg body wt) of this solution were repeatedly (every 10 min) ingested during 85 min of cycling exercise (65% \( \text{V}_{\text{O2max}} \)) in a cool (22°C) environment.

There are at least two possible explanations for these divergent findings. First, the pattern of drinking, a single bolus or repeated ingestion, is well known to markedly alter gastric function and, therefore, the delivery rate of both fluids and carbohydrates to the small intestine (25). Our finding that gastric emptying rates of WP were maintained at high values during exercise hypohydration are in agreement with two previous studies (26, 31) that employed repeated ingestion of water or dilute CES during prolonged exercise in the heat. We suggest that repeated ingestion of fluids, by providing a strong stimulus to gastric emptying, may have been sufficient to overcome or mask any inhibitory effects of exercise and hypohydration on gastric function.

Second, body fluid deficits of ~4% BW may represent a threshold above which gastrointestinal dysfunction is likely to develop during strenuous exercise in mild environments (29). In other words, the physiological stresses imposed by moderate hypohydration (3% BW), when combined with cycling exercise (65% \( \text{V}_{\text{O2max}} \)) in a cool environment, may not have been sufficient to alter gastrointestinal function. The present study showed that, compared with exercise under euhydration conditions, hypohydration to ~3% of BW elicited similar increases in plasma osmolality (Fig. 6), heart rate (~152 beats/min), \( T_{re} \) (~38.6°C), and sweat rate (Table 3), and similar reductions in PV (Fig. 5). In contrast, in subjects running (50% \( \text{V}_{\text{O2max}} \)) in the heat (35°C) under severe hypohydration (5% BW), Neufert et al. (22) observed that impaired gastric emptying was associated with markedly elevated heart rate (~180 beats/min) and \( T_{re} \) (>39.0°C). Thus both the pattern of drinking and the physiological strains (magnitude of hypohydration, cardiovascular responses, and hyperthermia) associated with exercise appear to be important determinants for gastrointestinal function during exercise.

This study is the first to provide evidence that water absorption is not altered by exercise and moderate (3% BW) hypohydration (Figs. 1 and 2). The mechanisms for these responses are not known. However, there is considerable evidence, obtained in humans (21) and animals (16, 17, 35), that enhanced intestinal Na⁺ and water absorption will occur in response to dehydration and the consequent activation of the sympathetic and renin-angiotensin systems. In contrast, animal data also indicate that excessive activation of these two systems may actually inhibit intestinal absorption and promote secretion (17). These findings suggest that the intestinal response to exercise with hypohydration is likely to be influenced by the magnitude of physiological strain, and the consequent magnitudes of sympathetic and renin-angiotensin system activation (1). The marked gastrointestinal distress (abdominal cramps, diarrhea) frequently experienced by exhausted, heat-stressed, dehydrated runners (28, 29) may be partially explained by this mechanism. To conclude, the relatively moderate physiological strain associated with repetitive drinking, cycling at 65% \( \text{V}_{\text{O2max}} \) in a cool environment, and moderate (3% BW) hypohydration may explain why intestinal absorption was not altered in this study.

Solution osmolality and total solute flux are two major factors governing water absorption in the small intestine. Prior studies of resting subjects show that hyperosmotic solutions (~400 mosmol/kgH₂O) such as an 8, 10, or 17% glucose solution, will not cause intestinal secretion or net water movement into the intestinal lumen (30). On the other hand, Shi et al. (34) observed similar values for net water absorption when three different 6% CES, with osmolalities ranging from 186 to 403 mosmol/kgH₂O, were perfused into the distal duodenum and proximal jejunum (50-cm segment). The present study showed that, during exercise and hypohydration, repeated ingestion of test solutions, with osmolalities ranging from 3 to 594 mosmol/kgH₂O, also resulted in net water absorption within the duodenum and proximal jejunum (Fig. 1).

Intestinal water absorption was, however, greater during ingestion of WP and 6% CES compared with ingestion of the 8 and 9% CES. These differences in intestinal water absorption corresponded to calculated changes in original solution osmolality within the 50-cm intestinal test segment (i.e., solution osmolality — mean test segment osmolality). Solution osmolalities were increased during passage through the intestinal lumen with the repeated ingestion of water (~115 mosmol/kgH₂O) but were reduced during ingestion of 6% CES (~43 mosmol/kgH₂O), 8% CES (~79 mosmol/kgH₂O) and 9% CES (190 mosmol/kgH₂O) (Table 1, Fig. 4). Because water movement in the intestine is passive and moves down an osmotic gradient (13), these osmolality changes indicate that the 8 and 9% CES, compared with water and 6% CES, required relatively
greater water movement into the intestinal lumen. Thus differences in original solution osmolality (Table 1) provide one explanation why net intestinal water absorption was greater during ingestion of WP and 6% CES compared with the 8 and 9% CES.

Increments in intestinal water absorption are also related to increases in Na+ and carbohydrate absorption (34). In other words, water moves passively with an osmotic gradient created by intestinal solute absorption. The present study showed that repeated ingestion of the 6% CES resulted in Na+ absorption while ingestion of the 8 and 9% CES produced intestinal Na+ secretion (Fig. 2). On the other hand, repeated ingestion of the three CES resulted in similar intestinal absorption rates for glucose and fructose (Fig. 3). Our findings indicate that intestinal absorption of Na+, but not carbohydrate, may have contributed to enhanced water absorption during ingestion of the 6% CES compared with the 8 and 9% CES. It is likely that the higher Na+ entering the test segment with the 6% CES (33 ± 4 meq/l) stimulated Na+ absorption, whereas the lower Na+ in the 8% CES (16 ± 2 meq/l) and 9% CES (19 ± 3 meq/l), along with their higher osmolalities, stimulated early secretion of water and Na+. This would explain the lower net water fluxes observed for these beverages. The increased Na+ absorption for the 6% CES may have also occurred via solvent drag as water moved paracellularly with the opening of tight junctions in the presence of glucose (27). The fluid absorption rates observed during ingestion of the 6% CES (16.5 ± 3.7 ml · cm⁻¹ · h⁻¹) were similar to those reported by Lambert et al. (14), who examined intestinal water flux in subjects during either repeated ingestion (19.5 ± 2.6 ml · cm⁻¹ · h⁻¹) or direct perfusion (16.4 ± 1.9 ml · cm⁻¹ · h⁻¹) of a 6% CES into the duodenum while the subjects were cycling at 60–65% VO₂max. It should be noted that, although water absorption for the WP and 6% CES solutions was greater than for the 8 and 9% CES, this does not mean that the latter two solutions were not absorbed more distally. Accordingly, there were no differences among beverages in final BW (Table 3) or in PV change (Fig. 5). A limitation of the segmental perfusion technique is that it only determines absorption in the intestinal segment studied, which is not necessarily indicative of absorption in other segments or of the overall efficacy of a beverage for maintaining fluid homeostasis. However, segmental perfusion can indicate how rapidly fluid may be made available to the circulation when the segment studied spans the duodenum and proximal jejunum (15).

In the present study, similar reductions in PV were noted during the 85-min bouts of cycling exercise, regardless of the solution ingested or the hydration status (Fig. 5). This observation was surprising in view of the marked differences in intestinal water absorption exhibited during ingestion of water or the 6% CES compared with the 8 or 9% CES (Fig. 1); however, as noted above, the 8 and 9% CES were likely still absorbed distal to our measurement site. Rehrer et al. (30) also observed similar reductions in PV (~10–14%) during 80 min of cycling exercise (70% VO₂max · ambient temperature = 20°C), when subjects repeatedly ingested large volumes (~1.3 liters total) of either water or three different carbohydrate solutions. In their study, mean values for jejunal water absorption were greater for the 4.5% glucose solution (+10 ml · cm⁻¹ · h⁻¹) compared with water (+3 ml · cm⁻¹ · h⁻¹) and the 17% maltodextrin solution (+2 ml · cm⁻¹ · h⁻¹), whereas net jejunal water secretion was noted for the 17% glucose solution (~50 ml · cm⁻¹ · h⁻¹). Two additional studies suggest that reductions in PV during exercise may not adequately reflect fluid availability from ingested solutions. Although intestinal water absorption was not measured, Barr et al. (2) observed similar reductions in PV during the first 120 min of cycling exercise (50% VO₂max · ambient temperature = 30°C) performed under conditions of either no fluid replacement or water or saline ingestion at ~285 ml every 15 min. Similarly, Montain and Coyle (20) showed similar PV reductions during 80 min of cycling exercise (~65% VO₂max · ambient temperature = 35°C) conducted with either ingestion of 6% CES (1.5 liters in 65 min) or no fluid. Together, these findings suggest that changes in PV, observed during ~80–120 min of moderately intense cycling exercise, may not accurately reflect differences in fluid availability (intestinal water absorption) from ingested water, saline, or dilute CES. With our experimental model (12–16 h after dehydration), the body may have had adequate time to adjust to a 2.7% body fluid deficit, with physiological responses directed at minimizing changes in PV during exercise.

In summary, the present investigation provides evidence that repeated ingestion of either WP or 6–9% CES, at rates sufficient to replace the fluids lost via sweating, can result in relatively high rates of gastric emptying and intestinal water and carbohydrate absorption during prolonged moderate-intensity cycling exercise in a cool environment. Moreover, this study shows that, under the described conditions, moderate (3% BW) hypohydration does not appear to adversely affect gastrointestinal function.

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