Blood Glucose Responses to Carbohydrate Feeding Prior to Exercise in the Heat: Effects of Hypohydration and Rehydration

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This study assessed the plasma glucose (PG) and hormonal responses to carbohydrate ingestion, prior to exercise in the heat, in a hypohydrated state versus partial rehydration with intravenous solutions. On separate days, 8 subjects (21.0 ± 1.8 years; 57.3 ± 3.7 ml·kg⁻¹·min⁻¹) exercised at 50% \( \bar{VO}_{2\max} \) in a 33 °C environment until a 4% body weight loss was achieved. Following this, subjects were rehydrated (25 ml·kg⁻¹) with either: 0.45% IV saline (45IV), 0.9% IV saline (9IV), or no fluid (NF). Subjects then ingested 1 g·kg⁻¹ of carbohydrate and underwent an exercise test (treadmill walking, 50% \( \bar{VO}_{2\max} \), 36 °C) for up to 90 min. Compared to pre-exercise level (294 mg·dl⁻¹), PG increased significantly (>124 mg·dl⁻¹) at 15 min of the exercise test in all trials and remained significantly elevated for 75 min in NF, 30 min more than in the 2 rehydration trials. Although serum Insulin increased significantly at 15 min of exercise in the 45IV trial (7.2 ± 1.2 vs. 23.7 ± 4.7 μIU·ml⁻¹), no significant differences between trials were observed. Peak plasma norepinephrine was significantly higher in NF (640 ± 66 pg·ml⁻¹) compared to the 45IV and 9IV trials (472 ± 55 and 474 ± 52 pg·ml⁻¹, respectively). In conclusion, ingestion of a small solid carbohydrate load prior to exercise in the 4% hypohydration level resulted in prolonged high PG concentration compared to partial IV rehydration.

Key Words: insulin, catecholamines, intravenous-saline, dehydration

Introduction

Prolonged exercise in a hot environment is associated with large fluid losses, mainly via sweat secretion as a mechanism of heat dissipation. If fluid losses are not adequately restored during the exercise, the end result is hypohydration. This condition results in increased plasma osmolality (hyperosmolality) and a reduction in plasma volume (hypovolemia), which may lead to decreased venous return and stroke volume and cause a compensatory increase in heart rate (15). In addition to compromises in

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cardiovascular function, the reduction of plasma volume may hinder thermo-regulatory mechanisms by decreasing sweat rate (24) and skin blood flow for a given core temperature (23).

Besides fluid regulation, carbohydrate (CHO) metabolism is very important during prolonged exercise because glucose is the preferred fuel for skeletal muscle contraction. Intramuscular stores of the glucose in the form of glycogen are strongly correlated to exercise endurance (4). Furthermore, CHO ingestion during or before exercise has been demonstrated to increase exercise tolerance in a wide range of conditions (5). Therefore, it is very important for the individual who must perform for extended periods in hot environments to adequately replenish not only fluid but also carbohydrate losses.

The muscle uptake of glucose during exercise is primarily though a contraction-mediated mechanism (9). However, the presence of insulin is necessary for a maximal rate of glucose uptake to occur (7, 25); its action being synergistic with the contraction-mediated mechanism (1). Previous studies, in resting individuals, have shown that hypohydration (28) and hyperosmolality (3) can decrease the rate of insulin-mediated glucose uptake for a given insulin concentration (insulin sensitivity). However, none of the aforementioned studies evaluated the effects of hypohydration or hyperosmolality on insulin sensitivity and glucose homeostasis with concurrent exercise.

Only a few studies have evaluated some aspect of glucose homeostasis during exercise in the hypohydrated state. Lambert et al. (12) observed exercise of progressive intensity in humans at a 4% hypohydrated level and found that the respiratory exchange ratio (RER) was decreased (suggesting less CHO utilization) despite the fact that plasma glucose remained constant. Furthermore, during moderate intensity exercise, plasma glucose concentration was observed to increase in correlation with hypohydrated level (−3% vs. −6% of original body weight) (8). In addition, plasma lactate and respiratory exchange ratio (RER) decreased at the greater (−6%) hypohydrated level. On the other hand, Below et al. (2) found that CHO ingestion had a positive effect on exercise performance despite a body fluid loss of approximately 2% of body weight. The contrasting responses in those studies are probably the result of the different hypohydration levels tested.

Overall, the results of the available research generally point to decreased glucose utilization during submaximal exercise in the moderate-to-severely hypohydrated state despite its availability in plasma. Thus, the primary purpose of this study was to assess circulating glucose, insulin and catecholamine responses to carbohydrate consumed immediately prior to prolonged exercise in a hot environment in a hypohydrated state (−4% of body weight) or following rehydration with intravenous saline of two concentrations (0.45% NaCl vs. 0.9% NaCl) to −1.5% of body weight. It was hypothesized that if the −4% hypohydration level alters the metabolism of glucose, it would be reflected as higher blood glucose and Insulin responses to pre-exercise CHO feeding.

Methods

Subjects

Eight healthy males volunteered to participate in this study and completed a written statement of informed consent after a thorough explanation of the risks and benefits of their participation. Average age (±SE) was 21.0 ± 1.8 years; mass 76.0 ± 0.9 kg;
height 179.6 ± 0.2 cm; % body fat 8.2 ± 0.3% and $\dot{V}O_{2\max } = 57.3 ± 0.4$ ml · kg$^{-1}$ · min$^{-1}$. Subjects were recruited from The University of Connecticut and met the following criteria: (a) no chronic health problems; (b) no previous history of heat stroke or other heat illness; (c) no history of cardiovascular, metabolic, or respiratory disease; (d) a maximal aerobic capacity ($\dot{V}O_{2\max }$) of 55 to 65 ml O$_2$ · kg$^{-1}$ · min$^{-1}$; and (e) non heat-acclimated. The procedures for this study were approved by The University of Connecticut Institutional Review Board for Studies Involving Human Subjects.

**Pre-Testing**

In order to obtain baseline measurements of body weight, plasma osmolality, and urine specific gravity, subjects reported to the laboratory on 3 separate days prior to the beginning of testing. At the time, measurements of height and percentage of body fat by underwater weighing (26) were also taken. During one of the three visits, subjects underwent a progressive exercise test to volitional exhaustion, running on a treadmill, to determine maximal oxygen consumption rate ($\dot{V}O_{2\max }$). The $\dot{V}O_{2\max }$ test was a modification of the protocol by Costill and Fox (6). Subjects ran at 160–220 m · min$^{-1}$ for 4 min at a 0% grade. After 4 min, the grade was increased to 4% for 2 min and was then increased by 2% every 2 min until subjects reached exhaustion. Subjects were asked to keep a dietary record for the 3 days prior to each testing. They were also asked to maintain a similar diet on the days prior to each subsequent exercise trial and instructed to drink 500 ml of water the night before and on the morning of each trial. In addition, the subjects refrained from exercise for a 24-hour period and from eating for a 12-hour period prior to testing.

**Experimental Protocol**

Upon arrival at the laboratory (between 0700 and 0800 hours), subjects were asked to provide a urine sample. The specific gravity of the sample was measured with a hand-held refractometer (A300CL, Spartan, Japan) to insure that the subjects were properly hydrated. After collecting the urine sample, the subjects were fitted with a heart rate monitor (Polar Electro, Finland), a rectal thermistor (YSI, Yellow Springs, OH), and a 20-gauge indwelling cannula (for blood withdrawal) placed in an antecubital vein maintained patent with a heparin lock (Hep-Lock, Abbott, Chicago, IL). Following these procedures, the subjects proceeded to an environmental chamber (33 °C, Model 2000, Minus Eleven, Malden, MA) and stood for 20 min to permit body fluid and temperature equilibration. At the end of this period, a baseline blood sample was drawn, and the subjects consumed a standard breakfast consisting of one banana, a bagel, and 240–350 ml of fruit juice.

For each of the four experimental trials, subjects performed exercise for 2 to 4 hours to dehydrate to -4% of initial body weight. Exercise during each of the first 3 hours consisted of 25 min of cycle ergometry (Monark, Sweden) followed by 25 min of treadmill walking (5 to 10% grade), with a 5-min rest period after each exercise bout. If the desired 4% body weight loss was not achieved during the first 3 hours, the exercise during the 4th hour consisted of two 25-min bouts of treadmill walking. A fan was placed in front of the treadmill and cycle ergometer; it provided air flow at a speed of 2.3 m · s$^{-1}$. The intensity of both cycling and walking exercise was approximately 50% of $\dot{V}O_{2\max }$. Body weight measurements were taken at the conclusion of every 25-min period of exercise. Rectal temperature and heart rate were
constantly monitored throughout the exercise-induced dehydration protocol. The established criteria for cessation of the dehydration protocol, and removal from the environmental chamber were: (a) a rectal temperature of 39.5 °C, (b) a heart rate above 180 beats per minute for 5 min, or (c) signs or symptoms of heat exhaustion (i.e., dizziness, nausea, extreme fatigue).

At the end of the dehydration phase, a post-exercise blood sample was taken. The subjects then exited the environmental chamber and, after a 20-min rest in the moderate environment (22–25 °C), underwent rehydration by one of the following methods: (a) intravenous 0.45% saline solution (4IV; osmolality = 154 mOsm · kg⁻¹), (b) intravenous 0.9% saline solution (9IV; osmolality = 308 mOsm · kg⁻¹), or (c) a no rehydration control (NF). Subjects performed the trials in random order, with at least 14 days between trials. A certified IV nurse performed all IV procedures. A 21-gauge butterfly cannula was placed on the arm opposite of the indwelling cannula. The rate of IV infusion was 0.56 ml · kg⁻¹ · min⁻¹. The rehydration process took 45 min, and the total rehydration volume was 25ml · kg of body weight⁻¹. This volume was selected because it has been determined to be the greatest amount that can be consumed orally over a 45-min period (16).

Following rehydration, three more blood samples were drawn at 15-min intervals, while subjects remained standing for an additional 55 min. Subjects then re-entered the environmental chamber (36 °C). Following a 20-min standing equilibration period, a blood sample was taken, and the subjects consumed 1g · kg body weight⁻¹ of a commercial carbohydrate source (Skittles, M&M Mars, Hackettstown, NJ). The CHO was 90% glucose/sucrose by weight; therefore, the amount of CHO was corrected to achieve the proper dose. The subjects were also given a small amount (<25 ml) of water to moisten their mouths and aid swallowing.

For the 90-min performance test, subjects walked on a motorized treadmill, up a 5 to 10% grade, to achieve an exercise intensity of approximately 50% of their \( \dot{V}O_2 \text{max} \), for up to 90 min. \( \dot{V}O_2 \) and RER measurements were collected for a 2.5-min period every 20 min. During the performance test, 4-ml blood samples were taken at 15-min intervals. Heart rate and rectal temperature were constantly monitored during the performance trial; the criteria for the cessation of testing were identical to the dehydration phase (see above).

**Blood Analyses**

From whole blood, the hematocrit was determined in triplicate using the microcapillary technique and hematological centrifuge (Damon, Nedham Heights, MA). Hemoglobin was determined in triplicate using the cyanmethemoglobin technique (Kit 525, Sigma, St. Louis, MO) and spectrophotometer (Spectronic 88, Bausch & Lomb). From each sample, 2 ml of blood were placed in lithium-heparin tubes and 2 ml in serum separation tubes (Vacutainer, Becton Dickinson, Rutherford, NJ). The blood samples were centrifuged for 10 min at 1,200 × g (Sorvall RT600B) and the cells separated from plasma or serum, respectively. Plasma from the lithium-heparin tubes was analyzed in triplicate for glucose and lactate concentrations by enzymatic technique (YSI 2300 STAT glucose/lactate analyzer, Yellow Springs, OH), and its osmolality was determined with a freezing point depression osmometer (MicroOsmette, Precision Instruments, Natick, MA). The serum samples were frozen and stored at −80 °C and later analyzed for insulin concentration using a standard radioimmunoassay kit (Coat a Count, Diagnostic Products, Los Angeles, CA).
Additional blood was taken pre- and post-dehydration and at 15 and 45 min of exercise and then analyzed in triplicate for epinephrine and norepinephrine concentrations by high pressure liquid chromatography (HPLC) (Waters, Milford, MA). Six ml of blood were drawn and placed in chilled lithium-heparin tubes containing 120 μL of an EGTA/glutathione solution and centrifuged. From the plasma, epinephrine (EPI) and norepinephrine (NE) were extracted by absorption onto aluminum oxide. After washing, EPI and NE were eluted with an acidic solution and subsequently analyzed using 3,4-dihydroxybenzylamine as the internal standard. The mobile phase (flow rate, 0.9 ml · min⁻¹) consisted of 95% water and 5% methanol with added buffer salts.

**Statistics**

Statistical analysis of the data involved a repeated measures, two way (time × treatment) analysis of variance (ANOVA), to determine if significant differences existed across time and between treatments. Where significant differences were noted, Newman-Keuls post hoc comparisons were employed. Analysis of area under the curve (time vs. concentration), above the post-rehydration concentration, was accomplished using a trapezoid method; only positive values were used to compare differences in hormone and metabolite concentration across time and between conditions. The .05 level of significance was selected. All values were expressed as mean ± standard error.

**Results**

**Dehydration**

Body weight lost during the dehydration phase of the protocol averaged 4.1 ± 0.1% of the PRE DH weight and did not differ significantly among trials (p > .05). The time to achieve the desired body weight loss and the exercise intensity averaged 186.1 ± 12.1 min and 50.5 ± 6.1% of \( \dot{V}O_{2\text{max}} \), respectively. There were no differences in these measurements among trials (p > .05).

**Rehydration**

The volume of fluid infused during the rehydration phase of the study was not significantly different (p > .05) among any of the four rehydration trials. Overall, the volume of the rehydration averaged 24.7 ml · kg body weight⁻¹.

**Performance Trial**

Time to exhaustion during the performance trial (EX) was statistically similar (p > .05) among the two rehydration trials (45IV, 77.8 ± 5.2 min; 9IV, 76.0 ± 5.7 min). However, the NF trial resulted in a significantly shorter time to cessation of exercise than all other trials (58.3 ± 8.3 min, p < .05). Heart rate (HR) increased significantly (p < .001) through 45 min of exercise (45EX) in all four treatments. After 15 min of exercise (15EX), the NF treatment showed significantly higher HR (p < .001) compared to all other treatments and continued to be significantly elevated through 45EX. Mean rectal temperature (\( T_{re} \)) increased significantly with time through EX in all trials (p < .001). Pre-exercise \( T_{re} \) was significantly greater in the NF trial than in
all other trials ($p < .001$), and it remained significantly greater than the other four treatments through 75EX ($p < .01$). No significant differences among groups or across time were observed for RER values during the performance trial (see Table 1).

**Blood Variables**

Figure 1 depicts plasma osmolality (OSM) as a function of time throughout the protocol. Mean post-dehydration (POST DH) values were all significantly higher ($p < .01$) than the PRE DH values in all five trials. Following rehydration, (PRE EX), the 45IV values decreased to levels significantly lower ($p < .05$) than at POST DH. However, at PRE EX, the NF and 9IV OSM were not significantly different ($p > .05$) between them and not significantly different from POST DH. Throughout the performance trial (15EX to 90EX), OSM increased as a function of time with values significantly higher than PRE EX ($p < .05$) in all trials. OSM of the NF trial was higher ($p < .05$) than both rehydration trials at all time points during the performance trial, but no statistical difference existed between the rehydration trials (45IV and 9IV). Plasma lactate concentrations showed no significant differences throughout the entire protocol across time or among trials ($p > .05$).

**Glucose**

Because differences in hydration status affect plasma volume and thus concentration of metabolites, glucose measures were corrected for plasma volume change (Figure 2). Post dehyrdration mean plasma glucose concentration (GLU) was elevated in all trials, although the difference did not achieve statistical significance.

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**Figure 1 — Plasma osmolality (OSM) throughout the experimental protocol.**

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$^a$Significantly higher mean OSM POST DH versus PRE DH in all trials ($p < .01$).

$^b$Significantly lower OSM at PRE EX versus POST DH ($p < .05$). $^c$Significantly higher mean OSM in NF versus all other trials ($p < .05$). Values are means ± SE.
Figure 2 — Plasma glucose concentrations throughout the experimental protocol after correction for plasma volume changes. *Significantly higher mean glucose concentration compared to PRE EX in all trials except NC (p < .01). †Significantly higher mean glucose concentration in NF versus all other trials (p < .05). ‡Significantly greater area under the glucose curve during EX in NF versus all other trials (p < .05). Values are means ± SE.

(p > .05). No differences in GLU were observed during the rehydration phase. At 15EX, GLU was significantly higher (p < .01) than at all previous time points in all groups. GLU returned to levels not significantly different from PRE EX by 45EX in the 45IV and 9IV groups. GLU values in the NF group remained significantly elevated (p < .05) from PRE EX through 75EX. GLU was significantly (p < .05) higher in NF than in 45IV and 9IV at 60EX and 75EX. Area under the GLU curve (corrected for plasma volume change) during the performance trial was significantly greater (p < .03) in the NF trial (1966 ± 340 mg·dl⁻¹·min), when compared to the 45IV (1233 ± 162 mg·dl⁻¹·min) and 9IV (1390 ± 180 mg·dl⁻¹·min) groups.

**Hormonal Variables**

Figure 3 shows the values for serum insulin. At 15EX, INS in the 45IV treatment was significantly greater than PRE EX (p < .05). Nonetheless, when compared to the other trials, due to high inter-subject variability, the differences did not achieve statistical significance (p = .07).

No significant differences in plasma epinephrine concentration were observed among any of the treatments or across time. However, plasma norepinephrine concentrations were significantly greater at POST-DH as well as at 15EX and 45EX (p < .05) with respect to PRE-DH values in all trials. Furthermore, plasma norepinephrine concentrations observed in the NF group were significantly higher at 15EX and 45EX versus both IV trials (Figure 4).
Figure 3 — Serum insulin concentration. aSignificantly higher change at 15EX versus PRE EX in 45IV ($p < .05$). No significant differences existed between trials. Values are means ± SE.

Discussion

The results of this study, as expected, showed decreased exercise tolerance during the NF trial compared to all the treatments involving rehydration. However, the time to exhaustion was not different between rehydration treatments (45IV or 9IV). The shorter times to exhaustion in the NF trial were the consequence of the lower volume of body fluid, which decreased exercise capacity by decreasing the effective central blood volume and thus reduced venous return and stroke volume (21). In accordance with this, the NF group showed a significantly higher HR throughout EX in order to defend cardiac output. Furthermore, the significantly higher $T_a$ seen during the NF trial is consistent with compromises in heat dissipation known to result during exercise when large amounts of body fluid are lost (17, 23).

Consistent with the greater fluid volume loss, plasma osmolality (OSM) was significantly higher ($p < .05$) during EX in the NF trial than in the rehydration trials. Although, PRE-EX OSM was significantly higher in the 9IV trial compared to 45IV, after the first 15 min of exercise, OSM was similar in both treatments (Figure 1). These results were similar to those obtained by Owen et al. (18), who found no significant differences in OSM during exercise in a hot environment (35°C) when subjects rehydrated by drinking hypertonic or hypotonic glucose-electrolyte solutions. This response suggests that the body was able to defend plasma osmolality during exercise at the $-1.5\%$ hypohydration level when rehydrated with either a hypertonic (45IV = 154 mOsm·kg⁻¹) or slightly hypertonic (9IV = 308 mOsm·kg⁻¹) solution.

The most relevant finding of this study was that plasma glucose concentration following CHO ingestion remained significantly elevated (above 124 mg·dl⁻¹) for up to 30 min longer during EX in the NF trial compared to the treatments where subjects were rehydrated (45IV and 9IV). This is best illustrated by the significantly greater area under the glucose curve in NF. Although plasma volume during the NF trial was reduced compared to the rehydration trials, when glucose concentrations
Figure 4 — Plasma epinephrine (top) and norepinephrine (bottom). a) Significant increases in NE compared to PRE DH and PRE EX in all trials ($p < .01$). b) Significantly higher plasma norepinephrine concentration in the NF versus all other trials at 15EX and 45EX ($p < .05$). Values are means ± SE.

were corrected for this variable, the differences persisted. Thus, exercise in the heat with a fluid deficit equivalent to −4% body weight resulted in alterations of glucose homeostasis compared to the partially rehydrated trials. The fact that plasma glucose was significantly higher at 60EX and 75EX in the NF trial compared to the 45IV and 9IV trials but, at 90EX, significant differences were no longer detectable, was most probably owed to the loss of statistical power, as very few subjects were able to reach 90EX in the NF trial.

While some studies have shown that severe hypohydration can reduce the rates of gastric emptying during exercise following the ingestion of a large bolus (~400 ml) of 7 to 9% CHO and electrolyte solutions (20), such effects have not been observed in studies were small loads of fluid and CHO are ingested by subjects (22). In the present study, subjects ingested a single small (~75 g), bolus of CHO. In addition, parenteral administration of fluids was utilized, thereby eliminating possible effects of different fluid osmolality or oropharyngeal reflexes on gastric emptying or intestinal absorption rates. Furthermore, the initial rise in plasma glucose
(15EX) was similar for all treatments. Hence, it is unlikely that differences in gastric emptying or intestinal absorption could account for the differences in plasma glucose observed later on during EX.

Although sympathetic nervous system activity was increased to a greater extent (greater norepinephrine concentrations) in the NF trial than in the 45IV and 9IV trials, in the present study, plasma epinephrine that did not increase during EX was not different between trials. This response was consistent with the findings of Peskind et al. (19), who showed that infusion of a 5% NaCl solution to normal men resulted in marked increases in norepinephrine, but not epinephrine, by selectively activating the cardiovascular, but not the adrenomedullary components of the sympathetic nervous system. Since it has been shown that norepinephrine has no effect on glucose production during exercise (11), it is also improbable that it was responsible for the higher plasma glucose during EX.

The uptake of glucose into muscle cells during exercise occurs predominantly via a contraction-mediated mechanism (9). However, the presence of insulin is necessary to produce a maximal effect on glucose uptake, especially following the ingestion of a bolus of food (25). Research on non-exercising individuals has shown that increases in plasma OSM per se can lead to decreases in the insulin-mediated glucose uptake by tissues in diabetic (27) as well as in healthy individuals (3). In the present study, whereas the 45IV trial showed a significantly increment in insulin at 15EX compared to PRE EX, the NF and 9IV treatments showed a blunted insulin response. However, the actual values of the 45IV responses did not achieve a statistically significant \( p = .07 \) difference from the other treatments. Nevertheless, glucose concentrations, even when corrected for plasma volume changes, were significantly elevated in the NF trial compared to the other treatments after 45EX. Moreover, they remained significantly elevated compared to PRE EX through 75EX in that same trial. Thus, the concentrations of insulin that were sufficient to promote normal glucose disposal in the 45IV and even the 9IV trials appeared to be less effective in the NF trial. Therefore, these findings are consistent with the hypothesis of decreased muscle insulin sensitivity due to hyperosmolality in the NF trial (3, 27).

In this context, it has been shown that both hypovolemia (10) and hyperosmolality (19) can increase norepinephrine secretion, which by itself can also decrease muscle insulin sensitivity (13). In addition, high norepinephrine levels are known to inhibit pancreatic insulin secretion during exercise (14). Thus, the observed response is consistent with the known consequences of the high OSM (300–305 mOsm \( \cdot \) kg\(^{-1}\)) as observed during EX at the \(-4\%\) hypohydration level of the NF trial. The significantly higher osmolality in that trial may have caused the greater release of norepinephrine. Norepinephrine could have suppressed insulin release to levels not different from those in the rehydrated trials, despite the higher plasma glucose concentration, thus hindering glucose entry into cells for its metabolism.

A previous study by Below et al. (2) demonstrated that CHO and fluid ingestion have additive beneficial effects on performance. However, hypohydration level in that study was only \(-2\%\), and the highest osmolality achieved by their subjects was similar to our rehydrated subjects, around 295 mOsm \( \cdot \) kg\(^{-1}\). This points to the possibility of a threshold of hypovolemia or hyperosmolality at which the negative effects on glucose homeostasis become apparent. On the other hand, the detrimental effects of hypohydration on glucose homeostasis observed in this study were likely not the cause of decrements in performance. This assertion is supported by the fact that even in the NF trial, the respiratory exchange ratios (i.e., an index of carbohydrate
Table 1    Average Physiological Responses to the Performance Test

<table>
<thead>
<tr>
<th>Trial</th>
<th>Duration (min)</th>
<th>Max HR (bpm)</th>
<th>Max $T_r$ (°C)</th>
<th>Average exercise RER</th>
</tr>
</thead>
<tbody>
<tr>
<td>45IV</td>
<td>77.9 ± 5.2</td>
<td>143 ± 4</td>
<td>38.8 ± 0.1</td>
<td>0.86 ± 0.03</td>
</tr>
<tr>
<td>9IV</td>
<td>76.0 ± 5.7</td>
<td>144 ± 4</td>
<td>38.8 ± 0.1</td>
<td>0.88 ± 0.02</td>
</tr>
<tr>
<td>NF</td>
<td>58.3 ± 8.3*</td>
<td>158 ± 2*</td>
<td>39.3 ± 0.1*</td>
<td>0.86 ± 0.02</td>
</tr>
</tbody>
</table>

*Significantly different from 45IV and 9IV ($p < .05$).

utilization) were not different between trials. Under the circumstances of the present study (intensity of exercise = 50% of $\dot{V}O_{2\text{max}}$), the rate of glucose uptake by the exercising muscle cells may not have been a limiting factor. Future studies should be conducted at higher intensities of exercise (above 75% $\dot{V}O_{2\text{max}}$), where blood glucose utilization may become a limiting factor during prolonged exercise. Methods to determine precise partitioning of insulin-dependent and independent glucose uptake should also be utilized.

In conclusion, this study shows that hypohydration of −4% of body weight resulted in altered glucose homeostasis during exercise in the heat following ingestion of 1 g · kg$^{-1}$ CHO, such that plasma glucose remained elevated for a longer time (an additional 30 min) compared to the partially rehydrated state. The osmolality of the infused fluid (hypotonic or slightly hypertonic) had no significant effect on this phenomenon.

References


