Carbohydrate loading and supplementation in endurance-trained women runners

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Andrews, Jessica L., Darlene A. Sedlock, Michael G. Flynn, James W. Navalta, and Hongguang Ji. Carbohydrate loading and supplementation in endurance-trained women runners. J Appl Physiol 95: 584–590, 2003.—The purpose of this study was to examine the effect of carbohydrate (CHO) augmentation on endurance performance and substrate utilization in aerobically trained women. Eight endurance-trained women completed a 24.2-km (15 mile) self-paced treadmill performance run under three conditions: CHO supplementation (S), CHO loading and supplementation (L+S), and placebo (P). Dietary CHO was ∼75% of energy intake for L+S and ∼50% for both S and P. A 6% CHO-electrolyte solution (S and L+S) or placebo (P) was ingested preexercise (6 ml/kg) and every 20 min during exercise (3 ml/kg). Blood glucose was significantly higher at 40, 60, and 100 min during L+S, and at 60, 80, and 100 min during S compared with P (P < 0.05). Blood lactate was significantly higher (P < 0.05) during L+S than S and P. Blood glycerol was significantly lower (P < 0.05) at 20, 80, and 100 min during L+S, and at 80 and 100 min during S than P. The proportion of CHO (%) utilized during exercise was significantly higher (P < 0.05) during L+S (71.3 ± 3.8%) and S (67.3 ± 4.3%) than P (59.2 ± 4.6%). Performance times (P > 0.05) were 132.5 ± 6.3 min (S), 134.4 ± 6.3 min (L+S), and 136.6 ± 7.9 min (P). In conclusion, it appears that when CHO availability in women is increased through CHO loading and/or CHO supplementation, there is a concomitant increase in CHO utilization. However, this may not necessarily result in significantly improved performance.

endurance performance; substrate utilization; glucose; glycerol; lactate

CARBOHYDRATE (CHO) loading is known to produce an increase in stored muscle glycogen, often allowing exercise to be prolonged and/or performance to be improved (6, 9, 20, 34). Whereas the performance-enhancing effect of CHO augmentation has been demonstrated in male athletes, CHO loading has not been shown to be equally effective in female athletes. For example, Tarnopolsky et al. (29) found that women neither increased muscle glycogen concentration nor improved cycling performance after 4 days of ingesting a high-CHO diet (75% of energy intake). In contrast, Walker et al. (32) reported a 13% increase in muscle glycogen and a significant increase in cycling time to fatigue in women after 6 days on a high-CHO diet (78% of energy intake). However, the magnitude of these changes was smaller than those previously observed in male athletes.

CHO supplementation during prolonged endurance exercise is thought to prevent a decline in blood glucose concentration, thus facilitating a high rate of CHO oxidation during the latter stages of exercise (4, 12, 16, 31, 35). Maintaining blood glucose concentration may also delay the onset of fatigue, allow exercise intensity to be sustained, and result in improved performance. The relationship among CHO ingestion, blood glucose, and performance enhancement has been extensively investigated (9, 12, 15, 22, 24, 31), particularly with respect to male athletes. Although there is an increased interest in understanding the interaction of CHO intake and exercise performance in women, investigators have primarily examined CHO loading (29, 32) with less attention being given to CHO supplementation (18). Thus the response of women to CHO feedings during endurance exercise, alone or in combination with CHO loading, requires more research.

It is important to understand how women respond to CHO augmentation during endurance running for two reasons. First, female endurance runners believe that CHO intake can be beneficial despite the fact that there is a paucity of scientific evidence in support of this notion. Second, during prolonged endurance exercise, men tend to utilize CHO to a greater extent than women, whereas women tend to preferentially utilize more lipid (13, 17, 18, 28). Possible reasons include differences between men and women in the distribution and/or activation of α- and β-adrenergic receptors (5), aerobic capacity and/or fitness level (13), exercise intensity (13, 17), and the lack of a sufficient amount of CHO ingested by women (compared with men) during and before exercise (5, 13, 17, 30). However, the gen-
eral consensus among researchers is that it is likely mediated by endocrine differences. Regardless of the mechanisms involved, it could be hypothesized that CHO feedings would not enhance performance in female athletes to the extent previously seen in similarly trained male athletes because of their relatively greater reliance on lipid.

Cycling has been selected most often as the mode of exercise used to investigate the effect of CHO feedings on performance (18, 22, 24, 36). This may be due to the fact that athletes tend to have more difficulty ingesting fluid and experience a greater level of discomfort from fluid ingestion during running than cycling (9, 10). Additionally, it is logistically easier for researchers to obtain physiological and hematologic measures during cycling compared with running. To our knowledge, there is no information regarding the effect of CHO supplementation or the combination of CHO loading and supplementation on run performance in women. Thus the purpose of this study was to examine the combined effects of CHO loading and supplementation, as well as the effects of CHO supplementation alone, on metabolic, hematologic, and performance variables in endurance-trained female athletes during prolonged exercise. We hypothesize that augmenting CHO intake in women, either by supplementation alone or supplementation combined with loading, will result in greater CHO utilization during exercise but little increase in performance.

METHODS

Subjects. Eight female athletes, 20–40 yr of age, volunteered to participate in the study. Criteria for inclusion were a training history of at least 12 mo of endurance-type physical activity at a frequency of four times per week and/or a duration of 6–7 h/wk, weight stable (<2.5 kg) for at least 1 yr, eumenorrheic with a normal cycle length of 23–38 day, and on a dietary regimen in which CHO intake was not >65% of the total energy intake. Potential subjects with unstable eating tendencies (e.g., skipping meals on a daily basis, macronutrient deficiencies such as a no-fat or no-protein diet, and so forth) were excluded from participation. All procedures were approved by the Institutional Committee on the Use of Human Subjects in Research before data collection. Additionally, subjects provided written, informed consent before admission into the study.

Experimental design. Subjects completed three different 24.2-km (15 miles) treadmill performance runs. With the use of a Latin square (counterbalanced) design, subjects completed each of the following three trials: no CHO loading + CHO supplementation (placebo; P), CHO supplementation only (S), and CHO loading + CHO supplementation (L+S). Each trial was completed during the midfollicular phase of the menstrual cycle (between days 5 and 10 from the first day of menses) to minimize the effects of gonadotrophic hormones on fuel metabolism. Thus subjects performed one trial per month.

Preliminary measurements and procedures. Before experimental testing, subjects completed health, training, and menstrual cycle history questionnaires. They were also asked to complete a 3-day food diary and a 3-wk training log to ensure that they met the dietary and training criteria necessary for inclusion into the study. On meeting the study requirements, participants were given a list of food choices and asked to identify foods they consumed on a regular basis (familiar foods). From these choices, menus were generated for the women to follow for 4 days immediately before each experimental trial (Computer Planned Menus, Nutritional Computing Concepts, Indianapolis, IN). Additionally, subjects were given a dietary exchange book to help them approximate the amount of food they consumed on a daily basis and adhere to the prescribed dietary menus before each trial.

Maximal oxygen consumption ($V_{O2\text{ max}}$) was measured ~2 wk before the first experimental trial by using a continuous, incremental treadmill running test to exhaustion. On a separate day, subjects completed a self-paced 60-min treadmill run to familiarize themselves with the procedure for controlling treadmill speed and the drinking patterns and measurements to be used during the experimental trials.

Exercise taper. Subjects ran for 60, 40, 40, 20, 20, and 0 min for the 6 days immediately before each trial, respectively. They were also required to log their daily activity during the week preceding each trial to ensure that they were following the prescribed tapering protocol.

Subjects followed a 4-day diet before each experimental trial and recorded their dietary intake during this time to ensure they were ingesting a high-CHO diet (75% energy intake) before the L+S trial and a moderate-CHO diet (50% energy intake) before the S and P trials. Total energy intake was unchanged, but dietary composition was altered to either increase or decrease the percentage of dietary CHO. The high-CHO regimen required subjects to ingest a greater percentage of energy in the form of complex CHO and commercially available glucose polymers in solution (Gatorlode, Quaker Oats, Chicago, IL).

Experimental procedure. Experimental beverages were administered in a double-blind fashion during the performance trials. Subjects received a 6% CHO-electrolyte solution (Gatorlode, Quaker Oats) during trials S and L+S and a similarly flavored artificially sweetened placebo during the P trial. The placebo solution was similar to the supplement solution in electrolyte and mineral content, coloring, and flavoring, but it did not contain the CHO.

Subjects arrived at the laboratory between 0700 and 0800 on the morning of the trial after a 10-h fast. Body weight was measured, a fingertip blood sample was obtained from a prewarmed hand, and 6 ml/kg of either the placebo or the CHO supplement was ingested. Subjects completed a 3-min self-paced warm-up run, rested for 2 min, and then began the 24.2-km time trial. Every 20 min throughout exercise, subjects ingested 3 ml/kg of the prescribed solution. The 24.2-km time trial was self-paced, and subjects were encouraged to complete the exercise in the shortest time possible. Pacing was accomplished through the use of photocells positioned at the front and rear of the treadmill, which allowed treadmill speed to be controlled by the subject simply by moving forward or “drifting” back on the treadmill to interrupt a light beam to increase or decrease speed, respectively. The treadmill was interfaced with a desktop computer and a software program that allowed subjects to see continuous updates of speed and distance on a nearby monitor.

Oxygen consumption ($V_{O2}$), heart rate (HR), and ratings of perceived exertion (RPE) were measured every 30 min during the run starting at minute 25. Subjective level of gastrointestinal (GI) discomfort was obtained throughout the run by using a nonvalidated scale (1 = comfortable, 2 = partially full, 3 = full, 4 = uncomfortably full, 5 = nauseous). A small blood sample (200 µl) was collected from a fingertip every 20 min, with the beverage administered immediately after the blood collection. No measurements were taken and no bever-
ages were administered after the 21-km (~13 mile) mark; i.e., subjects ran uninterrupted for the remainder of the run. On completion of the run, subjects were weighed and a final blood sample was obtained 5 min postexercise.

**Measurements.** Expired air was collected into Douglas bags. Volume was determined by forcing the contents of the bag through a dry-gas meter (Parkinson-Cowan). Respiratory gases were measured by using O2 (paramagnetic) and CO2 (infrared) analyzers (ParvoMedics, Salt Lake City, UT). The analyzers were calibrated before each series of analyses by using gases with known concentrations. VO2 and carbon dioxide production were determined from expired volume and the percentages of O2 and CO2. Nonprotein respiratory exchange ratio (RER) and VO2 were used to estimate the amount of CHO oxidized during the run. RPE was measured by using the Borg 6–20 category-ratio scale of perceived exertion, and HR was measured telemetrically (Polar Vantage XL, Polar, Stamford, CT).

**Blood sample preparation, storage, and analysis.** Blood collected before, during, and after the run was used for the subsequent determination of glucose, lactate, and lactate concentration. Approximately 200 µl of whole blood (WB) were obtained at each sampling point and dispensed into a prechilled tube. Fifty microliters of WB were deproteinized in 200 µl of cold 8% perchorlic acid. After centrifugation, an aliquot of the perchloric acid extract was stored at ~80°C for subsequent analysis of lactate concentration. Of the remaining acid extract, 120 µl were mixed with 50 µl of cold potassium hydroxide and then stored at ~80°C for subsequent analysis of glycerol. WB was centrifuged and stored at ~80°C for future determination of serum glucose concentrations.

Glucose was determined spectrophotometrically by using a commercially available glucose kit [Infinity Reagent, procedure no. 18-UV; standards and controls (Accutrol); Sigma-Aldrich, St. Louis, MO]. The perchloric acid extract was assayed for lactate by using an enzymatic technique (23). Twenty microliters of sample, standard, or controls were added to a cuvette with 1 ml of a reagent cocktail containing excess NAD+ (~12 mmol/l), lactate dehydrogenase (bovine heart, >300 U/ml), and 1 M glucose-0.8 M hydrazine buffer (pH 9.2). Glycerol was analyzed fluorometrically by using an enzymatic method (3).

**Statistical analyses.** Values are presented as means ± SE. A repeated-measures one-way ANOVA was used to determine the effects of the experimental treatments on performance time and total grams of CHO oxidized. Blood lactate, glucose, and glycerol values were analyzed by using separate two-way ANOVA with repeated measures. Tukey’s post hoc tests were used where appropriate. Additionally, a Friedman’s nonparametric matched-sample statistical test was conducted to test for a possible order or training effect. Statistical significance was accepted at P < 0.05.

### Results

**Subject characteristics.** Subject characteristics are presented in Table 1. Subjects were endurance trained and had been running an average of ~53 km/wk for at least 12 mo before the study.

**Pretrial conditions.** Training diaries collected on the morning of each trial showed that all subjects complied with pretrial training requirements; i.e., each subject followed the prescribed exercise taper. Diet records for the 4 days preceding each trial showed that subjects achieved the CHO intake goals for each experimental condition. Significantly greater CHO intake (P < 0.05) was consumed before the L+S trial (72.8 ± 2.0% CHO) than either the P trial (52.3 ± 1.2% CHO) or S trial (53.3 ± 1.4% CHO). This corresponded to 334.9 ± 29.0 g of CHO (5.45 ± 0.43 g/kg) for the L+S trial vs. 214.2 ± 15.1 g CHO (3.49 ± 0.21 g/kg) and 238.2 ± 15.3 g CHO (3.88 ± 0.21 g/kg) for the P and S trials, respectively. Total energy intake was not significantly different for the 4 days before each trial (P > 0.05).

**Laboratory conditions.** Laboratory temperature was 23.4 ± 0.6, 23.3 ± 0.5, and 23.6 ± 0.8°C, and relative humidity was 53.3 ± 3.3, 51.0 ± 4.3, and 50.4 ± 7.7% for the L+S, S, and P trials, respectively (P > 0.05). The women were cooled by an electric fan throughout the run.

**Performance time.** There was no significant difference in performance time among the trials (P > 0.05). Mean performance times were 132.5 ± 6.3 min for the S trial, 134.4 ± 6.3 min for the L+S trial, and 136.6 ± 7.9 min for the P trial. Six of the eight subjects completed the run faster with CHO augmentation (S and L+S trials) compared with ingestion of the placebo. Results of the Friedman’s test indicated that there was no order effect for performance times (P > 0.05), suggesting that learning and/or training effects did not occur over the course of the investigation.

**HR.** No significant treatment effect or treatment × time interaction was found. HR was maintained at ~172.8 ± 3.5, 172.6 ± 3.9, and 167.7 ± 3.1 beats/min during the L+S, P, and S trials, respectively (P > 0.05).

**RPE.** The RPE response did not differ among experimental conditions, nor was there a treatment × time interaction (P > 0.05). There was a significant time effect such that RPE was higher (P < 0.05) at 120 min (16.0 ± 1.5) than at 30 min (11.6 ± 1.1) across all trials. VO2. There were no treatment, time, or treatment × time effects for VO2 (P > 0.05). Exercise elicited an average VO2 of 36.2 ± 1.2, 37.2 ± 2.0, and 37.2 ± 1.6 ml·kg−1·min−1, which corresponded to 68.5 ± 0.02, 69.8 ± 0.02, and 69.6 ± 0.02% of VO2max for the S, P, and L+S trials, respectively.

**RER and substrate utilization.** There was no significant time effect or treatment × time interaction for RER, but there was a significant treatment effect (P < 0.05). As shown in Fig. 1, RER values were significantly higher during the L+S (0.92 ± 0.01) and S

### Table 1. Descriptive data for the trained female runners

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SE</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height, cm</td>
<td>165.9 ± 2.4</td>
<td>155–170</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>61.4 ± 2.3</td>
<td>52–69</td>
</tr>
<tr>
<td>V02max, ml·kg−1·min−1</td>
<td>51.7 ± 2.7</td>
<td>44–63</td>
</tr>
<tr>
<td>HRmax, beats/min</td>
<td>192.1 ± 2.9</td>
<td>182–202</td>
</tr>
<tr>
<td>Training history, km/wk</td>
<td>52.9 ± 3.36</td>
<td>32–60</td>
</tr>
<tr>
<td>days/wk</td>
<td>4.9 ± 0.5</td>
<td>3–6</td>
</tr>
</tbody>
</table>

Values are for 8 subjects. V02max, maximal oxygen consumption; HRmax, maximal heart rate.
Postexercise. No significant differences were detected between the S and L+S trials. Plasma glucose was similar among treatments before each run, averaging 3.9 ± 0.2, 3.9 ± 0.2, and 4.0 ± 0.2 mmol/l for the P, L+S, and the S trials, respectively (P > 0.05).

Blood lactate. As shown in Fig. 3, blood lactate concentration was similar among treatments before each run (P > 0.05). A significant treatment effect was observed (P < 0.05) such that blood lactate during exercise was significantly higher in the L+S (3.0 ± 0.4 mmol/l) compared with both the P trial (2.6 ± 0.3 mmol/l) and the S trial (2.7 ± 0.3 mmol/l). There was no significant treatment × time interaction (P > 0.05).

Blood glycerol. Mean glycerol values are displayed in Fig. 4. Glycerol levels were similar among treatments before each run (P > 0.05). A significant treatment × time interaction was found (P < 0.05) such that glycerol levels were significantly higher at 20 min, 80 min, 100 min, and postexercise during the P trial compared with the L+S trial. Glycerol levels were also significantly higher at 80 min, 100 min, and postexercise during the P trial compared with the S trial (P < 0.05). No significant differences were detected between the S and L+S trials.

Fluid consumption and weight loss. Total fluid consumption was 1,434.8 ± 108.9 ml and did not differ among treatments. The initial bolus was 374.0 ± 13.3 ml, and the amount ingested every 20 min throughout the run was 191.8 ± 8.9 ml. Thus the hourly fluid consumption averaged 575.5 ± 26.7 ml. Total CHO intake during the S and L+S trials averaged 86.1 ± 6.5 g (1.40 ± 0.07 g/kg), which included 22.4 ± 0.8 g CHO (0.37 g/kg) in the initial bolus and an additional 28.8 ± 5.1 g/h (0.47 ± 0.04 g·kg⁻¹·h⁻¹) during the run. Body weight was significantly lower after exercise.
Blood glucose was maintained at or above resting values throughout exercise in all three trials and was significantly higher in the L+S and S trials compared with the P trial. Maintenance of blood glucose concentration during the P trial is not unusual and has been observed in previous run studies (6, 9, 27, 34). Lactate concentration was significantly higher during the L+S trial compared with both the P and S trials, indicating that CHO loading in addition to supplementation may significantly increase CHO oxidation, perhaps by utilizing muscle glycogen. It has been shown that the primary substrate for lactate production in muscle is glycogen (14). Previous researchers have reported higher blood lactate levels after CHO loading compared with normal or depressed glycogen levels (1, 21), indicating that glycogen levels influence its utilization during exercise. It has also been shown that when lactate and glucose are infused simultaneously during exercise, lactate turnover exceeds that of glucose, indicating that muscle glycogen provides most of the CHO oxidized during exercise (7). Muscle glycogen concentrations were not measured in the present study, so it is uncertain whether CHO ingestion throughout exercise had any effect on muscle glycogen utilization during exercise. However, the significantly higher RER values observed during the L+S and S vs. P trials are reflective of a higher rate of CHO metabolism, indicating that when CHO is made available through CHO augmentation, trained female runners will preferentially utilize CHO.

Glycerol concentrations were significantly higher in the P trial compared with the L+S and S trials, indicating a greater contribution of fat metabolism during the P condition. Although glycerol concentrations rose steadily during all trials, the increase was significantly different in weight loss among the trials.

GI discomfort. GI discomfort was similar among the three trials, averaging 3.56 ± 0.37, 3.44 ± 0.49, and 3.44 ± 0.47 for the L+S, P, and S trials, respectively. On average, subjects felt somewhere between full and uncomfortably full throughout the run. They tended to experience increased GI discomfort later in the run during all three trials (P > 0.05), suggesting that the discomfort was a result of the volume rather than the type of beverage ingested.

DISCUSSION

Both CHO loading (31) and CHO supplementation (2) have been shown to increase cycling performance in women. However, to our knowledge, there is no information on the effect of CHO supplementation and/or supplementation combined with CHO loading on metabolism, substrate utilization, and endurance performance in women. The main finding of this study was no significant difference in performance time among the three experimental trials. Although mean performance time of the S and L+S trials were ~4 and 2 min faster than the P trial, respectively, these differences did not reach statistical significance. Additionally, the RPE, HR, and VO2 responses did not differ among experimental conditions, indicating that subjects were running at a similar level of physical exertion for each experimental condition. A post hoc power analysis indicated that it would have taken ~20 female subjects to detect a performance difference in this study with 80% power at α = 0.05. Therefore, it is possible that the inclusion of more subjects would have resulted in a different outcome with respect to performance. However, despite the lack of a significant difference in performance across the three exercise trials, significant differences were noted in substrate utilization and in the blood glucose, lactate, and glycerol responses.
Women in the present study consumed ~335 g CHO/day, or 5.45 g CHO·kg⁻¹·day⁻¹, before the L+S trial. Because only one subject performed optimally during that trial, it is possible that the amount of CHO consumed was inadequate for improving performance. These findings are similar to results of a previous study by Tarnopolsky et al. (29), where CHO loading failed to increase glycogen stores and also failed to significantly improve cycling performance. The women in their study consumed ~6.4 g CHO·kg⁻¹·day⁻¹ (370 g CHO/day) during the CHO loading regimen. However, in a study by Brewer et al. (6), male and female subjects significantly increased run time to exhaustion after consuming ~7–7.5 g CHO·kg⁻¹·day⁻¹ for 3 days before the run trial. In absolute terms, the subjects were consuming 462–507 g CHO/day. Because male and female data were pooled, the relative and absolute amount of CHO the women were consuming is unclear. In another study (32), female cyclists had a significant increase in glycogen stores and a significant improvement in performance after a CHO-loading regimen requiring them to consume 8.14 g CHO·kg⁻¹·day⁻¹ (464 g CHO/day). Tarnopolsky et al. (30) recently suggested that perhaps women fail to increase their muscle glycogen stores to the same extent as male athletes in response to CHO loading because they do not consume the same absolute amount of CHO as men.

It has been suggested that a CHO dosage of 30–60 g/h is necessary for subjects to benefit from CHO supplementation (12, 15). However, Murray et al. (26) found on two separate occasions that the ingestion of 26 and 78 g CHO/h increased cycle performance to a similar extent, suggesting that 26 g CHO·h⁻¹ was adequate to enhance performance. CHO dosage throughout the S and L+S trials of the present study was ~29 g/h (0.47 g·kg⁻¹·h⁻¹), with an additional 22.4 g (0.37 g/kg) administered in the preexercise bolus. Relative to body weight, this was comparable to the amount of CHO administered to male runners in previous studies (10, 31). Run time to exhaustion in those men was significantly increased when ingesting CHO at a rate of 0.33 g·kg⁻¹·h⁻¹ (39 g/h) in addition to a 0.44 g/kg bolus (31), and 0.30 g·kg⁻¹·h⁻¹ (35 g/h) plus an additional 0.55 g/kg in the bolus (10). Women in the present study oxidized significantly more CHO with the ingestion of ~29 g CHO/h when compared with a placebo, suggesting that this dosage was adequate to increase CHO utilization.

The optimal dose eliciting performance improvements when CHO feedings are administered throughout prolonged exercise has not been established. Hargreaves (15) has suggested there is little added benefit in ingesting CHO solutions more concentrated than 6–8% because consuming higher dosages does not increase the rate of exogenous glucose oxidation. Evidently, there is a plateau in exogenous CHO oxidation during prolonged exercise whereby additional CHO ingestion above a certain amount will have no effect. On the basis of findings from Mitchell et al. (25), ingesting a CHO beverage that is too concentrated (i.e., 18% CHO) can impair gastric emptying and cause discomfort. Similarly, ingesting a less concentrated beverage in a greater volume of fluid also causes discomfort to the subjects. The difficulty is to administer a beverage that will provide an adequate amount of CHO to the subject without causing GI discomfort. Subjects in the present study experienced GI distress late in exercise during all three experimental trials, indicating that fluid volume rather than CHO concentration caused the discomfort. Chryssanthopoulos et al. (11) suggested that one cause of GI distress is that the volume of fluid prescribed and consumed by runners in laboratory studies is greater than the volume consumed during competition. Therefore, it is possible that subjects in the present study were experiencing GI distress because they were consuming more beverage during the experimental trials than they would during training and/or competition.

In summary, the aim of the present study was to gain a better understanding and provide more information on the response of women runners to CHO augmentation. On the basis of the findings, it appears that when CHO availability was increased through CHO loading and/or CHO supplementation there was a concomitant increase in CHO utilization. However, this did not translate into significantly improved performance.

DISCLOSURES

This investigation was supported by a grant from the Gatorade Sport Science Institute.

REFERENCES


