Carbohydrate Supplementation of Horses During Endurance Exercise: Comparison of Fructose and Glucose

Sharon R. Bullimore* Joe D. Pagan*,2 Patricia A. Harris† Kari E. Hoekstra* Kathleen A. Roose* Sonja C. Gardner* and Ray J. Geor*

*Kentucky Equine Research, Inc., Versailles, Kentucky 40383 and † Equine Studies Group, WALTHAM Centre for Pet Nutrition, Waltham-on-the-Wolds, Leicestershire, LE14 5RT, UK.

ABSTRACT To delay the onset of fatigue, endurance horses are often fed at rest stops during races. The resulting increase in blood insulin may adversely inhibit lipolysis. In humans, ingestion of fructose produces a smaller insulin rise than glucose. This study compared glucose and fructose as carbohydrate supplements for endurance horses. Three Arabian geldings were given 300 g of fructose (F), glucose (G) or 50% glucose: 50% fructose (GF), in 1.5 L water, by stomach tube. In the Resting Test, carbohydrate was administered at rest. Following treatment, blood samples were taken every 30 min for 8 h, and feces were collected for 24 h. Treatment did not affect fecal weight or water content. Plasma glucose and insulin responses did not differ among treatments. Post-treatment (60 min), plasma L-lactate tended to be higher (P = 0.06) after the F and GF treatments than after the G treatment. In the Exercise Test, two treadmill exercise bouts at 0° incline (Bout 1: 90 min; Bout 2: 120 min) were separated by a 1-h rest period. A total distance of 36.84 km was covered at a mean speed of 2.9 m/s. Carbohydrate was administered 45 min before Bout 2. Plasma glucose and insulin at the start of Bout 2 were higher (P = 0.02 and 0.03, respectively) with the GF treatment than with the F treatment. However, during exercise, plasma glucose concentrations did not differ among treatments. We conclude that fructose is well-absorbed by horses and rapidly converted to glucose. J. Nutr. 130: 1760–1765, 2000.

KEY WORDS: • horse • carbohydrate • fructose • glucose • endurance exercise

Carbohydrate availability is believed to be a performance-limiting factor for horses during prolonged moderate intensity exercise. Hypoglycemia and depletion of liver glycogen have been described in horses after completion of 72 km of low-intensity exercise (Lindholm et al. 1974). The slow twitch fibers of the middle gluteal muscle (7 to 38% of the total fiber population) may become totally glycogen-depleted during an 80-km endurance ride (Snow et al. 1981). Furthermore, an intravenous glucose infusion has been shown to delay the development of fatigue in horses during submaximal treadmill exercise (Farris et al. 1995). To increase carbohydrate availability, competitive endurance horses are often fed at rest stops during races. These meals are usually high in starch (grain) and, when hydrolyzed in the small intestine, such feeds provide a large amount of glucose. In horses fed grain, marked increases in plasma glucose and insulin concentrations occur within 1 h of ingestion (Pagan and Harris 1999). When ~1 kg corn is given 1 h before exercise, there is a rapid decrease in plasma glucose at the onset of exercise, and free fatty acid concentrations are suppressed compared to unfed horses (Lawrence et al. 1995, Stull and Rodiek 1995). Moreover, consumption of grain 1 h before exercise has been shown to accelerate glycogen utilization (Lawrence et al. 1995).

Fructose, an isomer of glucose, may be a useful alternative source of supplemental carbohydrate. In resting humans, the increases in blood glucose and insulin after fructose ingestion are only 10 to 20% of those measured after an equivalent amount of glucose (Crapo et al. 1980). Ingestion of fructose 30 to 60 min before exercise results in a lower insulin peak and less marked fluctuations in blood glucose concentrations during exercise, in comparison to glucose (Guezennec et al. 1989, Ventura et al. 1994).

In humans, a potential drawback with the use of fructose, as a carbohydrate supplement, is the risk of development of gastrointestinal distress. Abdominal discomfort and diarrhea are frequently experienced when ~1 g/kg body weight fructose is ingested before or during exercise (Erickson et al. 1987, Murray et al. 1989). These effects are caused by the limited capacity for fructose absorption (Ravich et al. 1983). Fructose that is not absorbed in the small intestine passes into the large intestine and undergoes fermentation. The products of fermentation result in excess gas formation and development of osmotic diarrhea. In humans, co-ingestion of glucose with fructose facilitates fructose absorption (Shi et al. 1997).Therefore, compared to fructose alone, a mixture of fructose and glucose may be preferable as a carbohydrate supplement for human athletes.

Anecdotally, honey is sometimes fed to endurance horses

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2 To whom correspondence should be addressed.
before and during competitions. Honey contains, on average, 38% fructose and 31% glucose (Berlitz and Grosch 1987). Horses can consume 1 g/kg body weight of a 50% glucose/50% fructose mixture, as a 200 g/L solution, without adverse effects (Roberts 1975). However, to the authors’ knowledge, there has been no research into the effects of feeding fructose without glucose to the exercising horse. The high incidence of gastrointestinal disturbances in humans following ingestion of fructose raises concern of similar problems in horses after consumption of meals high in fructose. In horses, fermentation of soluble carbohydrate in the large intestine can result in lactic acidosis and diarrhea (Garner et al. 1977, Roberts 1975).

The overall aim of the present study was to evaluate the suitability of fructose as a carbohydrate supplement for horses. Specifically, we determined the glycemic response following ingestion of fructose, glucose and a 50% glucose/50% fructose mixture, both at rest and during an endurance exercise protocol.

MATERIALS AND METHODS

The procedures used were in accordance with the Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching (1988). Three Arabian geldings (ages: 7, 9 and 10 y; bodyweights: 395, 442 and 478 kg) were used. Horses were housed in 3 × 3 m box stalls during the night and at pasture during the day (0830 to 1600 h). While at pasture, horses were muzzled to prevent grazing. Horses had access to water at all times and to a salt block while housed in stalls. Horses were exercised between 0830 and 1600 h. For the 5-mo period before the study, the horses were exercised 3 d/wk on a treadmill at 0° incline (Stratton Equine Enterprises Inc., Lexington, KY). On all other days, the horses were walked for 1 h on a mechanical exerciser. Treadmill exercise was gradually increased in intensity until the horses could complete 1 h of trotting at 3.6 m/s without difficulty.

The horses consumed a constant basal diet throughout the study. Daily, each horse was fed 6.8 kg mixed grass/alfalfa hay (divided and fed at 0700, 1600 and 2200 h) [As fed (g/kg): 153 crude protein, 346 acid detergent fiber, 511 neutral detergent fiber, 26 crude fat, 149 nonstructural carbohydrate, 9.3 calcium, 3.4 phosphorus, 1.7 magnesium, 22.9 potassium, 0.09 sodium, 0.127 iron, 0.022 zinc, 0.008 copper, 0.063 manganese, 0.001 molybdenum.] and 2 kg of an unfortified grain mix (450 g/kg whole oats, 430 g/kg cracked corn, 120 g/kg molasses), divided into two feedings (0700 and 1600 h). [As fed (g/kg): 91 crude protein, 63 acid detergent fiber, 141 neutral detergent fiber, 46 crude fat, 585 nonstructural carbohydrate, 1.4 calcium, 3.2 phosphorus, 1.5 magnesium, 7.9 potassium, 0.58 sodium, 0.103 iron, 0.024 zinc, 0.024 manganese, 0.0019 molybdenum.]. In addition, each horse was fed 84 g of a pelleted mineral and vitamin supplement (Microphase; Kentucky Equine Research, Inc., Lexington, KY) [As fed (g/kg): 133 protein, 27 calcium, 13 phosphorus, 0.88 copper, 2.5 zinc, 0.009 selenium, 0.21 vitamin A, 0.0009 vitamin D, 2.2 vitamin E, 0.21 thiamin, 0.58 choline, 0.11 folic acid, 1.06 niacin, 0.44 pantothenic acid, 0.35 riboflavin, 0.001 vitamin B-12].

Experimental protocol. Three treatments were used: d-fructose (F), d-glucose (G) and 50% fructose/50% glucose (GF). The study was divided into three periods of 10 d each, with no interval between periods. In period 1, one horse was allocated to each treatment. In periods 2 and 3, the treatments were rotated between horses using a replicated 3 × 3 Latin square design. Fig. 1 summarizes the timetable for each period. On d 1 of each period, no treatment was given. On d 2–6, the assigned treatment was administered in the grain fed at 0700 h. The amount of carbohydrate used was gradually increased (25 g on d 2, then 50 g, 100 g, 200 g and 300 g on d 3–6, respectively). On d 4 and 5 an incremental exercise test (HR3.6, Test) was performed for determination of heart rate at a running speed of 3.6 m/s (see below). On d 7, the Resting Test was conducted (see below). On d 8, no treatment was given. On d 9 and 10, the Exercise Test was conducted (see below). On days when the horses were not tested, they were walked for 1 h on a mechanical exerciser. Three tests were used.

(i) HR3.6 Test: This protocol was used to monitor fitness over the course of the study. The horses were exercised on a treadmill at 0° incline as follows: 5 min at 1.3 m/s, 8 min at 3.1 m/s, followed by 3-min intervals at 2.7, 3.1, 3.6, 4.5 and 5.4 m/s. HR was recorded during the last 10 s of each 3-min speed step, allowing HR at 3.6 m/s (HR3.6) to be calculated by linear regression.

(ii) Resting test: On the day before the resting test, the horses were fed 3.4 kg hay and 84 g vitamin and mineral supplement at 2200 h. No feed was provided the following morning. At 1000 h, 300 g of the assigned treatment (dissolved in 1.5 L water; 200 g/L) was administered by stomach tube. Before treatment, two venous blood samples were collected from the jugular vein via a catheter [Abocath, 14 g, 13 cm; Abbott Laboratories, Abbott Park, IL]. Following treatment, samples were collected at 30-min intervals over an 8-h period. The plasma was harvested and stored at −18°C. Throughout the study, the water was provided in 18-L buckets. Before and 24 h after treatment, the water buckets were weighed to the nearest 1 g (Ohaus I-10; Ohaus Corporation, Florham Park, NJ) for determination of water consumption. Feces were collected using a commercially available equine harness (Equisan PTY Ltd., Melbourne, Australia). The harness was emptied at 7, 15 and 24 h post-treatment and the feces were weighed. For each collection period, a 400-g sample of feces was taken and frozen at −18°C.

(iii) Exercise test: In each period, horse 1 was tested on the morning of d 9, horse 2 on the afternoon of d 9 and horse 3 on the morning of d 10. Grain was withheld for at least 15 h before each exercise test. A normal allotment of hay was fed 5 h before the protocol. Horses completed two bouts of exercise with the treadmill set at 0° incline. The total distance was 36,840 m, at a mean speed of 2.9 m/s. The first exercise period (Bout 1) comprised a 5-min walk (1.3 m/s), 60-min trot (3.6 m/s) and 5-min walk (1.3 m/s) on the treadmill, followed by a 20-min walk on a mechanical exerciser (1.2 m/s). Bout 2 comprised a 5-min walk (1.3 m/s), 90-min trot (3.6 m/s) and 5-min walk (1.3 m/s) on the treadmill, followed by a 20-min walk on the exerciser (~1.2 m/s). Water was offered when the horses were transferred from the treadmill to the exerciser. Between exercise bouts, horses were returned to their stalls and allowed access to water. After completion of Bout 1 (~10 min), 300 g of the assigned treatment (dissolved in 1.5 L water) was administered by stomach tube. Bout 2 commenced 45 min after treatment. Venous blood samples were collected from the jugular vein via a catheter. Samples were obtained: before Bout 1 (Pre-ex 1), after 30 min and 60 min of the first trot (Trot 1), before carbohydrate treatment, 30 min after treatment, before Bout 2 (Pre-ex 2), after 15, 30, 45, 60, 75 and 90 min of the second trot (Trot 2), and 30 min after completion of Trot 2 (Post 30). Plasma was harvested and stored at −18°C. HR was recorded at 15-s intervals from 20 min before exercise until 5 min after Bout 2, using a HR monitor (Vantage XL; Polar, Woodbury, NY). Water consumption was measured from the end of Trot 1 until 1 h after Trot 2. Four analyses were performed.

(ii) Fecal analysis: To determine fecal water content, three samples of ~50 g were taken from each 400-g sample. These samples were...
weighed on an analytical balance (Ohaus Corporation, Florham Park, NJ) while still frozen, dried overnight (12 h) at ~100°C and then weighed at 1-h intervals until a constant weight (to the nearest 0.1 g) was achieved. Percentage water content was calculated and the results for the three samples averaged.

(ii) Plasma analysis: Plasma concentrations of l-lactate and glucose were measured by use of an automated analyzer (2300 STAT; YSI, Yellow Springs, OH). All samples were analyzed in duplicate; the reported values represent the average of the two measurements. Total protein concentration was determined by refractometry (Rood and Riddle Equine Hospital, Lexington, KY). All samples from the Exercise Test, except those obtained at 15, 30, 60 and 75 min of Trot 2, were analyzed for total plasma protein. From the data for total plasma protein, the percentage change in plasma volume was calculated using the method of van Beaumont et al. (1972).

Plasma insulin concentration was determined by radioimmunoassay (BET Labs, Lexington, KY). In the Resting Test, plasma insulin was measured in samples collected before treatment and in samples that correspond to peak plasma glucose and 30 min after peak glucose. The highest insulin concentration in these latter two samples was taken as peak insulin concentration. In the Exercise Test, insulin was determined in samples collected before and 45 min after carbohydrate administration (Pre-ex 2). Free fatty acid concentration was determined in samples obtained at Pre-ex 1, 60 min of Trot 1, Pretreatment, Pre-ex 2, 45 and 90 min of Trot 2 and Post 30, by use of an enzymatic colorimetric assay (NEFA C Kit, Wako Chemicals, Richmond, VA).

(iii) HR analysis: The effect of study period on HR and HR3.6 was used to detect changes in fitness during the study. Linear regression analysis was used to detect changes in HR over each bout of trotting (excluding the first 20 min). A significant, positive regression was taken to indicate cardiovascular drift.

(iv) Calculations and statistical analysis: The plasma glucose and lactate data from the Resting Test were used to calculate a number of parameters: (i) the area enclosed between the glucose or lactate vs. time curve and the time axis (AUC2); (ii) peak plasma glucose or lactate concentration during the 8-h measurement period; and (iii) time elapsed between carbohydrate administration and the occurrence of the peak glucose and lactate concentrations. The average of the two samples obtained before treatment was taken as the baseline value.

Treatment effects were detected using the general linear model for ANOVA (Minitab version 12.22; Minitab Inc., State College, PA), with horse, period and treatment as model terms and horse as a random factor. When the F ratio indicated a significant treatment effect, planned pair-wise comparisons for treatment were made using Tukey’s procedure. Homogeneity of variance was tested separately with each factor using Bartlett’s test. Where variances differed significantly, natural logs were taken before analyzing the results as above. Where a skewed distribution was suspected, because of a large standard deviation in relation to the mean, ranks were taken before analyzing the results as above. All results are given as mean ± SEM. The null hypothesis was rejected at α = 0.05. In the Exercise Test, the pretreatment blood sample for horse 3 in the fructose treatment was not obtained and is treated as a missing value throughout.

RESULTS

Resting test. The treatments were delivered without complications over an ~1-min period. None of the horses showed signs of gastrointestinal discomfort (colic) or diarrhea.

Fecal weight and water content (overall mean: 76.92 ± 0.46%) were not affected by treatment and were not different for the three fecal collection periods (0 to 7 h post-treatment, 7 to 15 h post-treatment and 15 to 24 h post-treatment).

Water intake between 0700 and 1700 h was not significantly affected by treatment (F: 9.91 ± 1.35; G: 9.18 ± 0.31; GF: 8.91 ± 0.66 kg). However, water intake between 1700 and 0700 h the next day was greater (F = 0.01) after the F treatment (11.42 ± 1.27 kg) than after the G (9.03 ± 1.48 kg) and GF (8.01 ± 0.57 kg) treatments.

With the exception of 300-min post-treatment, when plasma glucose was lower after the G treatment than after the F or GF treatments (P = 0.01), the magnitude and time course of the plasma glucose response did not differ among treatments (Fig. 2A). Treatment had no effect on peak glucose concentration, time of peak glucose concentration or AUC (F: 180.5 ± 26.3; G: 162.4 ± 44.9; GF: 179.6 ± 26.0 mmol · min/L).

After carbohydrate administration (60 min), there was a...
exercise. However, plasma glucose subsequently increased and, in each treatment, mean concentrations were relatively stable at ~4.5 to 5.0 mmol/L for the remainder of exercise. During exercise, there were no differences between treatments for plasma glucose concentration.

The increase in plasma glucose from pre-treatment to 30 min post-treatment was greater (P < 0.001) in the Exercise Test (F: 1.14 ± 0.03; G: 1.60 ± 0.05; GF: 1.99 ± 0.22 mmol/L) than in the Resting Test (F: 0.74 ± 0.12; G: 0.82 ± 0.25; GF: 1.08 ± 0.34 mmol/L).

Plasma lactate concentration was below baseline during both exercise bouts, but increased during the interval between exercise bouts (Fig. 4B). There was a nonsignificant trend (P = 0.1) for plasma lactate to be higher after the GF treatments than after the G treatments at Pre-ex 2 and at 60 min of Trot 2. At 30-min post-exercise, plasma lactate was higher (P = 0.02) after the F and GF treatments, than after the G treatments.

Plasma free fatty acid concentration decreased (P = 0.004) following carbohydrate administration and increased (P < 0.001) with exercise (Fig. 4C). At 45 min of Trot 2, there was a nonsignificant trend (P = 0.1) for a lower free fatty acid concentration after the GF treatments, compared to the F treatments.

Plasma insulin concentration increased (P < 0.001) following carbohydrate administration. At Pre-ex 2, plasma insulin concentration was higher (P = 0.03) after the GF treatments than after the F treatments (Fig. 4D). There was also a nonsignificant trend (P = 0.06) for higher insulin after the GF treatments, compared to the F treatments, at Pre-ex 2.

**DISCUSSION**

**Resting test.** Oral administration of 300 g fructose in 1.5 L water (200 g/L) appears to be safe in horses, as indicated by the absence of adverse effects or alterations in fecal weight or water content. The plasma glucose concentration vs. time curves were similar for all treatments. These results suggest that fructose is well-absorbed by horses and is rapidly converted to glucose. In agreement with the current study, Roberts (1975) found that the glycemic response to a 50% glucose/50% fructose mixture was very similar to the response to an equivalent dose of glucose. In contrast, fructose administration (50 g) in humans produces only 10 to 20% of the increase in plasma glucose concentration observed after an equivalent glucose load (Crapo et al. 1980). This difference suggests that fructose is better absorbed in horses than in humans, or that horses convert a greater proportion of absorbed fructose to glucose.

Water consumption between 10 and 24 h after carbohydrate treatment was significantly greater after the F treatments than after the G or GF treatments. The mechanism for this increase in water consumption is not readily apparent. Fructose ingestion did not increase fecal water loss. It is possible that the osmotic effects of unabsorbed fructose could draw water into the intestine, causing a compensatory increase in water consumption. However, it is unlikely that this would occur more than 10 h after fructose administration.

There was a nonsignificant trend for higher peak lactate concentration after the F and GF treatments, compared with the G after treatments (Fig. 4B). There are three possible reasons for the increase in plasma lactate concentration. Fructose may be converted to lactate in the liver and kidney, as has been described in humans (Atwell and Waterhouse 1971; Bjorkman and Felig 1982). Lactate-producing bacteria in the stomach and small intestine (Kern et al. 1974) may metabolize

**FIGURE 3** Mean heart rate (HR) response in three Arabian horses undergoing an exercise test three times each. Two bouts of trotting at 3.6 m/s (Trot 1: 60 min; Trot 2: 90 min) were separated by a rest period; 300 g fructose, glucose or 50% glucose/50% fructose in 1.5 L water was delivered by stomach tube 45 min prior to Trot 2. HR was recorded every 15 s from 25 min before Trot 1 to 30 min after Trot 2. Significant cardiovascular drift occurred during both periods of trotting.

**TABLE 2** Variations in plasma glucose concentration during the pre- and post-exercise period.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Pre-ex 1</th>
<th>Pre-ex 2</th>
<th>Post-ex 1</th>
<th>Post-ex 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>6.0 ± 0.3</td>
<td>4.5 ± 0.2</td>
<td>7.0 ± 0.4</td>
<td>8.8 ± 0.3</td>
</tr>
<tr>
<td>G</td>
<td>5.5 ± 0.3</td>
<td>4.2 ± 0.2</td>
<td>6.5 ± 0.3</td>
<td>8.2 ± 0.3</td>
</tr>
<tr>
<td>GF</td>
<td>7.2 ± 0.4</td>
<td>5.7 ± 0.3</td>
<td>8.0 ± 0.4</td>
<td>10.0 ± 0.5</td>
</tr>
</tbody>
</table>

**TABLE 3** Variations in plasma free fatty acid concentration during the pre- and post-exercise period.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Pre-ex 1</th>
<th>Pre-ex 2</th>
<th>Post-ex 1</th>
<th>Post-ex 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>0.3 ± 0.1</td>
<td>0.2 ± 0.1</td>
<td>0.4 ± 0.2</td>
<td>0.5 ± 0.3</td>
</tr>
<tr>
<td>G</td>
<td>0.2 ± 0.1</td>
<td>0.1 ± 0.1</td>
<td>0.3 ± 0.2</td>
<td>0.4 ± 0.3</td>
</tr>
<tr>
<td>GF</td>
<td>0.5 ± 0.1</td>
<td>0.3 ± 0.2</td>
<td>0.6 ± 0.3</td>
<td>0.7 ± 0.3</td>
</tr>
</tbody>
</table>

**TABLE 4** Variations in plasma insulin concentration during the pre- and post-exercise period.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Pre-ex 1</th>
<th>Pre-ex 2</th>
<th>Post-ex 1</th>
<th>Post-ex 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>8.0 ± 1.0</td>
<td>6.5 ± 1.0</td>
<td>9.0 ± 1.2</td>
<td>11.0 ± 1.3</td>
</tr>
<tr>
<td>G</td>
<td>7.5 ± 0.9</td>
<td>6.0 ± 0.8</td>
<td>9.5 ± 1.0</td>
<td>11.5 ± 1.1</td>
</tr>
<tr>
<td>GF</td>
<td>9.0 ± 1.1</td>
<td>7.5 ± 0.9</td>
<td>10.0 ± 1.2</td>
<td>12.0 ± 1.3</td>
</tr>
</tbody>
</table>
fructose. Lactate-producing bacteria in the large intestine also may contribute, as nutrients delivered by stomach tube can reach the cecum in as little as 60 min (Cuddeford 1999). In the bovine rumen, which has a similar microbial population to the equine cecum (Kern et al. 1974), both D- and L-lactate are produced by the fermentation of soluble carbohydrate (Slyter and Rumsey 1991). Because only L-lactate was measured in the current study, the amount of fructose that underwent microbial fermentation may have been underestimated. However, the similarity of the glycemic responses to each treatment suggests that only a small proportion of ingested fructose was converted to lactate. Further investigation is needed to determine whether ingested fructose is converted to lactate by horses and, if so, by what mechanism.

Exercise test. Plasma insulin concentration at Pre-ex 2 was lower when horses were given fructose, compared to a 50:50 mixture of fructose and glucose. There was also a non-significant trend for a lower insulin concentration following fructose administration, compared to glucose. In humans, commencement of exercise with elevated plasma insulin inhibits lipolysis and fat oxidation and increases reliance on carbohydrate oxidation (Costill et al. 1977). This is disadvantageous because it increases the rate at which carbohydrate stores are depleted. While the supply of fat in the body is almost unlimited, the supply of carbohydrate is not. Depletion of muscle glycogen stores may cause fatigue during endurance exercise. However, despite the lower pre-exercise insulin concentration after fructose administration, free fatty acid concentrations during exercise were similar for all treatment groups. Further research is needed to determine whether the lower plasma insulin concentrations in horses after fructose administration would influence substrate utilization during exercise.

Plasma free fatty acid concentration decreased following carbohydrate administration. However, it recovered rapidly at the onset of exercise and continued to increase throughout exercise. These results suggest that 300 g glucose or fructose could be administered to horses at rest stops during endurance rides, without adversely inhibiting lipolysis.

Pre-exercise elevation in plasma insulin following carbohydrate consumption can result in a sharp decrease in plasma glucose concentration early during exercise. This response has been attributed to an insulin-mediated increase in the rate of glucose uptake by the peripheral tissues (Horowitz et al. 1997). However, despite the lower plasma insulin concentration following fructose administration, plasma glucose concentrations during the subsequent bout of exercise were similar for all treatments (Fig. 4A). In humans, pre-exercise (1 h before) fructose ingestion also results in a smaller increase in plasma insulin concentration compared to an equivalent dose of glucose. Furthermore, the increase in whole-body glucose disposal and decrease in plasma glucose concentration during the first 20 min of low-intensity exercise are attenuated after fructose ingestion compared to glucose ingestion (Horowitz et al. 1997). Further study using isotopic tracer methods is required to determine the effects of these two carbohydrate treatments on glucose flux in horses during exercise.

Post treatment (30 min), plasma glucose concentration was significantly higher after the GF treatments, than after the G and G different; b = F and GF different; c = G and GF different. Blood samples were taken at: 1) pre-ex 1; 2) 30 min Trot 1; 3) 60 min Trot 1; 4) pre-treatment; 5) 30 min post-treatment; 6) pre-ex 2; 7) 15 min Trot 2; 8) 30 min Trot 2; 9) 45 min Trot 2; 10) 60 min Trot 2; 11) 75 min Trot 2; 12) 90 min Trot 2; 13) 30 min post-exercise.

FIGURE 4 Plasma glucose (A), L-lactate (B), free fatty acid (C) and insulin (D) concentrations in three Arabian horses undergoing an exercise test. Values are means ± SEM. n = 3. Two bouts of trotting at 3.6 m/s (Trot 1: 60 min; Trot 2: 90 min) were separated by a rest period; 300 g fructose (F), glucose (G) or 50% glucose/50% fructose (GF) in 1.5 L water was delivered by stomach tube, 50 min prior to Trot 2. Letters indicate significant differences between treatments (P < 0.05): a = F and G different; b = F and GF different; c = G and GF different. Blood samples were taken at: 1) pre-ex 1; 2) 30 min Trot 1; 3) 60 min Trot 1; 4) pre-treatment; 5) 30 min post-treatment; 6) pre-ex 2; 7) 15 min Trot 2; 8) 30 min Trot 2; 9) 45 min Trot 2; 10) 60 min Trot 2; 11) 75 min Trot 2; 12) 90 min Trot 2; 13) 30 min post-exercise.
treatments. It is not possible to determine the reason for this from the current study.

It is important to note that the exercise test used in the current study involved only low-intensity exercise. A different response may have been observed for higher intensity exercise. In addition, the use of a small sample size limits the statistical power of the study, so that some treatment effects may not have been detected. For this reason, the authors recommend that further work, utilizing a larger sample size, would be beneficial.

In resting horses, administration of fructose, or a 50% glucose/50% fructose mixture, results in a glycemic response similar to that measured after an equivalent glucose load. These findings indicate that fructose is well-absorbed and rapidly converted to glucose. Furthermore, fructose appears to be well-tolerated by horses, at least at the dose used in this study (~0.7 g/kg body weight). Pre-exercise plasma insulin concentration was lower following fructose administration, than in the other two treatments. The authors suggest fructose may be useful as a carbohydrate supplement for horses before and/or during endurance exercise. However, further research with a larger sample size and use of isotopic tracer methods is needed to assess the effect of fructose supplementation on exercise metabolism and performance.

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LITERATURE CITED


