

# Superior Endurance Performance with Ingestion of Multiple Transportable Carbohydrates

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## ABSTRACT

CURRELL, K., and A. E. JEUKENDRUP. Superior Endurance Performance with Ingestion of Multiple Transportable Carbohydrates. *Med. Sci. Sports Exerc.*, Vol. 40, No. 2, pp. 275–281, 2008. **Introduction:** The aim of the present study was to investigate the effect of ingesting a glucose plus fructose drink compared with a glucose-only drink (both delivering carbohydrate at a rate of  $1.8 \text{ g}\cdot\text{min}^{-1}$ ) and a water placebo on endurance performance. **Methods:** Eight male trained cyclists were recruited (age  $32 \pm 7$  yr, weight  $84.4 \pm 6.9$  kg,  $\dot{V}O_{2\text{max}}$   $64.7 \pm 3.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ,  $W_{\text{max}}$   $364 \pm 31$  W). Subjects ingested either a water placebo (*P*), a glucose (*G*)-only beverage ( $1.8 \text{ g}\cdot\text{min}^{-1}$ ), or a glucose and fructose (GF) beverage in a 2:1 ratio ( $1.8 \text{ g}\cdot\text{min}^{-1}$ ) during 120 min of cycling exercise at 55%  $W_{\text{max}}$  followed by a time trial in which subjects had to complete a set amount of work as quickly as possible (~1 h). Every 15 min, expired gases were analyzed and blood samples were collected. **Results:** Ingestion of GF resulted in an 8% quicker time to completion during the time trial (4022 s) compared with *G* (3641 s) and a 19% improvement compared with *W* (3367 s). Total carbohydrate (CHO) oxidation was not different between GF ( $2.54 \pm 0.25 \text{ g}\cdot\text{min}^{-1}$ ) and *G* ( $2.50 \text{ g}\cdot\text{min}^{-1}$ ), suggesting that GF led to a sparing of endogenous CHO stores, because GF has been shown to have a greater exogenous CHO oxidation than *G*. **Conclusion:** Ingestion of GF led to an 8% improvement in cycling time-trial performance compared with ingestion of *G*. **Key Words:** GLUCOSE, FRUCTOSE, ERGOGENIC AID, CYCLING, EXOGENOUS CARBOHYDRATE, TIME TRIAL

It was first suggested in the 1920s that ingestion of carbohydrate during exercise can lead to an improvement in endurance performance (25,26). The finding that carbohydrate intake during exercise results in an improvement in performance has since become a regular finding and has become generally accepted (20). Carbohydrate beverages have become a booming industry, with the U.S. market alone worth \$1.2 billion per year (4), and the use of carbohydrate drinks is now common practice among athletes.

For some time, it was thought that exogenous carbohydrate oxidation rates would not exceed  $1 \text{ g}\cdot\text{min}^{-1}$ , even at high rates of ingestion ( $>2 \text{ g}\cdot\text{min}^{-1}$ ) (21). However, recent research from our laboratory has shown that exogenous carbohydrate oxidation can be increased above  $1 \text{ g}\cdot\text{min}^{-1}$  when multiple transportable carbohydrates are ingested. In

the first of a series of studies, Jentjens et al. (14) examined exogenous carbohydrate oxidation during 120 min of cycling exercise at 50% maximum power ( $W_{\text{max}}$ ). Subjects were fed either  $1.2 \text{ g}\cdot\text{min}^{-1}$  glucose,  $1.8 \text{ g}\cdot\text{min}^{-1}$  glucose, or  $1.2 \text{ g}\cdot\text{min}^{-1}$  glucose +  $0.6 \text{ g}\cdot\text{min}^{-1}$  fructose. Using stable and radioisotope methodology, total exogenous carbohydrate oxidation of the mixture of glucose and fructose was reported to be as high as  $1.26 \text{ g}\cdot\text{min}^{-1}$ , whereas for both glucose drinks the exogenous carbohydrate oxidation was around  $0.80 \text{ g}\cdot\text{min}^{-1}$ . In subsequent studies, it was observed that when a glucose and fructose beverage in a 1:1 ratio was ingested at very high rates ( $2.4 \text{ g}\cdot\text{min}^{-1}$ ) during 150 min of exercise at 50%  $W_{\text{max}}$ , peak exogenous carbohydrate oxidation rate could even amount up to  $1.75 \text{ g}\cdot\text{min}^{-1}$  (13). This finding of increased exogenous carbohydrate oxidation has also been reported when the fructose has been replaced by sucrose (12,15,17), the glucose replaced by maltodextrin (36), and it has also been seen during exercise in the heat (16), where exogenous carbohydrate oxidation is usually suppressed (18).

As recently reviewed (20), it was thought that the limitation of the rate of exogenous carbohydrate oxidation to  $1 \text{ g}\cdot\text{min}^{-1}$  was not in gastric emptying or muscle glucose uptake. Rather, it seemed to be intestinal absorption of carbohydrate that limited exogenous carbohydrate oxidation (21). Glucose is absorbed in the intestine by the sodium-dependent glucose transporter SGLT1 (8). It has been suggested that the SGLT1 transporter becomes saturated at

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high glucose-ingestion rates. When glucose and fructose are combined, intestinal carbohydrate absorption can be increased, because fructose uses a different transporter whereas fructose is absorbed by the intestinal transporter GLUT 5 (33). This mechanism has been used as a way to explain the robust finding of increased exogenous carbohydrate oxidation with multiple transportable carbohydrates.

Although it is generally assumed that an increase in exogenous carbohydrate oxidation is beneficial because it reduces the reliance on limited endogenous stores, the ergogenic effects have not yet been demonstrated. During 5 h of exercise at 50%  $W_{max}$ , peak exogenous carbohydrate oxidation was shown to be greater with ingestion of a glucose and fructose beverage compared with glucose alone (22). In this study, indications for improved performance were reported towards the end of 5 h of exercise (22). When glucose and fructose were ingested, ratings of perceived exertion (RPE) were lower, and self-selected cadence was higher in the later stages of exercise compared with both glucose alone and water. These findings suggest a reduction in fatigue with the ingestion of a glucose and fructose beverage compared with a glucose-only drink. However, direct measurements of performance were not obtained.

Therefore, the aim of the present study was to investigate the effects of ingesting a glucose and fructose beverage compared with a glucose beverage (both delivering carbohydrate at high rates) and a water placebo on endurance cycling performance. The average ingestion rate of 1.8 g CHO·min<sup>-1</sup> was chosen because this had been used in a previous study by Jentjens et al. (14) and would ensure saturation of the SGLT1 transporter in both CHO trials. The experimental hypothesis was that ingestion of glucose and fructose in a 2:1 ratio would lead to an improvement in endurance performance when compared with glucose alone.

## METHODS

### Subjects

Eight male, trained cyclists gave their written informed consent to participate in the study, which was approved by the local ethics committee of the University of Birmingham. The subject characteristics are displayed in Table 1.

### Experimental Design

All exercise tests were carried out on an electrically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands). Subjects first undertook an incremental exercise test to exhaustion to determine maximum oxygen

uptake ( $\dot{V}O_{2max}$ ) and maximum power output ( $W_{max}$ ). This was then followed at least 1 wk later by three experimental trials, completed in a randomized order. Subjects either ingested a water placebo (*P*), a glucose-only beverage fed at a rate of 1.8 g·min<sup>-1</sup> (*G*), or a glucose and fructose (*GF*) beverage in a 2:1 ratio fed at a rate of 1.8 g·min<sup>-1</sup>. The carbohydrate concentration of the CHO beverages was 14.4%. After a 600-mL bolus was given before the beginning of exercise, 150 mL of the experimental drink was consumed every 15 min throughout the steady-state period and at 25, 50, and 75% of the time trial. Each trial was separated by at least 7 d and, at most, 14 d.

**Preliminary testing.** Subjects performed an incremental exercise test to volitional exhaustion at a self-selected cadence on a cycle ergometer. The appropriate seat position, handlebar height, and orientation were used during testing and replicated in each subsequent visit. The initial workload was 95 W, which was increased by 35 W every 3 min until volitional fatigue. Ventilation, oxygen uptake ( $\dot{V}O_2$ ), and carbon dioxide production ( $\dot{V}CO_2$ ) were recorded continuously (Oxycon Pro, Jaeger, Germany), as was heart rate (Polar Vantage NV, Polar Electro Oy, Finland). Maximum power output was calculated as the power output from the last completed stage plus the fraction of time spent in the next stage multiplied by 35 W. Maximal oxygen consumption ( $\dot{V}O_{2max}$ ) was decided by the attainment of two of the following criteria:

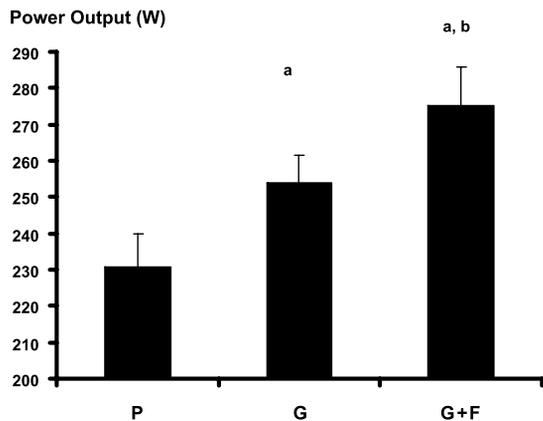
1.  $\dot{V}O_2$  did not increase with an increase in intensity (increase of no more than 0.2 L·min<sup>-1</sup>).
2. Heart rate was within 10 bpm of the age-predicted maximum of 220 – age.
3. Respiratory exchange ratio (RER) was greater than 1.05.

**Experimental visits.** Subjects entered the laboratory between 7:00 and 9:00 a.m. the following morning. Thereafter, a flexible 21-gauge catheter (BD Venflon, Helsingborg, Sweden) was inserted into an antecubital vein and attached to a three-way stopcock (Simms Portex, Kingsmead, UK), and a baseline blood sample was collected. The catheter was kept patent by flushing with 1.0–1.5 mL of isotonic saline (0.9%, Baxter, Norfolk, UK) after every blood sample. Thereafter subjects ingested a 600-mL bolus of the experimental beverage. Subjects then cycled for 2 h at an intensity of 55%  $W_{max}$ , with the cycle ergometer set in cadence-independent mode. Immediately on completion of the steady-state period, the cycle ergometer was set to linear mode (workload increases as the pedaling rate increases). Subjects were asked to perform a certain amount of work (equal to about 60 min of cycling at 75%  $W_{max}$ ) as fast as possible. The total amount of work was based on the maximal workload ( $W_{max}$ ) (19), and the total amount of work to be performed was calculated according to the formula:

$$\text{Total amount of work} = 0.75 W_{max} \times 3600 \text{ kJ} \quad [1]$$

TABLE 1. Subject characteristics.

	Age (yr)	Weight (kg)	$\dot{V}O_{2max}$ (mL·kg <sup>-1</sup> ·min <sup>-1</sup> )	$W_{max}$ (W)
Mean	32	84.4	64.7	364
SD	7	6.9	3.9	31

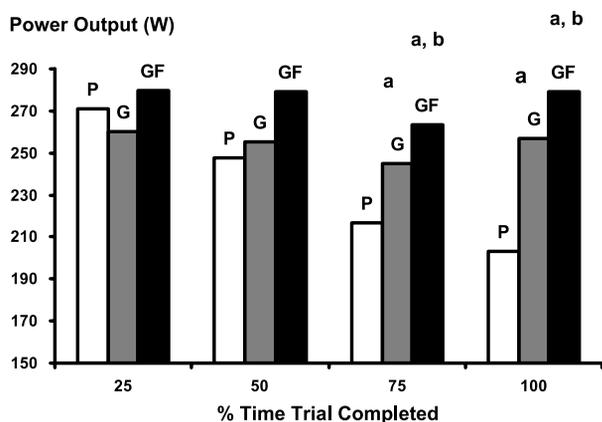


**FIGURE 1**—Power output during the time trial, where *P* is the water placebo trial, *G* is glucose fed at 1.8 g·min<sup>-1</sup>, and *G + F* is glucose fed at 1.2 g·min<sup>-1</sup> plus fructose fed at 0.6 g·min<sup>-1</sup>. *a*, significantly different from *P*; *b*, significantly different from *G*. Data are presented as means ± SE.

The ergometer was set in the linear mode according to the formula:

$$W = L(\text{rpm})^2 \quad [2]$$

in which the rpm is the pedaling rate, and *L* is a linear factor. This factor was chosen in a way that would evoke a pedaling rate of 90 rpm at 75% *W*<sub>max</sub>. The ergometer was connected to a computer, which recorded power output, cadence, and total work completed online. The only information the subject received was the amount of work performed, target work, and percentage of work completed relative to the target work. This information was presented on a computer screen in front of the subject. No verbal encouragement was given, no music was played, and no physiological measurements were taken throughout the time trial. Every effort was made to ensure that the subjects were not disturbed throughout the performance trials. No



**FIGURE 2**—Power output during each quarter of the time trial. *P*, water placebo trial; *G*, glucose fed at 1.8 g·min<sup>-1</sup>; *GF*, glucose at 1.2 g·min<sup>-1</sup> and fructose at 0.6 g·min<sup>-1</sup>. *a*, significantly different from *P*; *b*, significantly different from *G*. Data are presented as means ± SE.

**TABLE 2.** Oxygen uptake ( $\dot{V}O_2$ ), respiratory exchange ratio (RER), total carbohydrate oxidation (CHO<sub>tot</sub>), and total fat oxidation (FAT<sub>tot</sub>) during the steady-state period.

	TRIAL		
	<i>W</i>	<i>G</i>	<i>GF</i>
$\dot{V}O_2$ (L·min <sup>-1</sup> )	2.94 ± 0.18	2.91 ± 0.21	2.93 ± 0.21
RER	0.84 ± 0.01	0.91 ± 0.01 <sup>a</sup>	2.93 ± 0.21 <sup>a</sup>
CHO <sub>tot</sub> (g·min <sup>-1</sup> )	2.03 ± 0.24	2.03 ± 0.24 <sup>a</sup>	2.54 ± 0.25 <sup>a</sup>
FAT <sub>tot</sub> (g·min <sup>-1</sup> )	0.65 ± 0.07	0.44 ± 0.08 <sup>a</sup>	0.44 ± 0.08 <sup>a</sup>

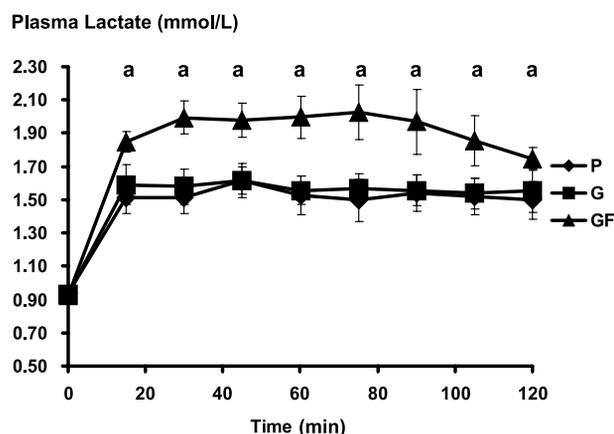
*W*, water placebo trial; *G*, glucose fed at 1.8 g·min<sup>-1</sup>; *GF*, glucose at 1.2 g·min<sup>-1</sup> and fructose at 0.6 g·min<sup>-1</sup>. Data are presented as means ± SE. <sup>a</sup> Significantly different from *W*.

performance results were shown to the subjects until the completion of the study.

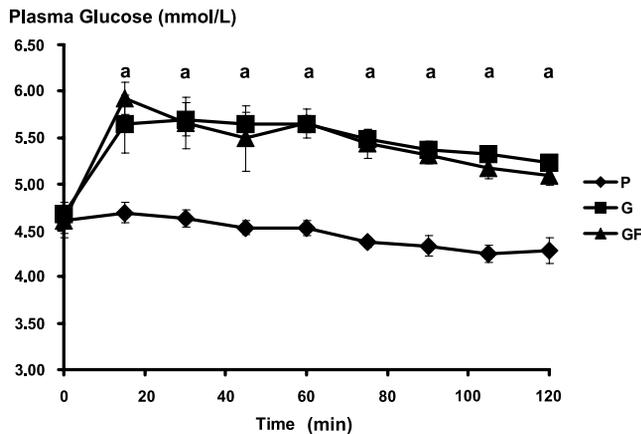
All exercise tests were performed under normal environmental conditions (20–23°C dry bulb temperature and 50–60% humidity). During the exercise trials, subjects were cooled with a floor-standing fan to minimize thermal stress.

**Blood sampling.** Blood samples (7 mL) were collected on entering the laboratory, at the start of the steady-state period, and every 15 min throughout the steady-state period. No samples were collected throughout the time trial. Blood samples were placed into prechilled EDTA-containing tubes (100 μL of 0.2 M EDTA) and centrifuged at 3200g for 10 min at 4°C. Aliquots of plasma were stored at -20°C. Blood plasma was analyzed for glucose (Glucose HK kit, Sigma Aldrich, Dorset, UK) and lactate (Lactate kit, Sigma Aldrich, Dorset, UK), using a semiautomated analyzer (COBAS BIO, Roche, Basel, Switzerland).

**Gas-exchange measurements.** Measures of  $\dot{V}O_2$  and carbon dioxide production ( $\dot{V}CO_2$ ) were collected for 5 min every 15 min throughout the steady-state period, using an automated online gas-analysis system (Oxycon Pro, Jaeger, Wuerzberg, Germany). Volume was calibrated by a 3-L syringe, and gas analyzers by a 5.03% CO<sub>2</sub>–94.97% N<sub>2</sub> gas mixture.



**FIGURE 3**—Plasma lactate concentrations during the 2-h steady-state period. Plasma lactate was significantly greater from 15 min after exercise in *GF* compared with both *P* and *G*. *P*, water placebo trial; *G*, glucose fed at 1.8 g·min<sup>-1</sup>; *GF*, glucose at 1.2 g·min<sup>-1</sup> and fructose at 0.6 g·min<sup>-1</sup>. *a*, *GF* significantly different from *G* and *P*. Data are presented as means ± SE.



**FIGURE 4**—Plasma glucose concentrations during the 2-h steady-state period. Plasma glucose was significantly greater from 15 min after exercise in GF and G compared with W. P, water placebo trial; G, glucose fed at 1.8 g·min<sup>-1</sup>; GF, glucose at 1.2 g·min<sup>-1</sup> and fructose at 0.6 g·min<sup>-1</sup>. a, G and GF significantly different from P. Data are presented as means ± SE.

From  $\dot{V}CO_2$  and  $\dot{V}O_2$  (L·min<sup>-1</sup>), total CHO and fat oxidation rates (g·min<sup>-1</sup>) were calculated, using the stoichiometric equations of Jeukendrup and Wallis (24):

$$\text{CHO oxidation} = 4.210 \dot{V}CO_2 - 2.962 \dot{V}O_2 \quad [3]$$

$$\text{Fat oxidation} = 1.695 \dot{V}O_2 - 1.701 \dot{V}CO_2 \quad [4]$$

All expired air data was averaged over the steady-state period.

**Heart rate and RPE.** Heart rate was measured via a radiotelemetry heart rate monitor (Polar Vantage NV, Kempele, Finland) continuously and later averaged for 5-min intervals. RPE was measured by the Borg 6–20 scale every 15 min during the steady-state period.

## Statistics

The mean power output from the time trials and expired air data from the three trials were analyzed using a two-way ANOVA for repeated measures. Blood glucose and lactate, heart rate, cadence, and RPE were analyzed using a two-factor (time and treatment) ANOVA with repeated measures. In case of a significance, *post hoc* analysis was undertaken with Tukey's HSD to locate the difference. Sphericity was upheld during statistical analysis. Data evaluation was performed using SPSS version 12.0.1 (Chicago, IL). Significance was set at the  $P < 0.05$  level. Data are presented as means ± SE.

## RESULTS

**Performance.** The mean power output during the time trial was  $231 \pm 9$  W for P,  $254 \pm 8$  W for G, and  $275 \pm 10$  W for GF. Ingestion of a GF at a rate of 1.8 g·min<sup>-1</sup> resulted in an 8% (95% CI 4.8–12.1%) higher power output in the time trial compared with G alone ( $P < 0.05$ ). GF also

resulted in a 19% (95% CI 14.1–25.2%) greater power output compared with P. G alone also improved performance compared with P by 10% (95% CI 6.7–13.8%) (Fig. 1). Ingestion of GF resulted in an 8% quicker time to completion during the time trial (4022 s) compared with G (3641 s) ( $P < 0.05$ ) and a 19% improvement compared with W (3367 s) ( $P < 0.05$ ).

Average power output between 25 and 50% of the target work during the time trial was significantly greater in GF than both G and P, as it was between 50 and 75% and between 75 and 100%. Ingestion of G led to a significantly greater power output than P between 50 and 75% and between 75 and 100% (Fig. 2).

**Gas-exchange measurements, and substrate metabolism.** The expired gas measurements are displayed in Table 2. There was no difference in the  $\dot{V}O_2$  between the three trials. Ingestion of G and GF led to an increase in RER compared with W ( $P < 0.05$ ), but no differences were seen between the two carbohydrate trials. Carbohydrate oxidation was significantly greater in both carbohydrate trials compared with water ( $P < 0.05$ ), and fat oxidation was concomitantly lower, but there was no difference between the carbohydrate trials.

**Plasma lactate and glucose.** Ingestion of GF led to significantly higher plasma lactate concentrations compared with both G and W after 15 min of exercise ( $P < 0.05$ ); G and W were not different from each other (Fig. 3). Ingestion of both carbohydrate drinks led to greater plasma glucose concentrations compared with water after 15 min of exercise ( $P < 0.05$ ) but were not different from each other (Fig. 4).

**Heart rate, cadence, and RPE.** There were no differences between any of the trials for heart rate, cadence, and RPE.

## DISCUSSION

The main finding from the present study is that ingesting a carbohydrate beverage containing both glucose and fructose in a 2:1 ratio ingested at a rate of 1.8 g·min<sup>-1</sup> resulted in an 8% improvement in performance compared with ingestion of glucose alone at the same rate. This improvement is on top of a 10% improvement in performance with glucose ingestion compared with water placebo.

Previous studies have demonstrated that the ingestion of a single carbohydrate source results in oxidation rates up to 1 g·min<sup>-1</sup> (20,21). Even ingestion of very large amounts of a carbohydrate ( $> 2$  g·min<sup>-1</sup>) did not further increase the exogenous carbohydrate oxidation rates (21). It is believed that the limitation is caused by intestinal absorption; as the intestinal sodium-dependent glucose transporters (SGLT1) become saturated, glucose absorption approaches its limit, and exogenous carbohydrate oxidation can not increase further. However, ingestion of multiple transportable carbohydrates (carbohydrates that use different intestinal carbohydrate transporters) can result in higher total exogenous

carbohydrate oxidation, well above  $1 \text{ g}\cdot\text{min}^{-1}$ . Jentjens et al. (14) have shown that during 2 h of cycling at 50%  $\dot{V}_{\text{max}}$ , identical beverages and rates of ingestion seen in the present study led to a 55% increase in exogenous carbohydrate oxidation with ingestion of a glucose and fructose beverage compared with glucose only. There was a trend toward a decrease in endogenous carbohydrate oxidation with ingestion of glucose and fructose. Similar results were obtained for glucose and fructose, maltodextrin and fructose, and a mixture of glucose, fructose, and sucrose (12–17,36). The increased delivery and oxidation of the ingested carbohydrate is generally assumed to be beneficial because it reduces the reliance on limited endogenous stores. However, direct evidence for this was lacking. The only indications of a possible ergogenic effect were from a study in which subjects cycled for 5 h with either glucose, glucose plus fructose, or water (22). In this study, RPE was lower, and self-selected cadence was higher in the later stages of exercise with glucose plus fructose compared with both glucose alone and water, suggesting a reduction in fatigue.

This is the first study to show clear performance benefits of glucose plus fructose over and above the effects of glucose ingestion. These findings, in combination with previous studies in our lab, suggest that increased exogenous carbohydrate oxidation can be linked to increased performance. Early studies investigated the effects of different types of carbohydrates on performance. In a recent review (20), it has been concluded that glucose was oxidized at higher rates than fructose. Studies have also demonstrated that glucose ingestion resulted in a greater endurance capacity than ingesting the same amount of fructose (1,27). Although these findings do not necessarily mean a causal relationship, the observations seem in line with the findings of this study. Some studies have shown little or no difference in performance or endurance capacity when glucose (or glucose polymers) plus fructose have been ingested compared with glucose alone (7,9,27,31). Murray et al. (32) investigated the effect of ingesting a 5% glucose polymer, a 4% glucose plus 2% sucrose beverage, or a 5% glucose polymer plus 2% fructose beverage on subjects' ability to perform a time trial of 480 pedal revolutions after 2 h of cycling at 55–65%  $\dot{V}_{\text{O}_{2\text{max}}}$ . Ingestion of both the glucose and fructose beverage and the glucose and sucrose beverage led to significant improvement in performance when compared with water placebo alone, whereas glucose ingestion did not. Although this may give some evidence that ingestion of a combination of glucose and fructose leads to improved performance compared with glucose alone, the glucose and fructose beverage was ingested at a greater rate ( $0.6$  vs  $0.4 \text{ g}\cdot\text{min}^{-1}$ ), which could explain the improved performance. Tarnopolsky et al. (34) have shown an increase in time to exhaustion when a mixture of glucose polymers and fructose was ingested compared with placebo. No effect was seen compared with glucose. However, in this study the amount of carbohydrate ingested would have

been insufficient to observe benefits from a drink with multiple transportable carbohydrates, because the transporters would not have been saturated. In addition, time to exhaustion may not be the most valid method by which to assess exercise performance (19).

Previous studies have shown that the maintenance of plasma glucose with carbohydrate ingestion delays fatigue (6). The data from the present study suggest that improvements in performance may not be attributed to a better maintenance of plasma glucose or total carbohydrate oxidation, because these were similar in the GF and G trials during the initial 2 h of steady-state exercise. However, it can be assumed, on the basis on previous work, that GF resulted in a sparing of endogenous carbohydrate sources during this time (12–17). This could be important because there would have been a greater reliance on carbohydrate as a fuel during the time trial (35). Coggan and Coyle (3) have shown that even with carbohydrate ingestion, carbohydrate oxidation can fall late in exercise, and, therefore, carbohydrate oxidation may have been maintained with ingestion of GF compared with G during the time trial, possibly through oxidation of lactate, which is increased with the ingestion of glucose and fructose. No respiratory exchange measurements were taken during the time trial, so this cannot be confirmed; further research needs to be conducted in this area.

The possible sparing in endogenous carbohydrate stores could be attributable either to a sparing of muscle or liver glycogen. During cycling exercise, there does not seem to be any sparing of muscle glycogen when carbohydrate is ingested (2,5,9,10,23,30). However, ingestion of carbohydrate leads to a sparing of liver glycogen (23). It is likely that the ingestion of glucose and fructose led to maintenance of hepatic glucose output late in exercise compared with glucose. Ingestion of GF resulted in increased plasma lactate concentrations. Fructose ingestion has repeatedly been shown to lead to an increase in plasma lactate compared with glucose (12–14,16,36). Fructose is phosphorylated to fructose-1-phosphate in the liver. Increased concentrations of fructose-1-phosphate lead to increases in the activity of pyruvate kinase, leading to formation of pyruvate and, thus, lactate (11). This increase in lactate production may have enabled a greater rate of gluconeogenesis to occur (29), although gluconeogenesis is usually believed to be suppressed in the presence of glucose ingestion. Probably more important is the fact that lactate produced from fructose can be oxidized in the muscle, providing another energy source (29).

In addition, fructose could have been used as a substrate. Assuming that hexokinase was not rate-limiting to CHO uptake, and that two sources of CHO (glucose and fructose) were able to enter the glycolytic pathway, at least in theory, the cell could have a greater CHO flux capability and ability to produce ATP. Although in this study CHO oxidation was not different between G and GF during work at the same constant power output, it is possible that motor units that would have been substrate limited were not recruited.

However, when the subjects were asked to self-select their power output, this might have been the case. In this study, we did not measure muscle fructose uptake or oxidation, and we do not have substrate oxidation data for the self-paced time trial, so it is impossible to dismiss or confirm this potential mechanism.

The higher power outputs in GF compared with *G* indicate a higher rate of energy turnover. One of the mechanisms by which the increased exogenous carbohydrate oxidation improves performance is by enabling the working muscle to match the rate of ATP resynthesis with the rate of degradation (28). McConell et al. (28) have shown that during prolonged cycling exercise, IMP accumulation in muscle was decreased when carbohydrate was ingested, suggesting a greater ability to maintain muscle energy

balance. Therefore, in the present study, the improvement in performance with GF compared with *G* may be attributable to a better maintenance of ATP resynthesis, enabling the maintenance of a higher power output. This could be the result of increased lactate oxidation in the muscle with GF.

In conclusion, the present study shows that ingestion of glucose fed at a rate of 1.2 g·min<sup>-1</sup> and fructose at a rate of 0.6 g·min<sup>-1</sup> improved endurance cycling performance by 8% compared with ingestion of glucose at a rate of 1.8 g·min<sup>-1</sup>. This is the first study to provide evidence that increased exogenous carbohydrate oxidation may result in increased endurance exercise performance.

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