

Glucose–Fructose Enhances Performance versus Isocaloric, but Not Moderate, Glucose

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ABSTRACT

BAUR, D. A., A. B. SCHROER, N. D. LUDEN, C. J. WOMACK, S. A. SMYTH, and M. J. SAUNDERS. Glucose–Fructose Enhances Performance versus Isocaloric, but Not Moderate, Glucose. *Med. Sci. Sports Exerc.*, Vol. 46, No. 9, pp. 1778–1786, 2014. **Purpose:** The effects of glucose-and-fructose (GF) coingestion on cycling time trial (TT) performance and physiological responses to exercise were examined under postprandial conditions. **Methods:** Eight trained male cyclists (age, 25 ± 6 yr; height, 180 ± 4 cm; weight, 77 ± 9 kg; $\dot{V}O_{2\max}$, 62 ± 6 mL·kg⁻¹·min⁻¹) completed the study. Subjects ingested either an artificially sweetened placebo (PL), a moderate-glucose beverage (MG, 1.03 g·min⁻¹), a high-glucose beverage (HG, 1.55 g·min⁻¹), or a GF beverage (1.55 g·min⁻¹, 2:1 ratio) during approximately 3 h of exercise, including 2 h of constant-load cycling (55% W_{\max} , 195 ± 17 W), immediately followed by a computer-simulated 30-km TT. Physiological responses (\dot{V}_E , $\dot{V}O_2$, RER, HR, blood glucose level, blood lactate level, and RPE) and incidences of gastrointestinal distress were assessed during early (15–20 min), middle (55–60 min), and late exercise (115–120 min) and during the TT. Magnitude-based qualitative inferences were used to evaluate differences between treatments. **Results:** In comparison with that in PL (52.9 ± 3.7 min), TT performances were faster with GF (50.4 ± 2.2 min, “very likely” benefit), MG (51.1 ± 2.4 min, “likely” benefit), and HG (52.0 ± 3.7 min, “possible” benefit). GF resulted in a “likely” improvement versus HG (3.0%) and an “unclear” effect relative to MG (1.2%). MG was “possibly” beneficial versus HG (1.8%). Few incidences of GI distress were reported in any trials. **Conclusions:** GF ingestion seems to enhance performance, relative to PL and HG. However, it is unclear whether GF improves performance versus moderate doses of glucose. **Key Words:** CARBOHYDRATE, CYCLING, PERFORMANCE, GASTROINTESTINAL TOLERANCE, ERGOGENIC AIDS

Carbohydrate (CHO) ingestion is recommended during prolonged exercise (35) because it can maintain blood glucose levels (5), spare endogenous CHO stores (8,23), sustain high rates of CHO oxidation late in exercise (5), enhance motor output (3), and improve time to fatigue (5) and performance (40). Recent evidence suggests that the ergogenic effects of CHO are dose dependent. Smith et al. (40) reported progressive improvements in prolonged cycling performance with increasing glucose dosages up to 1.0 g·min⁻¹ and up to 1.3 g·min⁻¹ with coingestion of glucose, maltodextrin, and fructose (39).

The dose–response effect of CHO ingestion likely results from augmented exogenous CHO oxidation, which is limited by intestinal absorption (34). Glucose is absorbed via the sodium–glucose linked transporter 1 at a peak rate of

approximately 1.1 g·min⁻¹, whereas fructose is absorbed via GLUT5 at rates up to approximately 0.6 g·min⁻¹ (20). When glucose (and/or glucose polymers) and fructose are consumed simultaneously, absorption and exogenous oxidation rates increase up to approximately 1.5 g·min⁻¹ (14,42) likely as a result of noncompetitive intestinal transport (38). This enhanced oxidation may be responsible for reported improvements in cycling performance with glucose–fructose (GF) coingestion (6,36,41).

Two studies have reported that GF ingestion during prolonged cycling augments performance by a substantial degree (approximately 8%) in comparison with isocaloric amounts of glucose (6,41). However, the volume of glucose consumed during the glucose-only trials in these studies (1.5–2.4 g·min⁻¹) exceeded maximal intestinal uptake rates (approximately 1.1 g·min⁻¹), likely resulting in considerable malabsorption. CHO malabsorption can cause gastrointestinal (GI) distress (33), and Triplett et al. (41) reported that four of nine subjects registered substantial GI symptoms during their glucose-only trial. This represents an important limitation in the existing literature because “excess” glucose in the beverages used for comparison against GF may have impaired performance. This hypothesis is supported by findings from Rowlands et al. (36), who reported that performance benefits from maltodextrin–fructose ingestion were partially influenced by changes in GI discomfort. Thus,

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it is possible that previous studies have overestimated the performance benefits of GF versus those of the recommended amounts of glucose, and no studies have directly compared the ergogenic effects of GF versus those of glucose beverages provided at doses below maximal intestinal uptake rates. Furthermore, subjects in the previous studies completed all cycling trials after an overnight fast, which might magnify performance benefits of CHO ingestion in comparison with those in trials conducted in the postprandial state (26).

The purpose of the current study was to examine the effects of GF ingestion on prolonged cycling performance under conditions that were consistent with current sports nutrition recommendations. Specifically, we tested cyclists in the postprandial state and examined the efficacy of GF in comparison with that of a moderate dose of glucose (i.e., $<1.1 \text{ g}\cdot\text{min}^{-1}$), an isocaloric high dose of glucose, and a placebo (PL) beverage.

MATERIALS AND METHODS

Subjects. Ten male endurance-trained cyclists and triathletes ($\dot{V}O_{2\text{max}} >55 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) from James Madison University and the Harrisonburg, VA, area volunteered to participate in this study. All subjects were experienced cyclists with a minimum of 3 yr of experience in cycling/triathlon events, without any recent breaks in training (self-reported minimum of at least 3 d of cycling per week in the 2 months before the study). Two subjects withdrew before completion because of circumstances unrelated to the study, resulting in complete data from eight subjects (age, 25 ± 6 yr; height, 180 ± 4 cm; weight, 77 ± 9 kg; $\dot{V}O_{2\text{max}}$, $62 \pm 6 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Subjects were provided written and oral information about experimental procedures and potential risks before giving informed consent. All procedures were approved by the James Madison University institutional review board before any testing.

Cardiorespiratory fitness. Subjects performed an incremental exercise test to exhaustion on a bicycle ergometer (Velotron; Racermate, Inc., Seattle, WA) to determine $\dot{V}O_{2\text{max}}$ using a similar protocol to those described in previous studies (10,41). Subjects completed a 5-min warm-up at 100 W and then began the test at a self-selected workload intended to be a comfortable pace for a 60-min ride (approximately 150 W); power was subsequently increased by 25 W every 2 min until volitional exhaustion. Metabolic measurements were assessed throughout each stage of the test using a Moxus Modular Metabolic System (AEI Technologies, Pittsburgh, PA). $\dot{V}O_{2\text{max}}$ was determined by the highest 30-s mean oxygen uptake value. Peak power at $\dot{V}O_{2\text{max}}$ (W_{max}) was defined by the power corresponding to the final completed stage and was used to prescribe workloads for the 120-min constant-load segment of subsequent trials.

Exercise trials. Subjects completed five trials (one familiarization trial followed by four experimental trials) on the aforementioned cycle ergometer. Trials consisted of 120 min

of constant-load cycling at 55% W_{max} ($195 \pm 6 \text{ W}$), followed by a simulated 30-km time trial (TT). Subjects were permitted to use the restroom (if needed) during the short period required to switch the ergometer into TT mode (approximately 3 min), and this period was matched across trials for each subject. Trials were separated by 6–14 d. The familiarization trial was identical to the experimental trials (see later portion), except that no blood samples were obtained and subjects received only water while cycling. Subjects were asked to void their bladder before all trials. A pedestal fan was placed approximately 2 m from the handlebars and used on a high-speed setting for uniform cooling during each trial. Subjects were encouraged to treat the TT portion of each trial as a competitive event and provide a maximal effort. Trials were conducted independently in a quiet room with minimal disruptions from the researchers (i.e., other than to provide beverages and obtain dependent measurements). In addition, no verbal encouragement was provided during the trials and subjects did not receive any feedback during the TT (such as time or power output) other than the distance completed.

Treatments. A randomly counterbalanced, double-blind, PL-controlled design was implemented to compare the effects of four separate treatment conditions on performance, cardiovascular, and metabolic physiology. During each trial, subjects consumed a total of 2250 mL of fluid according to the following protocol. Immediately before exercising, subjects received 600 mL of treatment beverage. Thereafter, subjects received a 150-mL bolus every 15 min during the constant-load portion of the trial (1200 mL in total) and at three points during the 30-km TT (7.5, 15, and 22.5 km; 450 mL in total). Treatments consisted of either 1) a 12% (2:1 ratio) GF beverage (Tate and Lyle, Decatur, IL), 2) an 8% glucose beverage (moderate-glucose beverage (MG)), 3) a 12% glucose beverage (high-glucose beverage (HG)), or 4) a noncaloric, artificially sweetened PL beverage (Splenda, Fort Washington, PA). Each solution also contained $470 \text{ mg}\cdot\text{L}^{-1}$ of sodium chloride (Morton Salt, Chicago, IL) and $200 \text{ mg}\cdot\text{L}^{-1}$ of potassium chloride (NOW Foods, Bloomingdale, IL). Average CHO ingestion rates for the entire trial, including the constant-load period, 3-min ergometer transition period, and TT portion of the trial, were as follows: $1.03 \text{ g glucose}\cdot\text{min}^{-1} + 0.52 \text{ g fructose}\cdot\text{min}^{-1}$ (GF), $1.03 \text{ g glucose}\cdot\text{min}^{-1}$ (MG), $1.55 \text{ g glucose}\cdot\text{min}^{-1}$ (HG), and $0 \text{ g glucose}\cdot\text{min}^{-1}$ (PL). The MG ingestion rate was chosen because it falls at the upper end of intestinal uptake rates for glucose (20). CHO delivery rates for GF and HG were chosen for comparison with those in studies using similar amounts (17,22,24) and to permit us to compare the GF beverage against beverages matched for glucose (MG) and total CHO/calories (HG).

30-km TT performance. Finishing time and mean power output (W) during the preloaded 30-km TT were used as performance criteria. We previously assessed the reproducibility of cycling time/power measurements using identical equipment in our laboratory. Using a similar performance trial (20 km of cycling over a simulated hilly course) and a comparable set of male subjects ($n = 10$, age 28 ± 8 yr, 73 ± 6 kg,

65 ± 9 mL·kg⁻¹·min⁻¹), the coefficient of variation between repeated trials (under PL conditions after a familiarization trial) was 1.4% for time and 2.6% for power output (10). Similarly, we obtained repeatability data from six pilot subjects, who performed repeated trials (under PL conditions) using the exact trial used in this study (i.e., 30-km trial after 2 h of constant-load cycling), and obtained a coefficient of variation of 3.4% for 30-km performance times.

Physiological measurements. Oxygen uptake ($\dot{V}O_2$), expired ventilation (\dot{V}_E), and RER were assessed using a Moxus Modular Metabolic System (AEI Technologies, Pittsburgh, PA) at the following time points: minutes 15–20, 55–60, and 115–120 of the constant-load phase and at 20 km of the TT. These time points were selected to correspond with early, middle, and late exercise and a representative value from the TT. Aggregates of the final 3 min of each phase were recorded.

HR (Suunto, Vaanta, Finland) and RPE (6–20 Borg scale) were recorded at minutes 20, 60, and 120 of constant-load cycling and at 20 km of the TT. Fingertick blood samples (approximately 0.5 mL) were obtained at rest and at the time points indicated above. Glucose and lactate levels were determined immediately from whole blood using automated instrumentation (YSI 2300 STAT glucose/lactate analyzer; YSI Life Sciences, Yellow Springs, OH). Total CHO oxidation during the TT was calculated from metabolic data using methods described previously (25).

GI distress scale. Subjects verbally indicated their perceived level of upper GI distress at minutes 30, 60, and 120 of constant-load cycling and at 20 km of the TT. Using a 10-point scale (1, not at all; 10, very, very much), subjects rated the following symptoms: stomach problems, GI cramping, bloated feeling, diarrhea, nausea, dizziness, headache, belching, and vomiting (19).

Dietary and exercise controls. Subjects were instructed to 1) maintain consistent dietary habits for 72 h before each trial, 2) record food intake 24 h before their first experimental trial, 3) replicate their food intake for the 24 h preceding each subsequent experimental trial, 4) refrain from heavy and/or unaccustomed exercise for 48 h before each experimental trial, 5) maintain consistent exercise habits between trials and record all physical activity performed during the 72 h preceding each experimental trial, and 6) abstain from alcohol and caffeine for 24 h and 12 h, respectively, before the experimental trials. Subjects performed all trials in a fed state. Specifically, subjects consumed 20%–25% of their estimated daily caloric expenditure (Harris–Benedict equation) in the form of a liquid meal replacement (Ensure® Shakes; Abbott Laboratories, Abbott Park, IL) in the evening before each trial (8–10 h before). Two hours before all exercise trials, subjects consumed a standardized meal consisting of approximately 500 kcal (cereal with milk, orange juice, and strawberry yogurt). Average dietary intakes during the day before the trials (i.e., not including the standardized meal replacement or standardized breakfast) did not differ significantly between trials for calories (1830 ± 203 kcal, $P = 0.74$),

CHO (231 ± 32 g, $P = 0.78$), protein (96 ± 15 g, $P = 0.64$), and fat (60 ± 4 g, $P = 0.56$).

Statistical analyses. Univariate ANOVA (randomized complete block design) were used to determine treatment differences for all variables, unless otherwise stated. Simple contrasts between treatment conditions were used to generate P values for subsequent analysis, as will be described later. Residuals from ANOVA analyses were visually inspected for nonuniformity of variance and assessed for normality (using Komolgorov–Smirnov normality tests). In cases of heteroscedasticity or when normality was violated ($\dot{V}O_2$ at 55 min and 115 min, RER at 115 min and TT, lactate level at 20 min, blood glucose level at 20 min, and RPE at 60 min), variables were log-transformed before analysis. Statistical analyses were performed using Statistical Package for Social Sciences 20.0 for Windows (SPSS Inc., Chicago, IL).

Probabilistic magnitude-based inferences about the data are reported in the manuscript using methods described by Hopkins et al. (13). This approach has been used in several recent studies, which similarly examined the effects of nutritional interventions on exercise performance (32,36,40). As discussed previously by Rowlands et al. (37), this approach has several advantages over traditional null hypothesis testing because the method emphasizes effect magnitudes and estimate precision and qualifies the probability of an important effect with interpretive descriptors. Ninety percent confidence intervals (CI) are presented to illustrate uncertainty in treatment effects because it represents an “unclear” effect having >5% chance of being positive and >5% chance of being negative (13). Threshold values for a substantial change were calculated as 0.2SD (from PL trial) (11). With respect to our primary outcome measurement (TT performance), this threshold related to a minimum “worthwhile” performance improvement/decrement of 0.72 min (or 8.0 W) during the TT, equivalent to an improvement from the 50th to the 58th percentile (11). A published spreadsheet (12) was used to classify treatment effects as beneficial/positive, harmful/negative, or trivial/negligible. Likelihoods of reaching the substantial change threshold were classified as follows: <1%, almost certainly no chance; 1%–5%, very unlikely; 5%–25%, unlikely; 25%–75%, possible; 75%–95%, likely; 95%–99%, very likely; and >99%, almost certain. If the 90% CI included values that exceeded the threshold values for both a negative and positive effect, effects were classified as “unclear”. For ease of interpretation, all data (including those that were log-transformed before analysis) are presented as means ± SD or means ± CI (where indicated).

GI distress scores were analyzed with a frequency table for severe symptoms (a score of ≥5), as described previously (18).

The inclusion of eight subjects provided us with ample statistical power to detect meaningful differences in performance between treatment beverages using the aforementioned methodology. Specifically, a 2% difference in TT performance (i.e., 62 s during the final 30 km) represented an effect size of 1.54 SD units based on an SD of 3.0 min and test–retest

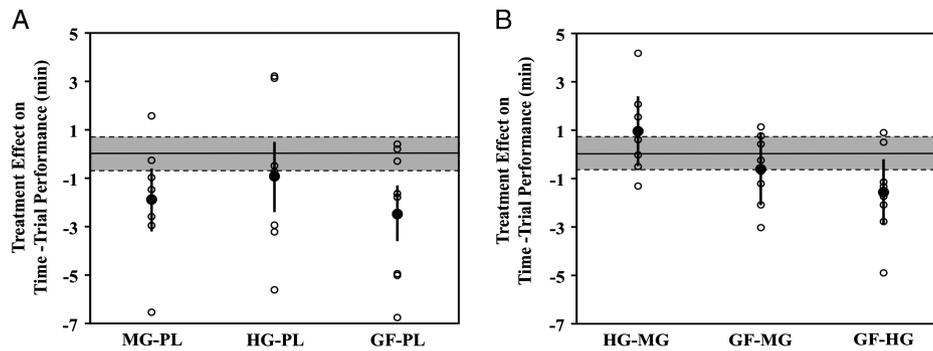


FIGURE 1—Effects of CHO beverages on TT performance. **A**, Treatment effects (mean differences \pm 90% CI) of each CHO beverage versus those of PL. **B**, Treatment effects between CHO beverages. *Filled circles* represent the mean value, and *open circles* are individual scores (in four cases, individual scores are obscured by the mean value). *Dashed lines* represent threshold values for substantial benefit/harm (0.2SD). MG, 1.03 g·min⁻¹; HG, 1.55 g·min⁻¹; GF, 1.55 g·min⁻¹. Qualitative inferences and *P* values: PL–MG, “likely” benefit (93.1%) for MG (*P* = 0.029); PL–HG, “possible” benefit (59.6%) for HG (*P* = 0.263); PL–GF, “very likely” benefit (98.8%) for GF (*P* = 0.005); MG–HG, “possible” harm (61.3%) for HG (*P* = 0.247); MG–GF, “unclear” (*P* = 0.458); HG–GF, “likely” benefit (85.9%) for GF (*P* = 0.065).

reliability of $r = 0.95$ (values obtained from pilot testing) (28). An effect of this magnitude could be detected with a power ($1 - \beta$) of 0.80 at an α level of 0.05 (28). In addition, the use of magnitude-based inferences (described previously) allowed the investigators to make inferences regarding potentially meaningful differences between treatments with greater sensitivity than via traditional hypothesis testing (13).

RESULTS

30-km TT performance. No effects of trial order were observed for TT (*P* = 0.938) or power output (*P* = 0.952). Average performance times during the 30-km TT were as follows: PL, 52.9 \pm 3.7 min (217 \pm 40 W); MG, 51.1 \pm 2.4 min (237 \pm 30 W); HG, 52.0 \pm 3.7 min (229 \pm 38 W); and GF, 50.4 \pm 2.2 min (244 \pm 27 W). Differences in performance times between treatments (\pm 90% CI) are illustrated in Figure 1,

along with *P* values and qualitative inferences for each treatment comparison. Qualitative inferences are not shown for treatment effects on power output; for all cases, these were identical to those reported for performance time.

Metabolic measurements. $\dot{V}O_2$ and \dot{V}_E data are displayed in Table 1. There were no systematic differences between treatments during the constant-load portion of the trials. Differences in $\dot{V}O_2$ during the TT closely matched differences in workloads. Specifically, $\dot{V}O_2$ during all CHO treatments were “likely” (MG and HG) or “very likely” (GF) higher than that during PL. $\dot{V}O_2$ during GF was also “likely” higher versus those during MG/HG. $\dot{V}O_2$ differences between MG and HG were “unclear”. \dot{V}_E during the TT was “likely higher” with all CHO treatments versus that during PL. Among the CHO treatments, a “possibly trivial” increase in \dot{V}_E with GF versus that with MG during the TT was the only apparent difference between beverages.

TABLE 1. \dot{V}_E , $\dot{V}O_2$, RER, and HR responses during constant-load exercise and subsequent TT.

	15 min	60 min	120 min	TT
\dot{V}_E (L·min ⁻¹)				
PL	68.1 \pm 5.8	69.5 \pm 5.5	72.2 \pm 6.5	79.0 \pm 20.9
MG	67.8 \pm 5.6	70.4 \pm 5.0	71.6 \pm 5.1	88.9 \pm 20.0 ^a
HG	68.4 \pm 8.3	70.7 \pm 8.5	72.6 \pm 9.2	88.5 \pm 23.8 ^a
GF	67.3 \pm 6.8	71.9 \pm 7.0	70.8 \pm 5.3	92.0 \pm 14.6 ^{b,c}
$\dot{V}O_2$ (L·min ⁻¹ ; % _{max})				
PL	2.8 \pm 0.3 (59 \pm 6)	2.9 \pm 0.3 (61 \pm 6)	3.0 \pm 0.3 (63 \pm 6)	3.2 \pm 0.6 (67 \pm 12)
MG	2.8 \pm 0.2 (59 \pm 4)	2.9 \pm 0.2 (61 \pm 4)	2.9 \pm 0.2 (61 \pm 4)	3.4 \pm 0.4 ^a (71 \pm 8)
HG	2.9 \pm 0.4 (61 \pm 8)	2.9 \pm 0.3 (61 \pm 6)	3.0 \pm 0.4 (63 \pm 8)	3.4 \pm 0.6 ^a (71 \pm 12)
GF	2.8 \pm 0.2 (59 \pm 4)	2.9 \pm 0.3 (61 \pm 6)	2.9 \pm 0.3 (61 \pm 6)	3.6 \pm 0.4 ^{b,d} (76 \pm 8)
RER				
PL	0.91 \pm 0.03	0.87 \pm 0.03	0.84 \pm 0.03	0.84 \pm 0.02
MG	0.94 \pm 0.02	0.91 \pm 0.02 ^e	0.89 \pm 0.02 ^e	0.90 \pm 0.04 ^e
HG	0.92 \pm 0.02	0.90 \pm 0.03 ^b	0.88 \pm 0.02 ^e	0.89 \pm 0.02 ^e
GF	0.94 \pm 0.02	0.92 \pm 0.02 ^e	0.89 \pm 0.02 ^{e,p}	0.89 \pm 0.02 ^e
HR (bpm; % _{max})				
PL	118 \pm 8 (65 \pm 3)	122 \pm 10 (67 \pm 4)	129 \pm 12 (71 \pm 5)	140 \pm 13 (77 \pm 6)
MG	124 \pm 8 (68 \pm 3)	126 \pm 9 (69 \pm 4)	130 \pm 11 (71 \pm 5)	150 \pm 14 ^e (82 \pm 6)
HG	125 \pm 10 (68 \pm 4)	128 \pm 12 (70 \pm 6)	133 \pm 13 (73 \pm 6)	152 \pm 15 ^b (83 \pm 8)
GF	121 \pm 9 (66 \pm 4)	124 \pm 8 (68 \pm 4)	127 \pm 10 (69 \pm 5)	149 \pm 14 ^e (82 \pm 7)

Data are presented as mean \pm SD.

GF, 1.55 g·min⁻¹; HG, 1.55 g·min⁻¹; MG, 1.03 g·min⁻¹.

^a“Likely” positive effect versus that in PL.

^b“Very likely” positive effect versus that in PL.

^c“Possibly trivial” higher value versus that in HG.

^d“Likely” positive effect versus those in MG and HG.

^e“Almost certain” positive effect versus that in PL.

RER data are displayed in Table 1. CHO ingestion (all treatments) resulted in higher RER values versus those in PL at 55 min and 115 min of steady-state cycling. “Unclear” differences were observed in late exercise (115 min) of the constant-load portion between GF and MG as well as between MG and HG. There was a “possibly trivial” increase in RER with GF versus that with HG at the same time point. During the TT, all CHO treatments resulted in “almost certain” higher RER values than those in PL. Differences between CHO treatments were “unclear”.

All CHO trials (MG, $2.74 \pm 0.69 \text{ g}\cdot\text{min}^{-1}$; HG, $2.57 \pm 0.58 \text{ g}\cdot\text{min}^{-1}$; and GF, $2.79 \pm 0.34 \text{ g}\cdot\text{min}^{-1}$) resulted in “almost certain” higher total CHO oxidation versus that in PL ($1.77 \pm 0.46 \text{ g}\cdot\text{min}^{-1}$) during the TT. Differences between CHO treatments were “unclear”.

HR and RPE. HR data are displayed in Table 1. There was no evidence of any systematic differences between treatments during the constant-load portion of the trials. All CHO treatments resulted in “likely” higher HR values versus those in PL during the TT. Differences in HR between CHO treatments were “unclear”. No differences were observed between treatments for RPE.

Blood glucose and lactate. Blood glucose data are displayed in Figure 2. With all CHO treatments, blood glucose level was “very likely” increased versus that with PL during both constant-load cycling and the TT. HG was “very likely” and “likely” to increase late-exercise blood glucose level (120 min), relative to MG and GF, respectively. However, differences in blood glucose level with HG versus that with MG and GF were “unclear” during the TT. Differences in late-exercise blood glucose level were “unclear” between GF and MG. However, blood glucose level was “likely” increased with GF versus that with MG during the TT.

Blood lactate data are presented in Figure 3. Early-exercise blood lactate level in the GF trial was “very likely” higher than that in PL and “likely” higher than that in MG/HG. In addition, late-exercise blood lactate level was “likely” higher

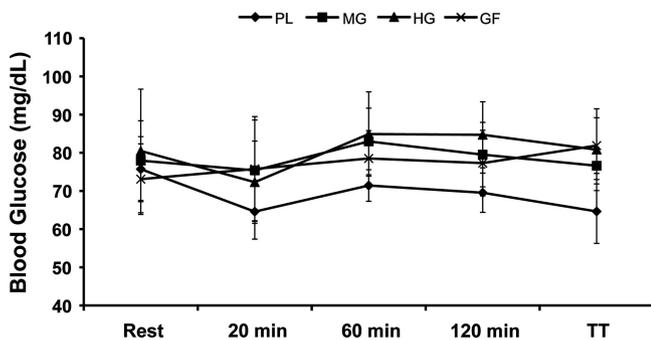


FIGURE 2—Blood glucose responses during cycling. Blood glucose level was “very likely” increased with MG, HG, and GF versus that with PL during both constant-load cycling and the TT. HG was “very likely” and “likely” to increase blood glucose level at 120 min, relative to MG and GF, respectively. GF resulted in “likely” higher blood glucose level versus that in MG during the TT, whereas other differences between CHO treatments were “unclear”. Data are presented as mean \pm SD. MG, $1.03 \text{ g}\cdot\text{min}^{-1}$; HG, $1.55 \text{ g}\cdot\text{min}^{-1}$; GF, $1.55 \text{ g}\cdot\text{min}^{-1}$.

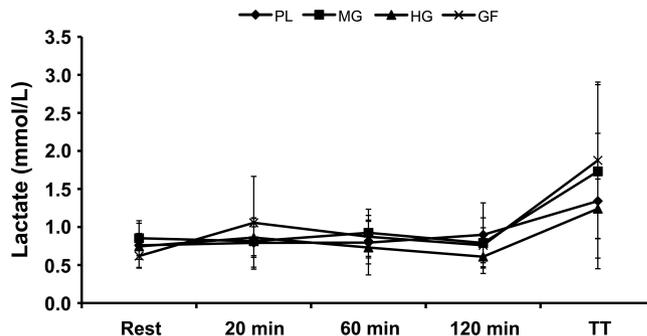


FIGURE 3—Blood lactate responses during cycling. At 20 min, blood lactate level in the GF trial was “very likely” higher than that in PL and “likely” higher than that in MG/HG. At 120 min, blood lactate level was “likely” higher with GF and MG versus that with HG. During the TT, lactate level was “likely” higher with GF versus that with HG and “likely” higher with GF and MG versus that with PL. All other differences were “unclear”. Data are presented as mean \pm SD. MG, $1.03 \text{ g}\cdot\text{min}^{-1}$; HG, $1.55 \text{ g}\cdot\text{min}^{-1}$; GF, $1.55 \text{ g}\cdot\text{min}^{-1}$.

with GF and MG versus that with HG. During the TT, lactate level was “likely” higher with GF versus that with HG and “likely” higher with GF and MG versus that with PL. All other effects were unclear.

GI distress symptoms. Reported symptoms of GI distress were generally low in all trials (average values were ≤ 1.75 for all individual symptoms, evaluated at all individual time points). Only two subjects reported any GI distress symptoms ≥ 5 (“severe” or higher) in the constant-load portion of any of the trials. These included symptoms in the following areas: stomach problems, stomach cramping, nausea, dizziness, headache, and vomiting (no ratings ≥ 5 were reported for bloated feeling, diarrhea, and belching). During the TT portion of the trials, three subjects (including the two aforementioned participants) reported GI distress symptoms ≥ 5 . These ratings were observed for the same symptoms as those reported during constant-load cycling, with the exception that no moderate/severe ratings were observed in the category of stomach cramping. Individual GI distress ratings are displayed for representative symptoms for the three individuals who reported moderate-to-severe symptoms (Table 2). No systematic differences in GI distress ratings were observed between individual treatments during constant-load cycling. Similarly, no systematic differences in symptoms were observed during the TT, other than the observation that dizziness ratings during the PL trial were higher than those during baseline levels in all three subjects.

DISCUSSION

Several recent studies have examined the effects of GF coingestion on cycling performance in comparison with those of isocaloric amounts of glucose (6,36,41). Although the results of these studies have been positive, they may have overestimated the performance benefits of GF because of high glucose levels in their comparison beverages. The present investigation was the first to compare the ergogenic effects of a GF beverage with those of a recommended

TABLE 2. Individual ratings of GI distress during cycling.

	Constant-Load Exercise ^a				TT			
	PL	MG	HG	GF	PL	MG	HG	GF
Stomach problems								
Subject C	6	1	3	1	3	1	3	1
Subject E	1	5	1	4	1	1	1	1
Subject G	1	1	1	1	1	6	1	1
Nausea								
Subject C	7	1	1	1	7	1	3	1
Subject E	1	1	1	1	1	1	1	1
Subject G	1	1	1	1	1	7	1	1
Dizziness								
Subject C	1	1	1	1	4	1	1	1
Subject E	1	1	1	1	5	1	1	1
Subject G	2	1	1	1	3	6	1	3

Ratings are shown only for subjects reporting ratings ≥ 5 during any of the trials.

GF, 1.55 g·min⁻¹; HG, 1.55 g·min⁻¹; MG, 1.03 g·min⁻¹.

^aRatings represent the highest values reported during constant-load cycling.

moderate dosage of glucose (MG, 1.03 g·min⁻¹). GF was also compared with an isocaloric glucose beverage (HG) and a PL. Unlike previous studies (6,36,41), all trials were performed in the postprandial state to replicate the conditions in which athletes typically compete. The primary finding of the current study was that GF “very likely” improved performance versus PL (4.7% faster TT) and “likely” improved performance when compared with HG (3.0%), whereas potential performance benefits for GF versus those for MG (1.2%) were “unclear”.

The CHO beverages examined in the present study produced potentially meaningful performance enhancements in comparison with those in PL. This finding is consistent with numerous previous studies reporting that CHO ingestion improves time to fatigue (5) and/or performance (6,36,40) during prolonged cycling, although not all studies have reported ergogenic effects (4,7). Beneficial effects have largely been attributed to the maintenance of euglycemia and higher rates of CHO oxidation throughout prolonged exercise via increased exogenous CHO oxidation (6) and the sparing of endogenous glycogen reserves in the liver (23) and possibly in the muscle (8). In addition, CHO ingestion may also influence exercise performance via stimulation of the CNS (3,26).

Our finding of a 3.0% improvement with GF versus that with HG is in general agreement with (although smaller than) findings of previous studies investigating cycling performance with GF consumption. Currell and Jeukendrup (6) reported that high rates of GF coingestion (1.8 g·min⁻¹, 2:1 ratio) resulted in 8% faster completion times during a 40-km TT (after 2 h of constant-load cycling) in comparison with that in a calorically matched glucose-only beverage. Similarly, Triplett et al. (41) observed an 8% improvement in power output during 100 km of intermittent cycling with GF intake (2.4 g·min⁻¹, 2:1 ratio). These performance enhancements have been primarily attributed to augmented exogenous CHO oxidation, which is influenced by intestinal absorption (34). Glucose transport occurs predominantly via the sodium-dependent sodium–glucose linked transporter 1 at rates up to approximately 1.1 g·min⁻¹, whereas fructose is transported

predominantly by GLUT5 at peak rates of approximately 0.6 g·min⁻¹ (20). Coingestion of GF at high rates have resulted in exogenous oxidation rates exceeding 1.5 g·min⁻¹ (14,42) likely as a result of noncompetitive intestinal transport (38). These rates are considerably higher than those reported with isocaloric amounts of glucose alone (14,15,24).

The precise mechanism by which augmented exogenous CHO oxidation enhances performance remains unclear. Exogenous oxidation can potentially spare endogenous reserves, providing greater CHO availability (and total CHO oxidation) in late exercise. However, although a previous study reported a trend toward endogenous CHO sparing with GF intake (15), most have reported no significant differences versus isocaloric glucose beverages (14,17,42). Coingestion of GF may also augment total CHO oxidation via enhanced lactate metabolism (15,16,27). To this end, we observed that early-exercise blood lactate level was increased with GF versus that with all other treatments (“likely”/“very likely”). In addition, late-exercise blood lactate level with GF was higher than that with the HG trial (“very likely”). However, late-exercise lactate level in the MG trial was also higher than that in HG (“very likely”) and not different from that in GF at this time point. Furthermore, differences in total CHO oxidation between CHO treatments during the TT were “unclear”, suggesting that differences in total CHO oxidation were unlikely to have been a major contributor to enhanced performance with GF. Although not a focus of the present investigation, previous studies have also reported that GF ingestion increases gastric emptying and fluid delivery versus isocaloric glucose-only beverages (17,22). Therefore, it is possible that GF may influence performance via mechanisms that are independent of CHO availability. Further study is warranted to determine the specific mechanisms by which endurance performance may be influenced by the combination of multiple CHO.

Although generally consistent with previous studies, the 3.0% improvement in TT performance observed with our GF treatment (versus that with the isocaloric HG beverage) is notably lower than the 8% improvements reported by Currell and Jeukendrup (6) and Triplett et al. (41). In addition, our novel finding of an unclear effect for GF versus MG (1.2%) indicates that the ergogenic effects of GF ingestion may be more modest than those previously reported (6,41). One explanation for the reduced benefit observed in the current study is that our trials were conducted in the postprandial state. Previous studies have reported that the ergogenic effects of CHO observed in high-intensity cycling trials (i.e., approximately 60 min at $>85\% \dot{V}O_{2peak}$) were likely the result of improved motor output via stimulation of oral CHO receptors (3). As others have speculated (36), beverages containing fructose may produce greater stimulation of oral CHO receptors, possibly contributing to the observed performance enhancements with GF ingestion. However, the influence of CHO on the CNS may be partially attenuated in the postprandial state (26), which could have reduced potential performance benefits from GF in the current study.

Furthermore, the ingestion of a preexercise meal would likely have an effect on endogenous CHO stores at the onset of exercise (31) and potentially influence substrate use (30). Collectively, these factors could explain the larger treatment effects reported in previous studies reporting ergogenic effects with GF, which were conducted after an overnight fast (6,41).

Another factor, which may have affected the magnitude of our treatment effects, was a lower total CHO intake rate compared with those used by Currell and Jeukendrup (6) and Triplett et al. (41) ($1.55 \text{ g}\cdot\text{min}^{-1}$ vs $1.8\text{--}2.4 \text{ g}\cdot\text{min}^{-1}$). Enhanced delivery of CHO via noncompetitive glucose and fructose transport results in increased oxidation of exogenous CHO (14,42), which seems to be dose dependent (39,40). Perhaps, the larger doses of GF administered by Currell and Jeukendrup (6) and Triplett et al. (41) resulted in greater absorption and subsequent oxidation of CHO, resulting in a larger performance improvement. However, Smith et al. (39) reported a curvilinear dose–response effect with maltodextrin/glucose/fructose, with optimal performance occurring at intake rates of approximately $1.3 \text{ g}\cdot\text{min}^{-1}$. Moreover, oxidation efficiency (i.e., proportion of ingested CHO that are oxidized) likely decreases at high CHO ingestion rates ($\geq 1.3 \text{ g}\cdot\text{min}^{-1}$) (21,32). Thus, our chosen CHO delivery rate of $1.55 \text{ g}\cdot\text{min}^{-1}$ would seem to approximate theoretically optimal levels.

Disparities between studies may also have been influenced by differences in exercise protocols and/or subject characteristics. For example, the total exercise durations in the studies by Currell and Jeukendrup (6) (approximately 180 min) and Triplett et al. (41) (approximately 204–221 min) were slightly longer than those in the present study (approximately 170–173 min). Hypothetically, increased exercise duration and/or intensity could impair metabolic status to a greater extent, which could magnify the potential ergogenic effects of CHO ingestion during exercise. Similarly, differences in subject characteristics between studies (e.g., training status, familiarization with exercise protocols, etc.) can influence within- and between-subject variability, which could alter the magnitude of treatment effects between studies.

Differences in the magnitude of benefits between the current study and previous studies (6,41) could also be attributed to GI tolerance of CHO. Previous studies used higher rates of CHO ingestion in their glucose-only trials (1.8 and $2.4 \text{ g}\cdot\text{min}^{-1}$ (6,41)), which exceeded the presumed maximal absorption rates of glucose ($1.1 \text{ g}\cdot\text{min}^{-1}$) (20). This likely resulted in greater CHO accumulation in the gut, which has been associated with GI distress (33). High incidences of substantial GI distress in glucose-only trials reported by Triplett et al. (41) (four of nine subjects versus none of eight subjects registering “severe” symptoms in our HG trials) support this notion (41). Assuming that severe GI distress limits performance (36), the large performance differences reported by Currell and Jeukendrup (6) and Triplett et al. (41) (8%) may be partly explained by GI distress related to “excess” glucose in the glucose-only comparison beverages. This hypothesis is supported by findings of Rowlands et al.

(36), who reported a 1.8% improvement in mountain bike performance times with maltodextrin–fructose (versus those with an isocaloric maltodextrin–glucose beverage) which was reduced to 1.1% after statistically removing the effects of GI discomfort (36). These values are similar to our observed differences between GF and MG treatments (1.2%, “unclear” effect).

The present findings suggest that high levels of glucose intake may reduce the performance benefits of CHO because the “possible” ergogenic effects of HG (1.7% improvement versus that in PL) were smaller than those observed for MG (3.4%) and GF (4.7%). This is also indicated by the larger performance benefits for GF versus HG, in comparison with those for GF versus MG. However, very few symptoms of severe GI distress were reported in our HG trials, possibly as a result of lower intake rates ($1.55 \text{ g}\cdot\text{min}^{-1}$), versus those reported in previous studies (6,41). Thus, the potentially detrimental effects of HG on performance in our study (compared with those of MG/GF) cannot be directly attributed to GI distress symptoms *per se*. However, we cannot dismiss the possibility that our subjects anticipatorily selected lower TT intensities during the HG trial to prevent severe GI distress. Furthermore, others (32) have speculated that nausea (presumably caused by CHO malabsorption (33)) may blunt motor output via stimulation of receptors in the gut. To this end, gut receptors that respond to distension (2) and taste (9) have been identified. Moreover, the appearance of GLUT2 transporters in the intestine seems to increase in response to highly concentrated amounts of glucose, presumably the result of chemoreceptors (29). This raises the possibility that receptors in the gut (responding to glucose concentration) may have preemptively blunted motor drive during the HG trials, contributing to the larger differences between GF/HG beverages (“likely”, 3.0%) versus those observed between GF/MG (“unclear”, 1.2%), although this idea is purely speculative. Furthermore, GI tolerances vary between individuals (41), so the glucose ingestion rate in the HG trial may have been excessive for some subjects but not for others. This supposition is supported by the higher between-subject variability in the HG trial (SD, 3.7 min) in comparison with that in the MG/GF trials (2.2–2.4 min) and the observation that two subjects had their fastest CHO-fed TT in the HG trial.

Under postprandial conditions, ingesting a GF beverage during exercise ($1.55 \text{ g}\cdot\text{min}^{-1}$) resulted in “very likely”/“likely” improvements in prolonged cycling performance versus those in PL/isocaloric glucose solutions, respectively. However, differences in performance for GF versus a glucose beverage containing $1.03 \text{ g}\cdot\text{min}^{-1}$ were “unclear”. Most current sports nutrition guidelines recommend ingesting $0.5\text{--}1.0 \text{ g}\cdot\text{min}^{-1}$ during exercise lasting $\geq 2 \text{ h}$ (35). Recent studies reporting performance enhancements with GF coingestion at rates $\geq 1.2 \text{ g}\cdot\text{min}^{-1}$ (6,36,41) have resulted in some guidelines recommending higher rates of GF ingestion during events longer than 2.5 h (1). Our findings suggest that the ergogenic effects of GF ingestion may be more modest than previously reported, particularly in the postprandial state

and when compared with that of moderate doses of glucose. Notably, our observed performance benefit for GF versus that for MG (1.2%) is also in line with recent findings (36), which have statistically corrected for differences in GI tolerance. Despite our reported statistical inference that this is an “unclear” effect, further study of the ergogenic effects of GF beverages is warranted because this would be deemed a functionally meaningful improvement to athletes if upheld in future studies.

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