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Oxidation of exogenous glucose, sucrose, and maltose during prolonged cycling exercise

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Jentjens, Roy L. P. G., Michelle C. Venables, and Asker E. Jeukendrup. Oxidation of exogenous glucose, sucrose, and maltose during prolonged cycling exercise. *J Appl Physiol* 96: 1285–1291, 2004. First published December 2, 2003; 10.1152/jappphysiol.01023.2003.—The purpose of the present study was to investigate whether combined ingestion of two carbohydrates (CHO) that are absorbed by different intestinal transport mechanisms would lead to exogenous CHO oxidation rates of >1.0 g/min. Nine trained male cyclists (maximal O_2 consumption: 64 ± 2 ml·kg body wt⁻¹·min⁻¹) performed four exercise trials, which were randomly assigned and separated by at least 1 wk. Each trial consisted of 150 min of cycling at 50% of maximal power output ($60 \pm 1\%$ maximal O_2 consumption), while subjects received a solution providing either 1.8 g/min of glucose (Glu), 1.2 g/min of glucose + 0.6 g/min of sucrose (Glu+Suc), 1.2 g/min of glucose + 0.6 g/min of maltose (Glu+Mal), or water. Peak exogenous CHO oxidation rates were significantly higher ($P < 0.05$) in the Glu+Suc trial (1.25 ± 0.07 g/min) compared with the Glu and Glu+Mal trials (1.06 ± 0.08 and 1.06 ± 0.06 g/min, respectively). No difference was found in (peak) exogenous CHO oxidation rates between Glu and Glu+Mal. These results demonstrate that, when a mixture of glucose and sucrose is ingested at high rates (1.8 g/min) during cycling exercise, exogenous CHO oxidation rates reach peak values of ~ 1.25 g/min.

carbohydrate absorption; stable isotopes; substrate utilization; metabolism; sodium-dependent glucose transporter 1; cyclists

THE OXIDATION OF INGESTED carbohydrate (CHO) has been intensively investigated by using stable and radioactive isotope techniques (for reviews, see Refs. 13, 17). Several authors have examined exogenous CHO oxidation rates during cycling exercise and have reported peak values approaching 1.0 g/min for exercise durations of up to 180 min (13, 18, 19). Although there are studies that have reported slightly higher values (6, 30, 32), it is generally believed that the maximal oxidation rate of ingested CHO is ~ 1.0 g/min. Suggestions have been made that the rate of exogenous CHO oxidation is limited by the rate of absorption of CHO by the intestine (14, 16) and subsequent transport of glucose into the bloodstream regulated by the liver (17, 19).

Interestingly, our laboratory has recently shown that, when a mixture of glucose and fructose is ingested at high rates (1.8 g/min) during cycling exercise, exogenous CHO oxidation rates can reach peak values of ~ 1.3 g/min (16). The ingestion of fructose in combination with glucose resulted in $\sim 55\%$ higher exogenous CHO oxidation rates compared with ingestion of an isoenergetic amount of glucose only. Of note, in a study by Shi et al. (31), it was demonstrated that a beverage

containing glucose and fructose resulted in $\sim 65\%$ higher CHO absorption rates compared with an isoenergetic glucose solution. This finding was attributed to the fact that glucose and fructose are absorbed by different intestinal transport mechanisms, and hence there may be less competition for absorption. A faster rate of intestinal CHO absorption might increase the availability of exogenous CHO in the bloodstream, and this could explain the higher exogenous CHO oxidation rates observed when glucose and fructose are ingested simultaneously (1, 16).

There have been suggestions that glucose, in combination with sucrose, also results in high rates of CHO (and water) absorption (31). Sucrose is hydrolyzed at the brush border of the intestinal epithelium to glucose and fructose. It has been postulated that monosaccharides from the hydrolysis of disaccharides are directly transported through the brush-border membrane without being released in the luminal space, a process often referred to as disaccharidase-related transport (20, 25). However, several studies have failed to find evidence for a disaccharidase-related transport system to be involved in the absorption of sucrose (9, 29) or maltose (12, 28). It is more likely that fructose and glucose released from sucrose are absorbed by conventional monosaccharide transport mechanisms (9, 29). Fructose is absorbed from the intestine by GLUT-5 transporters (4, 10), and intestinal glucose transport occurs via a sodium-dependent glucose transporter (SGLT) 1 (10). This suggests that intestinal transport of sucrose is at least, in part, different from that of glucose. It is possible that, when a mixture of glucose and sucrose is ingested, the rate of intestinal CHO absorption is higher compared with the ingestion of an isoenergetic amount of glucose (or sucrose) (31), and this might increase the availability and oxidation of exogenous CHO. Ingested sucrose is oxidized at a peak oxidation rate of ~ 0.9 g/min (32), and this is fairly similar to the peak exogenous oxidation rate reported for glucose (17). The purpose of the present study was to investigate whether combined ingestion of a large amount of glucose and sucrose (270 g) during 2.5 h of exercise would lead to exogenous CHO oxidation rates of >1.0 g/min. The second aim of the study was to examine the rate of exogenous CHO oxidation of a mixture of glucose and maltose compared with an isoenergetic amount of glucose. Maltose is a disaccharide consisting of two glucose molecules linked by an α -1,4 glycosidic bond. Orally ingested maltose is hydrolyzed to glucose at the brush border of the intestinal epithelium. The absorption of glucose released from maltose seems to occur via the same intestinal CHO transporters as free

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glucose (SLGT1) (12, 28). It is likely that, when maltose and glucose are ingested simultaneously, the glucose released from maltose and free glucose will compete for intestinal CHO transport, and hence the rate of CHO absorption may not be different compared with the ingestion of an isoenergetic amount of glucose. Therefore, we hypothesized that combined ingestion of glucose and maltose during prolonged cycling exercise would result in similar exogenous CHO oxidation rates as the ingestion of an isoenergetic amount of glucose.

METHODS

Subjects. Nine trained male cyclists or triathletes, aged 27.3 ± 2.5 yr and with a body mass of 74.1 ± 1.9 kg, took part in this study. Subjects trained at least three times a week for >2 h/day and had been involved in endurance training for at least 2–4 yr. Before participation, each of the subjects was fully informed of the purpose and risks associated with the procedures, and a written, informed consent was obtained. All subjects were healthy, as assessed by a general health questionnaire. The study was approved by the Ethics Committee of the School of Sport and Exercise Sciences of the University of Birmingham, United Kingdom.

Preliminary testing. At least 1 wk before the start of the experimental trials, an incremental cycle exercise test to volitional exhaustion was performed to determine the individual maximum power output (\dot{W}_{\max}) and maximal oxygen consumption ($\dot{V}O_{2\max}$). This test was performed on an electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands), modified to the configuration of a racing bicycle with adjustable saddle height and handlebar position. After the subjects reported to the laboratory, body mass (Seca Alpha, Hamburg, Germany) and height were recorded. Subjects then started cycling at 95 W for 3 min, followed by incremental steps of 35 W every 3 min until exhaustion. Heart rate (HR) was recorded continuously by a radiotelemetry HR monitor (Polar Vantage NV, Kempele, Finland). \dot{W}_{\max} was calculated from the last completed work rate, plus the fraction of time spent in the final noncompleted work rate multiplied by the work rate increment. The results were used to determine the work rate corresponding to 50% \dot{W}_{\max} , which was later employed in the experimental exercise trials. Breath-by-breath measurements were performed throughout exercise by using an online automated gas-analysis system (Oxycon Pro, Jaeger, Wuerzberg, Germany). The volume sensor was calibrated by using a 3-liter calibration syringe, and the gas analyzers were calibrated by using a 5.03% CO_2 -94.97% N_2 gas mixture. Oxygen consumption ($\dot{V}O_2$) was considered to be maximal ($\dot{V}O_{2\max}$) when at least two of the three following criteria were met: 1) a leveling off of $\dot{V}O_2$ with increasing workload (increase of no more than $2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), 2) a HR within 10 beats/min of predicted maximum (HR 220 minus age), and 3) a respiratory exchange ratio (RER) of >1.05 . $\dot{V}O_{2\max}$ was calculated as the average oxygen uptake over the last 60 s of the test. The $\dot{V}O_{2\max}$ and \dot{W}_{\max} achieved during the incremental exercise test were $64 \pm 2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and $364 \pm 5 \text{ W}$, respectively.

Experimental design. Each subject performed four exercise trials, which consisted of 150 min of cycling at 50% \dot{W}_{\max} while ingesting a glucose drink (Glu), an isoenergetic glucose+sucrose drink (Glu+Suc) (the ingested glucose-to-sucrose ratio was 2:1), an isoenergetic glucose+maltose drink (Glu+Mal) (the ingested glucose-to-maltose ratio was 2:1), or plain water (Wat). To quantify exogenous glucose oxidation (EGO), corn-derived glucose monohydrate and maltose and sugar cane-derived sucrose were used, which have a high natural abundance of ^{13}C [-10.8 , -10.6 , and -11.2 ‰ change (δ) vs. Pee Dee Bellemnitella (PDB), respectively]. The ^{13}C enrichment of the ingested glucose, maltose, and sucrose was determined by elemental analyzer-isotope ratio mass spectrometry (Europa Scientific GEO 20–20, Crewe, UK). To all drinks, 20 mmol/l of sodium chloride

were added. The order of the experimental drinks was randomly assigned in a crossover design. Experimental trials were separated by at least 7 days.

Diet and activity before testing. Subjects were asked to record their food intake and activity pattern 2 days before the first exercise trial and were then instructed to follow the same diet and exercise activities before the other three trials. In addition, 5–7 days before each experimental testing day, they were asked to perform an intense training session (“glycogen-depleting” exercise bout) in an attempt to empty any ^{13}C -enriched glycogen stores. Subjects were further instructed not to consume any food products with a high natural abundance of ^{13}C (CHO derived from C4 plants: maize, sugar cane) at least 1 wk before and during the entire experimental period to reduce the background shift (change in $^{13}CO_2$) from endogenous substrate stores.

Protocol. Subjects reported to the Human Performance Laboratory in the morning (between 7:00–9:00 AM) after an overnight fast (10–12 h) and having refrained from any strenuous activity or drinking any alcohol in the previous 24 h. For a given subject, all trials were conducted at the same time of the day to avoid any influence of circadian variance. On arrival in the laboratory, a flexible 21-gauge Teflon catheter (Quickcath, Baxter BV, Norfolk, UK) was inserted in an antecubital vein of an arm and attached to a three-way stopcock (Sims Portex, Kingsmead, UK) to allow for repeated blood sampling during exercise. The catheter was kept patent by flushing with 1.0–1.5 ml of isotonic saline (0.9%; Baxter) after each blood sample collection.

The subjects then mounted a cycle ergometer, and a resting breath sample was collected in 10-ml Exetainer tubes (Labco Brow Works, High Wycombe, UK), which were filled directly from a mixing chamber in duplicate to determine the ^{13}C -to- ^{12}C ratio ($^{13}C/^{12}C$) in the expired air.

Next, a resting blood sample (10 ml) was taken and stored on ice and later centrifuged. Subjects then started a 150-min exercise bout at a work rate equivalent to 50% \dot{W}_{\max} ($60 \pm 1\% \dot{V}O_{2\max}$). Additional blood samples were drawn at 15-min intervals during exercise. Expiratory breath samples were collected every 15 min until the end of exercise. $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$), and RER were measured every 15 min for periods of 4 min by using an online automated gas-analysis system, as previously described.

During the first 3 min of exercise, subjects drank an initial bolus (600 ml) of one of the four experimental drinks: Glu, Glu+Suc, Glu+Mal, or Wat. Thereafter, every 15 min, a beverage volume of 150 ml was provided. The total fluid provided during the 150-min exercise bout was 1.95 liters. The average rate of glucose intake in the Glu, Glu+Suc, and Glu+Mal trial was 1.8, 1.2, and 1.2 g/min, respectively. Furthermore, subjects ingested, on average, 0.6 g/min of sucrose in the Glu+Suc trial and 0.6 g/min of maltose in the Glu+Mal trial, which brought the total CHO intake rate in each condition to 1.8 g/min.

Subjects were asked to rate their perceived exertion (RPE) for whole body and legs every 15 min on a scale from 6 to 20, using the Borg category scale (2). In addition, subjects were asked every 30 min to fill in a questionnaire to rate possible gastrointestinal (GI) problems (15). All exercise tests were performed under normal and standard environmental conditions (19–22°C dry bulb temperature and 50–60% relative humidity). During the exercise trials, subjects were cooled with standing floor fans to minimize thermal stress.

Questionnaires. Subjects were asked to fill out a questionnaire every 30 min during the exercise trials. The questionnaire contained questions regarding the presence of GI problems at that moment and addressed the following complaints: stomach problems, GI cramping, bloated feeling, diarrhea, nausea, dizziness, headache, belching, vomiting, and urge to urinate and defecate. While subjects were on the bike and continued their exercise, each question was answered by simply ticking a box on the questionnaire that corresponded to the severity of the GI problem addressed. The items were scored on a

10-point scale (1 = not at all, 10 = very, very much). The severity of the GI symptoms was divided into two categories, severe and nonsevere symptoms, as was previously described by Jentjens et al. (15). Severe complaints included nausea, stomach problems, bloated feeling, diarrhea, urge to vomit, and stomach and intestinal cramps, because these are symptoms that commonly impair performance and may bring with them health risks. The above symptoms were only registered as severe symptoms when a score of ≥ 5 out of 10 was reported. When a score of < 5 was given, they were registered as nonsevere. All other symptoms were registered as nonsevere, regardless of the score reported.

Analyses. Blood samples were collected into prechilled EDTA-containing tubes (Beckton Dickinson, Plymouth, UK) and centrifuged at 2300 g and 4°C for 10 min. Aliquots of plasma were immediately frozen in liquid nitrogen and stored at -25°C until analyses for glucose and lactate. Glucose (glucose HK kit, Sigma-Aldrich, Dorset, UK) and lactate (lactate kit, Sigma-Aldrich) were analyzed on a COBAS BIO semiautomatic analyzer (La Roche, Basel, Switzerland).

Breath samples were analyzed for $^{13}\text{C}/^{12}\text{C}$ by gas chromatography continuous flow isotope ratio mass spectrometry (Europa Scientific, Crewe, UK). From indirect calorimetry ($\dot{V}\text{O}_2$ and $\dot{V}\text{CO}_2$) and stable isotope measurements (breath $^{13}\text{CO}_2/^{12}\text{CO}_2$), oxidation rates of total fat, total CHO, and exogenous glucose were calculated.

Calculations. From $\dot{V}\text{CO}_2$ and $\dot{V}\text{O}_2$ (l/min), total CHO and fat oxidation rates (g/min) were calculated by using the stoichiometric equations of Frayn (11), with the assumption that protein oxidation during exercise was negligible

$$\text{CHO oxidation} = 4.55 \dot{V}\text{CO}_2 - 3.21 \dot{V}\text{O}_2 \quad (1)$$

$$\text{Fat oxidation} = 1.67 \dot{V}\text{O}_2 - 1.67 \dot{V}\text{CO}_2 \quad (2)$$

The isotopic enrichment was expressed as $\delta\%$ difference between the $^{13}\text{C}/^{12}\text{C}$ of the sample and a known laboratory reference standard, according to the formula of Craig (8)

$$\delta^{13}\text{C} = \left[\left(\frac{^{13}\text{C}/^{12}\text{C} \text{ sample}}{^{13}\text{C}/^{12}\text{C} \text{ standard}} \right) - 1 \right] \cdot 10^3\% \quad (3)$$

The $\delta^{13}\text{C}$ was then related to an international standard (PDB).

In the CHO trials, the rate of EGO was calculated by using the following formula (24)

$$\text{Exogenous glucose oxidation} = \dot{V}\text{CO}_2 \cdot \left(\frac{\delta \text{ Exp} - \delta \text{ Exp}_{\text{bkg}}}{\delta \text{ Ing} - \delta \text{ Exp}_{\text{bkg}}} \right) \left(\frac{1}{k} \right) \quad (4)$$

where $\delta \text{ Exp}$ is the ^{13}C enrichment of expired air during exercise at different time points, $\delta \text{ Ing}$ is the ^{13}C enrichment of the ingested CHO solution, $\delta \text{ Exp}_{\text{bkg}}$ is the ^{13}C enrichment of expired air in the Wat trial (background) at different time points, and k is the amount of CO_2 (in liters) produced by the oxidation of 1 g of glucose ($k = 0.7467$ liter of CO_2 per gram of glucose).

Endogenous CHO oxidation was calculated by subtracting exogenous CHO oxidation from total CHO oxidation.

A methodological consideration when $^{13}\text{CO}_2$ is used in expired air to calculate exogenous substrate oxidation is the trapping of $^{13}\text{CO}_2$ in the bicarbonate pool, in which an amount of CO_2 arising from decarboxylation of energy substrates is temporarily trapped (27). However, during exercise, the $\dot{V}\text{CO}_2$ increases severalfold, so that a physiological steady-state condition will occur relatively rapidly, and $^{13}\text{CO}_2$ in the expired air will be equilibrated with the $^{13}\text{CO}_2/\text{H}^{13}\text{CO}_3^-$ pool, respectively. Recovery of $^{13}\text{CO}_2$ from oxidation will approach 100% after 60 min of exercise when dilution in the bicarbonate pool becomes negligible (23, 27). As a consequence of this, all calculations on substrate oxidation were performed over the last 90 min of exercise (60–150 min).

Statistical analyses. Two-way ANOVA for repeated measures was used to compare differences in substrate utilization and in blood-related parameters over time between the trials. A Tukey post hoc test

was applied in the event of a significant F ratio. Data evaluation was performed by using SPSS for Windows version 10.0 software package (Chicago, IL). All data are reported as means \pm SE. Statistical significance was set at $P < 0.05$.

RESULTS

Stable isotope measurements. The mean $^{13}\text{CO}_2$ enrichment of the resting breath samples was $-26.01 \pm 0.22 \delta\%$ vs. PDB. Changes in isotopic composition of expired CO_2 in response to exercise with ingestion of Wat, Glu, Glu+Suc, or Glu+Mal are shown in Fig. 1A. In the three CHO trials, there was a significant increase ($P < 0.001$) in the $^{13}\text{CO}_2$ enrichment of expired breath, reaching an enrichment difference of ~ 5 – $6 \delta\%$ vs. PDB toward the end of the 150-min exercise (compared with corresponding resting breath sample). From the 60-min point onward, breath $^{13}\text{CO}_2$ enrichment in Glu+Suc was sig-

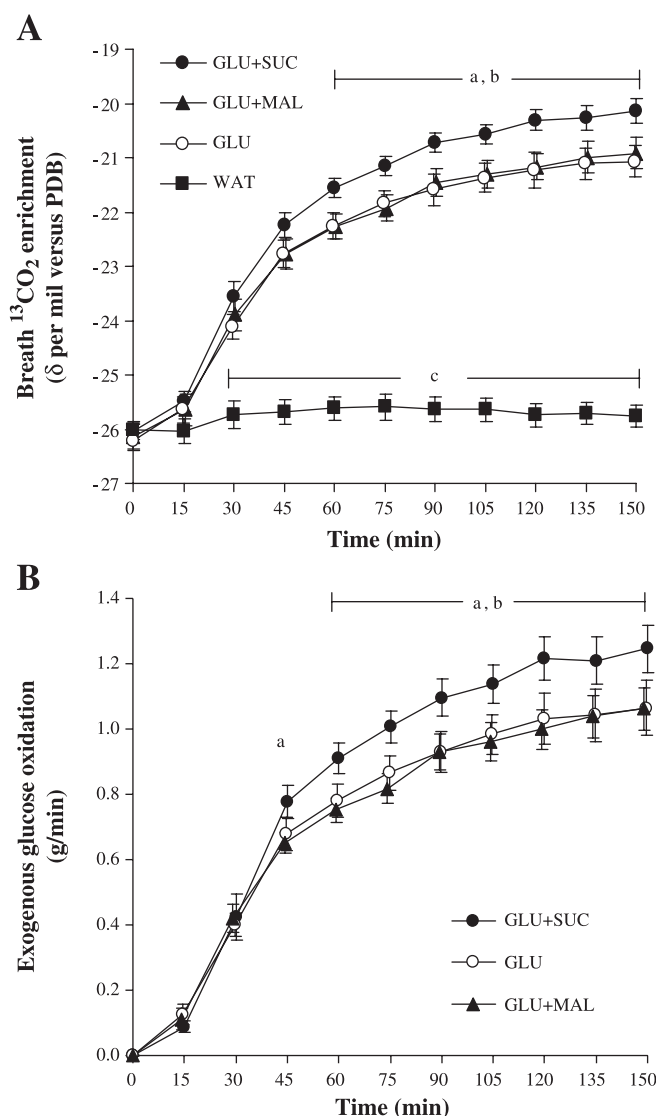


Fig. 1. Breath $^{13}\text{CO}_2$ enrichment (A) and exogenous carbohydrate (CHO) oxidation (B) during exercise without ingestion of carbohydrate [water (Wat)] or with ingestion of glucose (Glu), glucose and sucrose (Glu+Suc), or glucose and maltose (Glu+Mal). Values are means \pm SE; $n = 9$; PDB, Pee Dee Bellemintella. Significant differences: ^aGlu+Suc vs. Glu+Mal, ^bGlu+Suc vs. Glu, and ^cWat vs. CHO trials: $P < 0.05$.

Table 1. Oxygen uptake ($\dot{V}O_2$), respiratory exchange ratio (RER), total carbohydrate oxidation (CHO_{tot}), total fat oxidation (FAT_{tot}), endogenous carbohydrate (CHO) oxidation, and exogenous glucose oxidation during cycling exercise with ingestion of WAT, GLU, GLU + SUC, or GLU + MAL

	Time	$\dot{V}O_2$, l/min	RER, g/min	CHO _{tot} , g/min	FAT _{tot} , g/min	Endogenous CHO oxidation, g/min	Exogenous Glucose
WAT	60–90	2.84±0.06	0.82±0.01	1.51±0.15	0.84±0.06	1.51±0.15	
	90–120	2.84±0.05	0.81±0.01	1.41±0.15	0.88±0.05	1.41±0.15	
	120–150	2.89±0.05	0.81±0.01	1.36±0.16	0.92±0.05	1.36±0.16	
GLU	60–90	2.81±0.08	0.86±0.01 [†]	1.96±0.13 [†]	0.66±0.06*	1.10±0.10*	0.86±0.05
	90–120	2.82±0.08	0.86±0.01 [†]	1.95±0.13 [†]	0.67±0.07 [†]	0.97±0.09*	0.98±0.07
	120–150	2.82±0.08	0.86±0.01 [†]	1.93±0.15 [†]	0.68±0.07 [†]	0.88±0.10*	1.05±0.08
GLU+SUC	60–90	2.82±0.06	0.88±0.01 [†]	2.24±0.13 [†]	0.56±0.03 [†]	1.23±0.12	1.00±0.05 ^{‡,§}
	90–120	2.83±0.06	0.88±0.01 [†]	2.22±0.13 [†]	0.58±0.03 [†]	1.08±0.12*	1.15±0.06 ^{‡,§}
	120–150	2.85±0.06	0.87±0.01 [†]	2.20±0.13 [†]	0.60±0.03 [†]	0.97±0.13*	1.22±0.07 ^{‡,§}
GLU+MAL	60–90	2.84±0.06	0.86±0.01 [†]	2.02±0.11 [†]	0.66±0.03*	1.19±0.10	0.83±0.06
	90–120	2.83±0.06	0.86±0.01 [†]	2.01±0.11 [†]	0.66±0.03 [†]	1.05±0.10*	0.96±0.04
	120–150	2.85±0.06	0.86±0.01 [†]	1.99±0.11 [†]	0.67±0.03 [†]	0.96±0.10*	1.03±0.06

Values are means ± SE; *n* = 9. WAT, ingestion of water only; GLU, ingestion of glucose; GLU+SUC, ingestion of glucose and sucrose; GLU+MAL, ingestion of glucose and maltose. *Significantly different from WAT (*P* < 0.05). [†]Significantly different from WAT (*P* < 0.01). [‡]Significantly different from GLU (*P* < 0.05). [§]Significantly different from GLU+MAL (*P* < 0.01).

nificantly (*P* < 0.01) higher compared with that in the other two CHO trials. During the Wat trial, there was a small but significant increase in ¹³CO₂ enrichment of the expired air (*P* < 0.01). The changes in background ¹³CO₂ enrichment during the Wat trial were <7% compared with the rise in breath ¹³CO₂ enrichment provoked by the exogenous CHO in the Glu, Glu+Suc, and Glu+Mal trials. Although the background shift was relatively small in the present study, a background correction was made for the calculation of EGO in the three CHO trials by using the data from the Wat trial.

$\dot{V}O_2$, RER, total CHO, and fat oxidation. Data for $\dot{V}O_2$, RER, total CHO, and fat oxidation over the 60- to 150-min exercise period are shown in Table 1. No significant differences were observed in $\dot{V}O_2$ among the four experimental trials. RER in the Wat trial was significantly lower (*P* < 0.01) compared with that in the three CHO trials (Table 1). CHO oxidation was significantly higher (*P* < 0.01) after CHO ingestion compared with Wat ingestion. During the last 90 min of exercise (60–150 min), the average CHO oxidation rates were 1.43 ± 0.14 , 1.94 ± 0.14 , 2.21 ± 0.13 , and 2.00 ± 0.11 g/min for Wat, Glu, Glu+Suc, and Glu+Mal, respectively. No significant differences in total CHO oxidation were found among CHO trials. However, there was a trend (*P* = 0.07) for a higher total CHO oxidation in Glu+Suc compared with that in the other two CHO trials. Total fat oxidation was markedly suppressed with CHO ingestion (*P* < 0.01). The average fat oxidation rates over the 60- to 150-min exercise period were 0.88 ± 0.05 , 0.67 ± 0.07 , 0.58 ± 0.03 , and 0.66 ± 0.03 g/min for Wat, Glu, Glu+Suc, and Glu+Mal, respectively. The relative contribution of substrates to total energy expenditure during the 60- to 150-min period is depicted in Fig. 2.

Exogenous and endogenous CHO oxidation. Exogenous CHO oxidation rates gradually increased during the first 120 min of exercise and leveled off during the final 30 min of exercise (Fig. 1B). The average oxidation rates over the final 30-min exercise period were 1.05 ± 0.08 , 1.22 ± 0.07 , and 1.03 ± 0.06 g/min for Glu, Glu+Suc, and Glu+Mal, respectively (Table 1). Peak exogenous CHO oxidation rates were reached at the end of exercise (150 min) and were significantly higher (*P* < 0.05) in the Glu+Suc trial (1.25 ± 0.07 g/min

compared with the Glu and Glu+Mal trials (1.06 ± 0.08 and 1.06 ± 0.06 g/min, respectively) (Fig. 1B). Exogenous CHO oxidation in the Glu+Suc trial was ~18% higher (*P* < 0.05) compared with that in the isoenergetic Glu and Glu+Mal trials (Table 1 and Fig. 1B). No difference was found in (peak) exogenous CHO oxidation rates between Glu and Glu+Mal.

During the 60- to 150-min period of exercise, endogenous CHO oxidation was significantly lower (*P* < 0.05) in the CHO trials compared with the Wat trial, although Glu+Suc and Glu+Mal just failed to reach statistical significance between time = 60 and 90 min (*P* = 0.079) (Table 1 and Fig. 2). Furthermore, the contribution of endogenous CHO oxidation to total energy expenditure was significantly lower in the three CHO trials compared with Wat (Fig. 2). No differences were found in endogenous CHO oxidation among the three CHO trials.

Plasma metabolites. Plasma glucose and lactate concentrations at rest and during exercise are shown in Fig. 3, A and B, respectively. The fasting plasma glucose concentrations for the

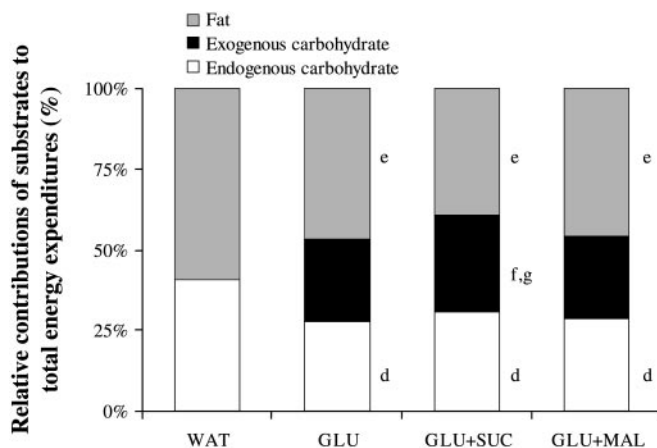


Fig. 2. Relative contributions of substrates to total energy expenditure calculated for the 60- to 150-min period of exercise without ingestion of Wat or with ingestion of Glu, Glu+Suc, or Glu+Mal. Values are means ± SE; *n* = 9. Significantly different from Wat (^d*P* < 0.05 and ^e*P* < 0.01), ^fGlu (*P* < 0.05), and ^gGlu+Mal (*P* < 0.01).

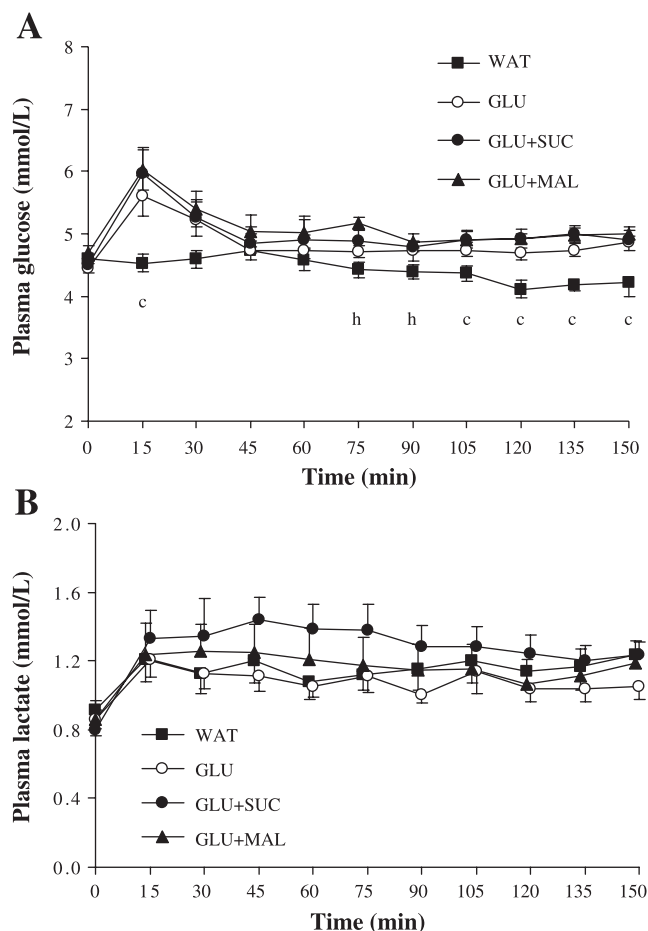


Fig. 3. Plasma glucose (A) and lactate (B) during exercise without ingestion of Wat or with ingestion of Glu, Glu+Suc, or Glu+Mal. Values are means \pm SE; $n = 9$. Significant difference: Wat vs. ^cCHO trials and ^hGlu+Mal: $P < 0.05$.

four experimental trials averaged between 4.5 and 4.7 mmol/l. Plasma glucose concentrations in the Wat trial remained relatively constant during exercise (values ranging from 4.1 to 4.7 mmol/l). With the ingestion of CHO at the start of exercise, plasma glucose concentrations peaked within the first 15 min of exercise at values ranging from 5.6 to 6.0 mmol/l. Plasma glucose concentrations then fell and were maintained at values of 4.7–5.0 mmol/l for the entire duration of exercise. During the final 45 min of exercise, plasma glucose concentrations were significantly higher ($P < 0.05$) in the CHO trials compared with the Wat trial. In addition, plasma glucose at time = 75 and 90 min was significantly higher ($P < 0.05$) in the Glu+Mal trial compared with the Wat trial.

Fasting plasma lactate concentrations were similar in the four trials (on average 0.9 ± 0.1 mmol/l). Plasma lactate concentrations increased ($P < 0.05$) during the first 15 min of exercise in all four trials. Thereafter, plasma lactate concentrations remained relatively constant until the end of exercise. No significant differences were observed in plasma lactate concentrations among the four experimental trials.

GI discomfort and RPEs. GI and related complaints were registered by a questionnaire. The results of the questionnaires obtained during the CHO trials are presented in Table 2. The most frequently reported complaints were stomach problems, nausea, flatulence, urge to urinate, belching, and bloated feel-

ing. More subjects reported severe GI discomfort (stomach problems, bloated feeling, and urge to vomit) in the Glu and Glu+Mal trials compared with the Glu+Suc trial. None of the subjects vomited or suffered from diarrhea during the exercise trails.

No significant differences in RPE overall or RPE in legs were observed among the four experimental trials. The mean values for RPE overall and RPE in legs during 150 min of exercise were 10.3 ± 0.6 and 10.5 ± 0.7 , respectively

DISCUSSION

Numerous studies have shown that, during prolonged cycling exercise, exogenous CHO oxidation does not exceed 1.0 g/min (3, 14, 19, 30, 32). However, it should be noted that, in the above-mentioned studies, the oxidation rate of only one type of exogenous CHO was examined at the time. Recently, our laboratory has shown that, when a mixture of glucose and fructose was ingested during cycling exercise, exogenous CHO oxidation rates reached peak values of almost 1.3 g/min (16). The present study investigated whether combined ingestion of Glu+Suc would result in exogenous CHO oxidation rates of >1.0 g/min. In addition, the effect of a Glu+Mal mixture on exogenous CHO oxidation was examined. The main finding of the present study was that a mixture of Glu+Suc, when ingested at a high rate (1.8 g/min), resulted in peak oxidation rates of ~ 1.25 g/min (Fig. 1B) and resulted in almost 20% higher exogenous CHO oxidation rates compared with the ingestion of an isocaloric amount of Glu (Fig. 1B and Table 1).

Previous studies have shown that, when the rate of glucose or maltodextrin ingestion is increased from 1.2 to 1.8 g/min, exogenous CHO oxidation rates do not increase, and oxidation rates seem to level off at ~ 1.0 g/min (16, 32). It has been suggested that intestinal glucose transporters (SGLT1) may

Table 2. Gastrointestinal and related complaints during 150 min of exercise ($n = 9$)

Complaint	GLU, n	GLU+SUC, n	GLU+MAL, n
<i>Nonsevere</i>			
Stomach problems	2	0	1
Nausea	1	0	2
Dizziness	0	0	0
Headache	1	0	0
Flatulence	2	2	2
Urge to urinate	3	4	5
Urge to defecate	0	0	1
Urge to vomit	0	0	0
Belching	5	3	4
Stomach burn	2	0	1
Bloated feeling	5	4	5
Stomach cramps	0	0	0
Intestinal cramps	0	0	0
Side aches left	0	0	0
Side aches right	0	0	0
Reflux	0	0	0
<i>Severe</i>			
Stomach problems	1	0	1
Nausea	1	0	0
Urge to vomit	1	0	1
Bloated feeling	0	0	0
Stomach cramps	0	0	0

Stomach cramps, urge to vomit, nausea, bloated feeling, and stomach problems were registered as "severe" when a score of 5 or higher (out of 10) was given.

become saturated when large amounts of glucose or glucose polymers are ingested (>1.2 g/min), and hence intestinal CHO absorption may be a limiting factor for exogenous CHO oxidation (16). Interestingly, in the present study, the average CHO ingestion rate was 1.8 g/min, and exogenous CHO oxidation rates in the Glu+Suc trial reached peak values of 1.25 ± 0.07 g/min. The present findings are in agreement with the results of a previous study from our laboratory, in which combined ingestion of glucose and fructose (average ingestion rate of 1.8 g/min) resulted in peak exogenous CHO oxidation rates of ~ 1.26 g/min (16). It should be noted that free fructose and most probably fructose released from sucrose use a different intestinal transporter (GLUT-5) than glucose (SGLT1). Shi et al. (31) demonstrated, with an intestinal perfusion study, that a beverage containing glucose and fructose leads to higher CHO absorption rates compared with a beverage containing an isoenergetic amount of glucose. This effect was attributed to the separate intestinal transport mechanisms for glucose and fructose. The absorption of glucose and fructose released from sucrose most likely occurs via the same intestinal CHO transporters as free glucose (SGLT1) and free fructose (GLUT-5) (9, 29). However, it cannot be excluded that sucrose is absorbed by a disaccharidase-related transport mechanism, which provides a direct transfer of glucose and fructose (released from sucrose) across the brush border membrane (20, 25). Although speculative, the higher oxidation rates in the Glu+Suc trial compared with the Glu trial may have been due to an increased intestinal CHO absorption rate, which may have increased the availability of exogenous CHO for oxidation. It is interesting to note that fewer subjects reported (severe) GI discomfort when a mixture of glucose and sucrose (or fructose, see Ref. 16) was ingested compared with ingestion of an isoenergetic amount of glucose. Suggestions have been made that incomplete absorption of CHO increases the risk of GI complications (26). The lower prevalence of (severe) GI complications when mixtures of glucose and sucrose or fructose are ingested seems to suggest that less CHO is accumulating in the GI tract, possibly because of a faster CHO absorption rate. Although the mechanism remains speculative, the present data and previous observations from our laboratory (16) clearly show that ingestion of glucose in combination with sucrose or fructose increases the rate of exogenous CHO oxidation by 20–50% and results in peak oxidation rates of ~ 1.25 g/min.

Exogenous CHO oxidation rates were similar when a mixture of Glu+Mal was ingested compared with the ingestion of an isoenergetic amount of Glu (Table 1 and Fig. 1B). As mentioned earlier, when large amounts of CHOs are ingested during exercise, the rate of intestinal CHO absorption may be a limiting factor for exogenous CHO oxidation. Maltose is hydrolyzed by a specific brush-border enzyme to two glucose molecules, and suggestions have been made that these glucoses are then rapidly absorbed by SGLT1 (12, 28). Because free glucose and glucose released from maltose appear to be absorbed via the same intestinal CHO transporter (SGLT1) (12, 28), glucose and maltose will compete for CHO transport when ingested in the same beverage. It is, therefore, likely that CHO absorption rates in Glu and Glu+Mal were the same, and hence this could explain the similar exogenous CHO oxidation rates. Interestingly, human duodenal perfusion studies have shown that glucose absorption from hydrolysis of maltose is

faster than from ingested glucose (5, 28). However, it should be noted that, in these studies, CHO perfusion rates were relatively low (<0.75 g/min) (5, 28). Although speculative, when larger amounts of glucose or maltose are perfused (>1.2 g/min), SGLT1 transports may become saturated, and hence intestinal CHO absorption rates of glucose and maltose may be similar. To our knowledge, only one study has investigated the oxidation of ingested maltose during exercise. In a study by Hawley et al. (14), six trained cyclists ingested 180 g of maltose or glucose during 90 min of cycling exercise at 70% $\dot{V}O_{2\max}$. It was shown that maltose and glucose are oxidized at similar rates, and peak oxidation rates for maltose and glucose were 0.9 and 1.0 g/min, respectively. The findings of Hawley et al. and those of the present study suggest that maltose and glucose are equally effective for exogenous CHO oxidation.

In the present study, ingestion of CHO resulted in lower endogenous CHO oxidation rates compared with the ingestion of Wat (Table 1). The contribution of endogenous CHO oxidation to total energy expenditure was 41 ± 4 , 28 ± 3 , 31 ± 3 , and $29 \pm 3\%$ for Wat, Glu, Glu+Suc, and Glu+Mal, respectively ($P < 0.05$) (Fig. 2). A decreased endogenous CHO oxidation with CHO ingestion during exercise has been found in several other studies (6, 21, 22, 32) and is most likely due to a reduced or complete absence of hepatic glucose oxidation (19). Interestingly, the higher exogenous CHO oxidation rate in the Glu+Suc trial compared with the Glu and Glu+Mal trials did not lead to a further reduction in the oxidation rate of endogenous CHO. It should be noted that there was a trend ($P = 0.07$) for a higher total CHO oxidation in the Glu+Suc trial compared with the other two CHO trials. These data suggest that the higher exogenous CHO oxidation in Glu+Suc may have contributed to a somewhat higher total CHO oxidation. High rates of CHO oxidation and the maintenance of high blood glucose concentrations toward the end of prolonged exercise have been associated with the improvements in performance observed when CHO is ingested during exercise (7). It remains to be investigated whether ingestion of a mixture of Glu+Suc during prolonged exercise will lead to an improved exercise performance compared with the ingestion of an isocaloric amount of Glu.

In summary, when a mixture of Glu+Suc was ingested at high rates (1.8 g/min) during cycling exercise, exogenous CHO oxidation rates reached peak values of ~ 1.25 g/min. Furthermore, combined ingestion of Glu+Suc resulted in almost 20% higher exogenous CHO oxidation rates compared with the ingestion of an isocaloric amount of Glu.

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