Substrate utilization during exercise with glucose and glucose plus fructose ingestion in boys ages 10–14 yr

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Riddell, M. C., O. Bar-Or, B. Wilk, M. L. Parolin, and G. J. F. Heigenhauser. Substrate utilization during exercise with glucose and glucose plus fructose ingestion in boys ages 10–14 yr. J Appl Physiol 90: 903–911, 2001.—We measured substrate utilization during exercise performed with water (W), exogenous glucose (G), and exogenous fructose plus glucose (FG) ingestion in boys age 10–14 yr. Subjects (n = 12) cycled for 90 min at 55% maximal O2 uptake while ingesting either W (25 ml/kg), 6% G (1.5 g/kg), or 3% F plus 3% G (1.5 g/kg). Fat oxidation increased during exercise in all ingesting either W (25 ml/kg), 6% G (1.5 g/kg), or 3% F plus 3% G (1.5 g/kg). Fat oxidation increased during exercise in all trials but was higher in the W (0.28 ± 0.23 g/min) than in the G (0.24 ± 0.023 g/min) and FG (0.25 ± 0.029 g/min) trials (P = 0.04). Conversely, total carbohydrate (CHO) oxidation decreased in all trials and was lower in the W (0.63 ± 0.05 g/min) than in the G (0.78 ± 0.051 g/min) and FG (0.74 ± 0.056 g/min) trials (P = 0.009). Exogenous CHO oxidation, as determined by expired 13CO2, reached a maximum of 0.36 ± 0.032 and 0.31 ± 0.030 g/min at 90 min in G and FG, respectively (P = 0.04). Plasma insulin levels decreased during exercise in all trials but were twofold higher in G than in W and FG (P < 0.001). Plasma glucose levels decreased transiently after the onset of exercise in all trials and then returned to preexercise values in the W and FG (~4.5 mmol/l) trials but were elevated by ~1.0 mmol/l in the G trial (P < 0.001). Plasma lactate concentrations decreased after the onset of exercise in all trials but were lower by ~0.5 mmol/l in W than in G and FG (P = 0.02). Thus, in boys exercising at a moderate intensity, the oxidation rate of G plus F is slightly less than G alone, but both spare endogenous CHO and fat to a similar extent. In addition, compared with flavored W, the ingestion of G alone and of G plus F delays exhaustion at 90% peak power by ~25 and 40%, respectively, after 90 min of moderate-intensity exercise.

IN ADULTS, EXOGENOUS CARBOHYDRATE (CHOexo) ingestion just before, or during, exercise improves performance and delays fatigue (5). CHOexo is thought to limit fatigue by maintaining high rates of total carbohydrate (CHOtot) oxidation, sparing endogenous glycogen stores, and elevating blood glucose concentrations late in exercise (4, 30). With the use of 13C labeling, the oxidation rates of a variety of CHOexo sources (e.g., glucose and glucose polymers, fructose, sucrose, malto-}

cx dextrins, and cornstarch) have been reported for various exercise intensities [see Pérnonet et al. (33) for a review]. The most common form of CHOexo investigated is exogenous glucose (G), which has been reported to be oxidized at a peak rate of ~1.0–1.2 g/min during prolonged, high-intensity (~60–70% peak O2 uptake [V̇O2peak]) exercise (14, 41). On average, glucose oxidation provides somewhere between 7 and 9% of total energy provision during 120 min of moderate- to high-intensity exercise (24) and appears to be the main substrate utilized during the later stages of endurance exercise (26, 27).

In contrast to G during exercise, exogenous fructose (F) has a delayed rate of intestinal absorption (35) and often causes gastrointestinal distress during exercise (29). In addition, compared with G, F has a lower rate of oxidation during exercise (13, 17, 25, 26), possibly as a result of its slower absorption rate and the necessity for its conversion to glucose by the liver before oxidation (17). The combination of F and G (FG), however, is well absorbed during exercise (12) and may facilitate a higher oxidation rate than either of the two monosaccharides ingested separately (1). The reason for the higher rate of oxidation during exercise after FG ingestion is currently unknown, but it may be related to enhanced intestinal absorption through the activation of additional transport mechanisms (7, 12, 36).

Less is known about CHOexo utilization during exercise in children and adolescents. During exercise performed in a fasting state, children seem to have a lower respiratory exchange ratio (RER) than adults, indicating that they oxidize more endogenous fat and less carbohydrate (CHO) (21–23). In addition, preexercise CHO snacks (i.e., fig or candy bar) do not appear to alter RER or increase exercise time to exhaustion in adolescent boys (15). In contrast, our laboratory has shown that intermittent feedings of glucose (~1.4 g glucose·kg body mass−1·h−1) during exercise increase blood glucose and insulin levels while suppressing plasma free fatty acid and glycerol release in healthy adolescent boys (38). In addition, by using 13C-labeled glucose, our laboratory found that this feeding pattern reduces endogenous CHO (CHOendo) and lipid utiliza-

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tion, lowers the rating of perceived exertion (RPE) (38a) and can contribute up to 25% of the total energy provision during prolonged exercise in healthy adolescent boys (37, 38).

Although G and F are commonly found in natural foods and sport beverages and are frequently consumed by youth, no studies exist that have examined substrate utilization or performance during exercise performed with FG intake in this segment of the population. The primary purpose of this study, therefore, was to compare the oxidation rates of G with FG during prolonged exercise in healthy, untrained adolescent boys. On the basis of the previous observations in adults (1), we hypothesized that FG oxidation would be higher than G oxidation in adolescent boys. In addition, we also investigated endogenous substrate sparing, metabolic responses, and exercise performance during exercise in these subjects when they ingested W, G, or FG.

METHODS

Subjects. Twelve 11- to 14-yr-old boys volunteered through local public service announcements. Table 1 shows their anthropometric and functional characteristics. All subjects were healthy, nonobese, and habitually active but were not competitive athletes. The purpose, nature, and possible risks of the experiment were explained to the boys and their parents. Subjects gave a verbal agreement to participate, and a parent then signed an informed consent. The Research Ethics Board of the Faculty of Health Sciences, McMaster University, approved the study.

Pretesting. Height, weight, percent body fat by bioimpedance, and V\textsuperscript{O2 peak} were measured during a preliminary session. V\textsuperscript{O2 peak} was determined for each subject during an “all-out” progressive exercise test on an electronically braked cycle ergometer (Corival 400, Lode, Gronigen, Netherlands), each stage lasting 2 min (2). Measurements of O\textsubscript{2} uptake (V\textsuperscript{O2}) and CO\textsubscript{2} production (V\textsuperscript{CO2}) were made continuously using a Quinton metabolic cart (Quinton Q-plex 1, Quinton Instrument, Seattle, WA) and averaged over the final 30 s of each workload. V\textsuperscript{O2 peak} was considered to have been reached if at least two of the following criteria were met: heart rate (HR) within 10 beats/min of age-predicted maximum, RER 1.0, and leveling of VO\textsubscript{2} with increasing intensity or volitional exhaustion (subject unable to maintain cadence above 60 rpm for 5 consecutive seconds, despite encouragement by the investigator).

To determine the relationship between VO\textsubscript{2} and power output during the test, a least squares regression curve was generated using Statistica for Windows (StatSoft, Tulsa, OK), taking the average VO\textsubscript{2} during the final 30 s of each stage for each power output. The corresponding power output for 55% V\textsuperscript{O2 peak} was determined from this curve. Peak power was determined as the final power output generated during the last 2-min stage of the test. Partial completion of the final stage was credited using the method of Bar-Or (2). HR was measured throughout the test using a Sports Tester PE3000 system (Polar Electro, Kempele, Finland).

Experimental trials. Each subject attended three experimental trials spaced 1–2 wk apart. Trials were identical except for postbreakfast fluid and CHO intake, and the order was counterbalanced among the subjects. Subjects were blinded to the content of drinks in each trial. During the trials, subjects drank either water (W trial), 6% glucose (G trial), or 3% glucose plus 3% fructose (FG trial) beverages intermittently for a total of 25 ml/kg body mass. All three beverages were grape flavored and contained 18 mmol/l NaCl. The W was also artificially sweetened with Aspartane. Each experimental trial consisted of three 30-min bouts of cycling at 55% of their predicted V\textsuperscript{O2 peak}, separated by 5-min rest periods. After the last bout, a 10-min rest was provided that was followed by an all-out performance ride to volitional exhaustion at 90% of their predetermined peak power. Exhaustion was considered to have been achieved when the subject could no longer maintain a cycling cadence of 60 rpm for 10 s despite encouragement from an investigator.

Protocol. Subjects were asked to eat their usual meals but refrain from consuming corn and corn-derived food items during the study period to reduce the amount of naturally enriched [13C]glycogen in muscle and liver. In addition, they were asked to avoid excessive physical activity the day before the trial. They arrived fasted to the laboratory on the morning of the trial (~0800). Breakfast was provided by the investigators and consisted of one slice of white bread, toasted, with one-half tablespoon peanut butter and 100 ml orange juice. After breakfast, an indwelling venous catheter was inserted into an arm vein for subsequent blood sampling. The start of the first bout of exercise (time = 0 min) on the cycle ergometer occurred ~90 min after the start of breakfast. Subjects were instructed to ingest the provided beverages within 30 s at 30 and 15 min before the start of the first exercise bout and at 0, 15, and 30 min during each bout (for a total of 9 times). The glucose in the G and FG trials was derived from corn (BDH-Chemical, Toronto, ON) and artificially enriched with uniformly labeled [13C]glucose (99 atom %excess, Isotec, Mi-

Table 1. Subjects’ physical and functional characteristics

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>Body Fat, %</th>
<th>V\textsuperscript{O2 peak}, ml·kg\textsuperscript{-1}·min\textsuperscript{-1}</th>
<th>Maximum Power, W</th>
<th>Tanner Stage</th>
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<td>17.0</td>
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<td>143.0</td>
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<td>67.2</td>
<td>22.0</td>
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<tr>
<td>Mean ± SD</td>
<td>12.5 ± 1.11</td>
<td>152.7 ± 8.73</td>
<td>44.9 ± 10.29</td>
<td>17.2 ± 4.47</td>
<td>44.8 ± 4.21</td>
<td>136.7 ± 34.10</td>
<td>2.8 ± 0.83</td>
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</table>

V\textsuperscript{O2 peak} = peak O\textsubscript{2} uptake.
amisburg, OH) to an isotopic composition of +47.729 delta per 1,000 difference (%) vs. the 13C-to-12C ratio from the international standard 13C Pee Dee Belemninitella-1 (PDB-1; i.e., +47.729 %δ[8-13C]PDB-1), as measured by dual-inlet isotope ratio mass spectrometry (VG-Sira 10, series II, Manchester, UK). The F in the FG beverage was derived from corn and enriched with uniformly labeled [13C]fructose (99 atom %excess, Isotec, Miamisburg, OH) to an isotopic composition of +48.011 %δ[5-13C]PDB-1. The resulting enrichment of the FG beverage was +47.888 %δ[8-13C]PDB-1. These high levels of enrichment, compared with that of normal expired gas (Fig. 1), provide a strong measurement signal and reduce the error associated with a small shift in the isotopic composition of CO₂ arising from the oxidation of endogenous substrates during exercise (24).

Respiratory gas and substrate oxidation. Resting Vo₂ and VCO₂ were determined with subjects breathing into a mouth piece connected to the metabolic cart while sitting quietly in a chair during a 5-min collection period at –40 min. In addition, Vo₂ and VCO₂ were determined during exercise from 5-min sampling periods at 5 and 25 min in each bout. CHOtot and total fat (fattot) oxidation rates were calculated at each time point from RER and Vo₂ averaged over the collection period using a table of nonprotein respiratory quotients (34). During gas sampling, 10-ml expired gas samples were drawn from Douglas bags connected to the exhaust port of the metabolic cart and stored in vacutainer tubes for subsequent determination of 13C/12C in expired CO₂ and CHOexo oxidation. The isotopic composition of CO₂ in expired gas samples was determined using an isotopic ratio mass spectrometer (BreathMat Plus, Finnigan MAT, Bremen, Germany) and expressed in %δ[8-13C]PDB-1.

CHOexo oxidation was calculated for the sampling periods using the Mosora et al. (28) formula

\[ \text{CHOexo} = \frac{V_{\text{CO2}}(R_{\text{exp}} - R_{\text{ref}})}{(R_{\text{exo}} - R_{\text{ref}})} \]

where VCO₂ is in liters per minute STPD, Rexp is the isotopic composition of expired CO₂ during exercise, Rexo is the isotopic composition of expired CO₂ at rest before CHOexo ingestion, and Rref is the isotopic composition of the CHOexo beverage, and k (0.7426 l/g) is the volume of CO₂ provided by the complete oxidation of glucose (34). This method of determining CHOexo oxidation assumes that 13CO₂ recovery in expired gas during exercise is complete or almost complete (6, 16, 19), although there is a delay in recovery due to the large labile bicarbonate pool (31). This delay in 13CO₂ recovery appears to be less in children, however (43). In North American studies, this method has been shown to overestimate the actual exogenous substrate oxidation, because of shifts in the isotopic composition of endogenous substrates caused by exercise (24). This methodological limitation can be avoided, as in this study protocol, if CHOexo enrichment is several magnitudes higher than the natural 13C enrichment of foods found in North American diets (32). Finally, CHOendo was determined from the following equation

\[ \text{CHOendo} = \text{CHOtot} - \text{CHOexo} \]

Blood variables Ten of the twelve subjects volunteered to give venous blood samples during the trials. From these volunteers, whole blood samples were drawn from an indwelling catheter inserted into a forearm vein into heparinized syringes at –40 min (baseline) and at 0, 2, 5, 10, 20, 30, 65, 90, and 100 min, as well as immediately after the performance test. Each sample was centrifuged at 15,900 g for 2 min, and the plasma supernatant was stored at –20°C and subsequently analyzed for glucose and lactate (model 2300 Select Analyzer, Yellow Springs Instruments, Yellow Springs, OH) and insulin (Coat-A-Count radioimmunoassay, DPC Diagnostics) concentrations.

HR, RPE, and stomach fullness scale. HR was monitored continuously throughout the VO₂peak test and the experimental trials using a Sports Tester PE3000 system (Polar Electro, Kempele, Finland). During the experimental trials, 30-s HR averages were determined at rest immediately before the ingestion of the first dose of CHOexo and at 5 and 25 min in each bout. Before exercise, Borg’s 6–20 RPE category scale was used to rate whole body perceived exertion at 5 and 25 min in each bout. Stomach fullness was also assessed at 5 and 25 min in each bout using a five-point category scale that included the following categories: 1) not full at all, 2) somewhat full, 3) full, 4) very full, and 5) extremely full.

Statistical analyses. Data are presented as means ± SE. For measurements taken repeatedly during the trials, a two-way ANOVA was used. Tukey’s honest significant difference post hoc test for equal cell size was used to determine significance among mean values. Comparisons between performance times among the trials were also made using the Wilcoxon matched-pairs test. Significance was set at P < 0.05 for all statistical tests.

RESULTS

Two subjects were unable to complete the third bout of exercise during the W trial. All subjects completed the three exercise bouts in G and FG trials. For measurement taken during all three trials (i.e., repeated-measures ANOVA), statistical operations were performed for the 10 subjects who completed them. For visual comparisons, figures and tables are shown, however, with n = 12 subjects, except for the measurements made in the final bout of the W trial.

Subjects successfully consumed the provided beverages in the allotted time periods and did not complain of gastric upset. CHOexo intake was identical during
the G and FG trials, averaging 67.3 ± 15.4 g in each trial.

Preexercise. Resting HR (Fig. 2, top) and RER (Fig. 2, bottom); plasma insulin, glucose, and lactate (see Fig. 5); and expired \( ^{13} \text{CO}_2 \) \( \delta \) PDB-1 (Fig. 1, top) values were similar among the trials, averaging 82 ± 3 beats/min, 0.85 ± 0.001, and -23.7 ± 0.41 \( \% \delta^{13} \text{C} \)PDB-1, respectively.

Prolonged exercise. Work rate was identical during exercise during all time points and in all three trials, averaging 50 ± 4.48 W. \( \dot{V}_{O_2} \), \( \dot{V}_{CO_2} \), and expired minute ventilation (\( \dot{V}_E \)) during exercise are given in Table 2. No differences in \( \dot{V}_{O_2} \), \( \dot{V}_{CO_2} \), or \( \dot{V}_E \) were found either between trials or among time points. On average, subjects exercised at 53 ± 1.17% \( \dot{V}_{O_2} \text{peak} \) during the trials.

Expired \( \delta^{13} \text{C} \)/PDB values during the trials are shown in Fig. 1, top. During the W trial, expired \( ^{13} \text{CO}_2 \) increased slightly, but significantly, from -23.52 ± 0.325 \( \% \delta^{13} \text{C} \)PDB-1 at rest to a maximum of -22.23 ± 0.282 \( \% \delta^{13} \text{C} \)PDB-1 at 60 min and then decreased slightly, but significantly, to -22.48 ± 0.231 \( \% \delta^{13} \text{C} \)PDB-1 of exercise [time effect; \( F = 8.81; P < 0.001 \)]. In the G and FG trials, expired \( ^{13} \text{CO}_2 \) values were -23.89 ± 0.416 \( \% \delta^{13} \text{C} \)PDB-1 and -23.66 ± 0.500 \( \% \delta^{13} \text{C} \)PDB-1 at rest (not significantly different), increasing to -3.51 ± 0.836 and -5.26 ± 0.939 \( \% \delta^{13} \text{C} \)PDB-1 at 90 min of exercise (not significantly different), respectively.

\( \dot{CHO}_{exo} \) oxidation rates during G and FG are shown in Fig. 1, bottom. \( \dot{CHO}_{exo} \) oxidation rates were similar between trials at 10 min of exercise and increased throughout exercise to maximal rates of 0.36 ± 0.032 and 0.31 ± 0.030 g/min at 90 min of exercise in the G and FG trials, respectively (trial-by-time interaction; \( F = 2.45; \text{df} = 5.55; P = 0.04 \)). \( \dot{CHO}_{exo} \) oxidation during the entire 90 min of exercise (i.e., area under the curves in Fig. 1) averaged 0.24 ± 0.020 and 0.22 ± 0.022 g/min in the G and FG trials, respectively (group effect; \( F = 2.05; \text{df} = 1.11; P = 0.18 \)).

HR levels are shown in Fig. 2, top. HR increased with exercise in all trials [time effect; \( F = 5.07; \text{df} = 5.45; P < 0.001 \)] and was higher in the W trial than in W and FG trials (trial effect; \( F = 6.83; \text{df} = 2.18; P = 0.01 \)). RER values as shown in Fig. 2, top, decreased in all trials (time effect; \( F = 35.14; \text{df} = 5.45; P < 0.001 \)) and were lower in W than in G and FG (trial effect; \( F = 6.03; \text{df} = 2.18; P = 0.010 \)).

**Table 2.** \( \dot{V}_{O_2} \), \( \dot{V}_{CO_2} \), and \( \dot{V}_E \) during the water trial, glucose, and glucose plus fructose trials

<table>
<thead>
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<th>5–10 min</th>
<th>25–30 min</th>
<th>5–10 min</th>
<th>25–30 min</th>
<th>5–10 min</th>
<th>25–30 min</th>
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</thead>
<tbody>
<tr>
<td>( \dot{V}_{O_2} )</td>
<td>( W )</td>
<td>1.06 ± 0.068</td>
<td>1.05 ± 0.065</td>
<td>1.02 ± 0.068</td>
<td>1.03 ± 0.065</td>
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<td>( G )</td>
<td>1.04 ± 0.077</td>
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<td>( FG )</td>
<td>1.05 ± 0.072</td>
<td>1.06 ± 0.071</td>
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<tr>
<td>( \dot{V}_{CO_2} )</td>
<td>( W )</td>
<td>0.92 ± 0.058</td>
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<td>( G )</td>
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<tr>
<td></td>
<td>( FG )</td>
<td>0.94 ± 0.067</td>
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<tr>
<td>( \dot{V}_E )</td>
<td>( W )</td>
<td>28.1 ± 1.596</td>
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Values are means ± SE given in l/min. \( \dot{V}_{O_2} \), \( O_2 \) uptake; \( \dot{V}_{CO_2} \), \( CO_2 \) production; \( \dot{V}_E \), expired minute ventilation; \( W \), water trial; \( G \), glucose trial; \( FG \), glucose plus fructose trial.
trials. CHO\textsubscript{tot} oxidation decreased during exercise in all three trials (time effect; \(F = 18.2; df = 5.45; P < 0.001\)) but was lower in the W trial (0.63 ± 0.05 g/min) than in the G trial (0.78 ± 0.051 g/min) and the FG trial (0.74 ± 0.056 g/min) (trial effect; \(F = 6.0; df = 2.18; P = 0.009\)). No difference in CHO\textsubscript{tot} oxidation was found between the G and FG trials. CHO\textsubscript{endo} oxidation decreased with exercise in all trials (time effect; \(F = 47.4; df = 5.45; P < 0.001\)) and tended to be higher in the W trial (0.63 ± 0.048 g/min) than in the G trial (0.55 ± 0.04 g/min) and FG (0.52 ± 0.05 g/min) (group effect; \(F = 1.98; df = 2.18; P = 0.17\)). Post hoc analysis indicated that CHO\textsubscript{endo} oxidation was significantly lower by 95 min in the G trial (\(P = 0.04\)) and in the FG trial (\(P = 0.03\)) than in the W trial. No differences in CHO\textsubscript{endo} oxidation was found between the G and FG trials.

Percent energy contributions from fat\textsubscript{tot}, CHO\textsubscript{tot}, CHO\textsubscript{endo}, and CHO\textsubscript{exo} to the total energy supply during exercise are shown in Fig. 3. The contribution from fat\textsubscript{tot} oxidation increased with time in all three trials (time effect; \(F = 33.0; df = 5.45; P < 0.001\)) but was higher in W (52.9 ± 3.06%) than in G (43.1 ± 2.29%) and FG (45.5 ± 3.29%) (trial effect; \(F = 6.1; df = 2.18; P = 0.009\)). CHO\textsubscript{exo} contribution increased with time (time effect; \(F = 302; df = 5.55; P < 0.001\)) and was similar between G and FG. CHO\textsubscript{endo} contribution decreased with time in all trials (time effect; \(F = 100.9; df = 5.45; P < 0.001\)) but was higher in W (47.1 ± 3.05%) than in G (40.0 ± 2.5%) and in FG (38.7 ± 3.56%) (trial effect; 3.3; df = 2.18; \(P = 0.05\)).

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**Table 3. Fat\textsubscript{tot}, CHO\textsubscript{tot}, CHO\textsubscript{endo}, and CHO\textsubscript{exo} oxidation rates during the water, glucose, and glucose plus fructose trials**

<table>
<thead>
<tr>
<th></th>
<th>Fat\textsubscript{tot}</th>
<th>CHO\textsubscript{tot}</th>
<th>CHO\textsubscript{endo}</th>
<th>CHO\textsubscript{exo}</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5–10 min</td>
<td>25–30 min</td>
<td>5–10 min</td>
<td>25–30 min</td>
</tr>
<tr>
<td>W</td>
<td>0.22 ± 0.022</td>
<td>0.27 ± 0.024</td>
<td>0.28 ± 0.027</td>
<td>0.30 ± 0.028</td>
</tr>
<tr>
<td>G</td>
<td>0.18 ± 0.021</td>
<td>0.25 ± 0.025</td>
<td>0.25 ± 0.023</td>
<td>0.24 ± 0.025†</td>
</tr>
<tr>
<td>FG</td>
<td>0.20 ± 0.030</td>
<td>0.26 ± 0.033</td>
<td>0.25 ± 0.028</td>
<td>0.26 ± 0.030†</td>
</tr>
<tr>
<td></td>
<td>W</td>
<td>0.82 ± 0.058</td>
<td>0.67 ± 0.050</td>
<td>0.61 ± 0.051</td>
</tr>
<tr>
<td>G</td>
<td>0.91 ± 0.068</td>
<td>0.76 ± 0.061</td>
<td>0.75 ± 0.054</td>
<td>0.76 ± 0.056†</td>
</tr>
<tr>
<td>FG</td>
<td>0.93 ± 0.096</td>
<td>0.74 ± 0.058</td>
<td>0.75 ± 0.055</td>
<td>0.72 ± 0.054†</td>
</tr>
</tbody>
</table>

Values are means ± SE given in g/min. Fat\textsubscript{tot}, total fat; CHO\textsubscript{tot}, total carbohydrate; CHO\textsubscript{exo}, exogenous carbohydrate; CHO\textsubscript{endo}, endogenous carbohydrate; df, degrees of freedom. Main effects for trial (\(F = 3.9; df = 2.18; P = 0.040\)) and time (\(F = 21.4; df = 5.45; P < 0.001\)) on fat\textsubscript{tot} oxidation were found. Main effects for trial (\(F = 6.0; df = 2.18; P = 0.009\)) and time (\(F = 18.2; df = 5.45; P < 0.001\)) on CHO\textsubscript{tot} oxidation were found. A main effect of time (\(F = 115.9; df = 5.55; P < 0.001\)) and a trial-by-time interaction (\(F = 2.4; df = 5.55; P = 0.045\)) on CHO\textsubscript{exo} oxidation were found. A main effect of time (\(F = 47.5; df = 5.45; P < 0.001\)) and a near-significant trial-by-time interaction (\(F = 1.8; df = 10.90; P = 0.08\)) on CHO\textsubscript{endo} oxidation were found. *Different from G, \(P < 0.05\). †Different from W, \(P < 0.05\).
In the G trial (40.6 ± 6.1% tended to be higher in the W trial (47.1 ± 6.6%) (trial effect; F = 3.56%) (trial effect; 3.0; df = 2,14; P = 0.001). Lactate levels were significantly lower during exercise in the W compared with G and FG trials (group effect; F = 5.53; df = 2,10; P = 0.02) but were not significantly different between the G and FG trials. Plasma glucose levels decreased after the onset of exercise in all trials, reaching a nadir at 10 min, and then increased toward baseline values in the W and the FG trials and above baseline values in FG (trial by time interaction; F = 2.15; df = 16,80; P = 0.01).

Performance test. Figure 6 shows individual exercise time to exhaustion during the performance test in each trial. Performance times averaged 142 ± 37, 177 ± 33, and 202 ± 40 s in the W, G, and FG trials, respectively (trial effect; F = 3.2; df = 2,22; P = 0.059). Post hoc analysis indicates that the performance time in the W trial was significantly less than in the FG trial (P = 0.049) but not significantly different from the G trial (P = 0.29). Seven of twelve subjects had higher performance times in the G than in W trial (Wilcoxon effect; F = 6.1; df = 2,18; P = 0.009). Percent energy contribution from fat_total oxidation did not differ significantly between the G and FG trials. Energy contribution from CHO_total oxidation decreased with time in all three trials (time effect; F = 33.0; df = 5,45; P < 0.001) but was lower in W (47.1 ± 3.06%) than in the G trial (56.9 ± 2.29%) and FG trial (54.4 ± 3.29%) (trial effect; F = 6.1; df = 2,18; P = 0.009). Percent energy contribution from CHO_total oxidation did not differ significantly between the G and FG trials. The percent contribution from CHO_oxidation in the CHO_intake trials increased with time (time effect; F = 302; df = 5,55; P < 0.001) and was similar in the G trial (16.9 ± 0.83%) and the FG trial (15.7 ± 0.88%) (trial effect; 3.0; df = 1,11; P = 0.11). The percent contribution from CHO_oxidation decreased with time in all trials (time effect; F = 100.9; df = 5,45; P < 0.001) and tended to be higher in the W trial (47.1 ± 3.05%) than in the G trial (40.0 ± 2.5%) and in the FG trial (38.7 ± 3.56%) (trial effect; F = 3.3; df = 2,18; P = 0.05). Percent energy contribution from CHO_oxidation did not differ significantly between the G and FG trials.

Figure 4 shows RPE (top) and stomach fullness (bottom) during the trials. RPE was similar among trials and increased from an average (collapsed across trials) of 10.3 ± 0.29 at 5 min to 14.6 ± 0.33 at 90 min (time effect; F = 49.0; df = 5,45; P < 0.001). Stomach fullness was also similar between trials and increased slightly with exercise from an average (collapsed across trials) of 1.7 ± 0.09 at 5 min to 2.3 ± 0.11 at 90 min (time effect; F = 3.8; df = 5,45; P = 0.006).

Figure 5 shows plasma insulin (top), lactate (middle), and glucose (bottom) concentrations during the trials. Insulin levels increased transiently after CHO_intake and decreased gradually during exercise in all three trials (time-by-trial interaction; F = 4.0; df = 8.56; P < 0.001). Insulin concentrations with the G trial were twofold higher than in the FG and W trials (trial effect; F = 11.3; df = 2,14; P < 0.001). Lactate levels peaked at 5 min of exercise and then decreased gradually (time effect; F = 23.11; df = 8,40; P < 0.001). Lactate levels were significantly lower during exercise in the W compared with G and FG trials (group effect; F = 5.53; df = 2,10; P = 0.02) but were not significantly different between the G and FG trials. Plasma glucose levels decreased after the onset of exercise in all trials, reaching a nadir at 10 min, and then increased toward baseline values in the W and the FG trials and above baseline values in FG (trial by time interaction; F = 2.15; df = 16,80; P = 0.01).
Spare endogenous fat and CHO by energy provision in boys ages 11–14 yr (Figs. 1 and 3, vs. 60% V\(\dot{O}_2\) peak) and for a similar duration (90 vs. 120 min) after a standardized breakfast. The exercise protocols differed somewhat, in that ours consisted of intermittent, rather than continuous, cycling. It seems unlikely, however, that the 5-min rest periods given in our study would alter the responses to one form of CHO\(_{exo}\) but not the other. It is possible, however, that the contradictory findings between studies may be explained by differences in subjects’ age or maturation level. In general, our subjects were pre- and early pubescent and untrained, whereas in the Adopo et al. study, subjects were trained adults. Further investigation is necessary, therefore, to determine whether various CHO\(_{exo}\) beverages have altered absorption or oxidation rates in children or adolescents compared with adults.

More important differences in study protocol that may explain the contrasting findings are the concentration and the timing of the CHO\(_{exo}\) beverages. In the Adopo et al. (1) study, subjects consumed a 20% CHO\(_{exo}\) solution at the onset of exercise, whereas, in our study, subjects drank 6% CHO\(_{exo}\) solutions intermittently during exercise. It is possible that the high G concentration in their study saturated intestinal glucose transport, thereby potentially limiting its oxidation rate. In contrast, the ingestion of FG in their study, however, was thought by Adopo et al. to stimulate absorption of the CHO\(_{exo}\) through additional transport mechanisms recruited by the addition of F, as has been shown previously (39). In other words, the lower oxidation rate of G relative to FG oxidation, in their study, may have been explained by a saturation of glucose transport mechanisms in the G trial that may not have occurred during the FG trial. Indeed, increasing CHO concentration from 8 to 25% markedly decreases glycemic responses in healthy adults, likely because of reduced CHO absorption (9). In our laboratory’s study, subjects ingested 6% CHO\(_{exo}\), 18 mmol/l NaCl solutions that have been shown to have high rates of intestinal absorption compared with more concentrated CHO beverages (40). It is unlikely, therefore, that intestinal absorption was limiting in either of the CHO\(_{exo}\) trials in our study. Thus FG oxidation may only exceed G oxidation if the CHO\(_{exo}\) is provided in a concentrated bolus at the onset of exercise. Further investigation is required, however, to test this hypothesis.

Another important difference between our study and the previous study by Adopo et al. (1) is the timing of CHO\(_{exo}\) ingestion. It may be that the bolus ingestion of CHO\(_{exo}\) at the onset of exercise in the study by Adopo et al. allowed for sufficient time for the absorbed F to be converted to G by the liver before its oxidation. This conversion of F to G appears to be obligatory before oxidation by muscle when F is ingested during exercise (17). In our study, the ingestion of FG during the exercise may not have allowed sufficient time for the F to be converted to G before its oxidation, which may explain why FG oxidation was slightly impaired during exercise in our study. This hypothesis appears unlikely, however, because rates of oxidation of the two CHO\(_{exo}\) beverages were similar for the initial 60 min of exercise (Fig. 1).

Plasma glucose levels decreased after the onset of exercise for a brief period regardless of the beverages that were consumed (Fig. 5). This pattern, which has also been observed previously by Delamarche et al. (11) in 8- to 11-yr-old boys and girls, is thought to indicate
some impairment in the glucoregulatory response to exercise in youth. Interestingly, the 11% decrease in glycemia during the initial 20 min of exercise, which was also observed previously by Delamarche et al., was not attenuated by CHOexo in our study (Fig. 5). After this hypoglycemic response, levels returned to baseline in W and in FG trials and were elevated by ~1 mmol/l above baseline during the G trial. These data appear to agree with our laboratory’s previous observation that blood glucose levels are higher during exercise with G vs. W ingestion in healthy adolescent boys (38) and boys with insulin-dependent diabetes mellitus (37).

In addition to a blunted glucose response to FG, we found that the plasma insulin levels were twofold higher during exercise with G than with FG or W (Fig. 5). A blunted insulin response to F compared with G intake has previously been observed in adults during exercise (18, 20). We extend these findings, for children and adolescents, by demonstrating that the insulin response to FG is considerably less than an isocaloric intake of G alone. Indeed, the failure of FG to elevate plasma insulin concentrations were considerably lower in the FG than in the G trial, even though the amount of CHOexo consumed was identical between the two trials. In addition, the observation that insulin levels increase dramatically after G intake in our study may support the previous hypothesis that, compared with adults, children and adolescents may have a decreased insulin sensitivity, which is usually compensated by a greater insulin secretion (43).

Plasma lactate concentrations increased at the onset of exercise during the initial 20 min, after which time they decreased gradually during exercise (Fig. 5). A decrease in lactate levels during prolonged moderate-intensity exercise has previously been shown by our laboratory (38) and others (15) in adolescents; however, it is currently unclear whether this indicates a reduction in lactate production or an increase in lactate clearance.

Compared with W ingestion, FG appears to increase the exercise time to volitional exhaustion at 90% peak power after 90 min of prolonged moderate exercise (Fig. 5). In our study, the exercise time to exhaustion was longer with FG intake (202 ± 40 s) than with W intake (142 ± 37 s) \( (P = 0.049) \), whereas the performance with G ingestion (177 ± 33 s) was not statistically significantly different from that with W intake \( (P = 0.29) \). These observations for adolescent boys are in line with others who show that, compared with W or G, F intake can postpone fatigue in exercising adults (for review, see Ref. 8) Evidence for improved high-intensity cycling performance with 6% sucrose (i.e., a disaccharide composed of G and F) compared with 6% F alone (29) or placebo (10) also supports the notion of an ergogenic effect of FG. The mechanism for the enhanced performance with FG is currently unclear but may be related to the muscle glycogen-sparing effect of F compared with either G or W intake during exercise (20). Had a muscle glycogen-sparing effect with FG ingestion occurred in our study, one might expect that CHOendo oxidation would be lower in that trial compared with the G trial. However, we found no difference in CHOendo oxidation (i.e., liver and muscle glycogen) between the G and FG trials (Fig. 3) that would support a difference in muscle or liver glycogen sparing between the two trials. It is possible that the relative proportions of muscle and liver glycogen to CHOendo may be different among the trials, which we are unable to assess with the methods used in the present study.

Although our experiment was not designed to compare substrate utilization in younger children with older adolescents, some interesting observations should be pointed out. Previously, our laboratory found that the percent energy contribution from fat was ~30% in older boys (ages 14–17 yr) during exercise performed at a moderate intensity (~60% \( V_{\text{O}_2\text{peak}} \)) with W intake (37, 38). In the present study, fat contribution was ~50% during exercise with W intake (Fig. 3) and at a similar relative intensity (~55% \( V_{\text{O}_2\text{peak}} \)) in younger boys (ages 10–14 yr). This finding supports previous studies that have shown that children use more fat and less carbohydrate compared with adults during exercise performed at the same relative intensity (21–23). In contrast to this difference in endogenous substrate utilization between younger and older adolescents, we found that the percent energy contribution from CHOexo was similar between younger \( (17 ± 0.8\%) \) and older \( (19 ± 0.8\%) \) boys during 90 min of similar relative intensity exercise (38). Thus it appears that the proportion of CHOexo utilized during exercise is relatively constant among children and adolescents but may be considerably higher than the 7–9% previously reported for adults in a similarly designed experiment (24). The observed differences in CHOexo oxidation between children and adolescents in our study compared with those conducted with adult subjects should be viewed with caution because of potential differences in the type, timing, and amount of CHO ingested; the duration and intensity of exercise performed; as well as the computation procedure used to calculate CHOexo oxidation (32, 33). Further investigation using standardized methodology is required, therefore, to probe the influences of maturation on endogenous and exogenous fuel utilization during exercise.

In summary, G and F plus G are oxidized at a similar rate in adolescent boys if beverages are consumed intermittently during exercise. In addition, both forms of CHOexo spare endogenous fat and CHOendo to a similar extent and contribute to ~16% of the total energy provision during moderate exercise in boys ages 10–14 yr.

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REFERENCES

1. Adopo E, Péronnet F, Massicotte D, Brisson GR, and Hillaire-Marcel C. Respective oxidation of exogenous glucose and


