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Fructose–Maltodextrin Ratio Governs Exogenous and Other CHO Oxidation and Performance

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ABSTRACT

O'BRIEN, W. J., S. R. STANNARD, J. A. CLARKE, and D. S. ROWLANDS. Fructose–Maltodextrin Ratio Governs Exogenous and Other CHO Oxidation and Performance. *Med. Sci. Sports Exerc.*, Vol. 45, No. 9, pp. 1814–1824, 2013. **Introduction**: Fructose coingested with glucose in carbohydrate (CHO) drinks increases exogenous-CHO oxidation, gut comfort, and physical performance. **Purpose**: This study aimed to determine the effect of different fructose–maltodextrin–glucose ratios on CHO oxidation and fluid absorption while controlling for osmolality and caloricity. **Methods**: In a crossover design, 12 male cyclists rode 2 h at 57% peak power then performed 10 sprints while ingesting artificially sweetened water or three equiosmotic 11.25% CHO-salt drinks at 200 mL·15 min^{-1}, comprising weighed fructose and maltodextrin–glucose in ratios of 0.5:1 (0.5 ratio), 0.8:1 (0.8 ratio), and 1.25:1 (1.25 ratio). Fluid absorption was traced with D_2O, whereas ^{14}C-fructose and ^{13}C-maltodextrin–glucose permitted fructose and glucose oxidation rate evaluation. **Results**: The mean exogenous-fructose and exogenous-glucose oxidation rates were 0.27, 0.39, and 0.46 g·min^{-1} and 0.65, 0.71, and 0.58 g·min^{-1} in 0.5, 0.8, and 1.25 ratio drinks, representing mean oxidation efficiencies of 54%, 59%, and 55% and 65%, 85%, and 86% for fructose and glucose, respectively. With the 0.8 ratio drink, total exogenous-CHO oxidation rate was 18% (90% confidence interval, ±5%) and 5.2% (±4.6%) higher relative to 0.5 and 1.25 ratios, respectively, whereas respective differences in total exogenous-CHO oxidation efficiency were 17% (±5%) and 5.3% (±4.8%), associated with 8.6% and 7.8% (±4.2%) higher fructose oxidation efficiency. The effects of CHO ratio on water absorption were inconclusive. Mean sprint power with the 0.8 ratio drink was moderately higher than that with the 0.5 ratio (2.9%; 99% confidence interval, ±2.8%) and 1.25 ratio (3.1%; ±2.7%) drinks, with total- and endogenous-CHO oxidation rate, abdominal cramps, and drink sweetness qualifying as explanatory mechanisms. **Conclusions**: Enhanced high-intensity endurance performance with a 0.8 ratio fructose–maltodextrin–glucose drink is characterized by higher exogenous-CHO oxidation efficiency and reduced endogenous-CHO oxidation. The gut-hepatic or other physiological site responsible requires further research. **Key Words:** GASTROINTESTINAL DISTRESS, GLYCOGEN, STABLE ISOTOPES, POWER, SWEETNESS

The ingestion of solutions containing multiple-transportable monosaccharides during prolonged exercise increases gastric emptying and intestinal fluid absorption (19,33), exogenous carbohydrate (CHO) oxidation (1,16,17,39), and endurance performance (8,37), relative to single CHO solutions. Noncompetitive intestinal transport of glucose through the sodium-glucose cotransporter-1 (SGLT1) and the fructose transporter (GLUT5) (40), additional expression and recruitment and trafficking of the universal monosaccharide transporter GLUT2 (20,22) and possibly other transporters (e.g., GLUT8 [10]) to the brush-border membrane or undefined hepatic metabolism may be responsible for these beneficial effects. The ratio of the coingested monosaccharides also influences intestinal CHO absorption (33) and exogenous CHO oxidation rates (24). Using a triple-lumen perfusion model, Shi et al. (33) reported faster CHO and fluid absorption with fructose and glucose or sucrose with effective (brush-border monosaccharide transport) fructose–glucose ratios of 0.7–1.0:1 compared with solutions with effective ratios of 0.5:1. In support of these findings, we have observed that a drink containing a 0.8 ratio of fructose–maltodextrin yielded a higher exogenous-CHO oxidation rate than either 0.5 or ~1.2 ratio drinks during prolonged endurance exercise (24,30). In these studies, individual oxidation rates and efficiency (ingestion rate/oxidation rate × 100) of exogenous fructose and maltodextrin were measured using radio (^{14}C) and stable (^{13}C) isotopes, respectively. Maltodextrin ingestion was clamped at 0.6 g·min^{-1}, whereas fructose ingestion rate was manipulated to produce effective fructose–glucose ratios of 0.5, 0.8, and 1.2. Total exogenous-CHO oxidation rate was highest with the 0.8 ratio drink because of an apparent increase in the oxidation efficiency for glucose, providing evidence for transport or metabolic synergy associated with coingested CHOs first proposed by Shi et al. (33). However, because net exogenous-CHO oxidation efficiency decreased with increasing fructose dose, the

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associated increasing drink concentration and osmolality may have influenced CHO absorption in the small intestine independent of any transepithelial transport synergism (13,33).

Our first objective in the current study was to determine whether increased fructose or increased glucose oxidation efficiency was responsible for higher total exogenous-CHO oxidation rate with ingestion of a 0.8 ratio fructose–maltodextrin–glucose drink during exercise. To control for concentration and osmolality, drinks were isocaloric and equiosmotic and ingested at a rate close to that likely most favorable for endurance performance (1.5 $g \cdot min^{-1}$) (34).

We recently reported a large increase in peak power in a slow-ramp incremental exercise test with ingestion of a 0.8 ratio fructose–maltodextrin drink relative to a 0.5 ratio (24). With respect to possible mechanisms to explain the ratio effect on performance (33), a mechanisms (covariate) analysis indicated that peak power was influenced by nausea, an integrated central perception of gut comfort. Any elevation in nausea perception during exercise may distract from effort, outweighing the benefit gained from increased exogenous-CHO oxidation. Minimal gastrointestinal distress may also indicate better gastric emptying and fluid absorption, which along with reduced gastrointestinal discomfort were reported with the ingestion of 0.5 ratio fructose–glucose drinks compared with isocaloric glucose-only drinks (16). Faster fluid absorption with multiple-transportable CHO solutions (9,19) supports the proposal of enhanced unilateral water absorption via osmosis following the absorbed solutes (33). Given the synergism between CHO and fluid absorption (33) and the role of circulatory-fluid homeostasis on high-intensity endurance performance (12), the second objective was to determine the effect of fructose–maltodextrin–glucose ingestion ratio on ingested fluid absorption.

The final objective was to provide further evidence to support the advantage of the 0.8 fructose–maltodextrin–glucose ratio over other ratios on endurance performance. A validated repeated-sprint endurance performance protocol (29–31,36) and a preload of higher intensity than used previously (10) was utilized in an ecologically valid model for high-intensity endurance performance.

We hypothesized that compared with the 0.5 and 1.25 ratio drinks, the 0.8 ratio would result in (a) the highest exogenous-CHO oxidation rate through a synergism generated via attainment of peak physiological exogenous-glucose absorption (unmeasured) and oxidation leading to higher relative exogenous-fructose oxidation (EFO) efficiency, (b) the more rapid fluid absorption associated with osmotic draw as measured by higher blood deuterium oxide concentrations, and (c) the combined effect of these outcomes providing better gut comfort and substantially enhanced performance.

METHODS

Participants

Twelve trained male cyclists, mountain bikers, and triathletes age 36.2 ± 8.0 yr and with a body mass of 79.4 ± 9.5 kg participated in the study (mean ± SD). Maximal oxygen uptake ($\dot{V}O_{2max}$) and peak power (W_{max}) were 59.1 ± 5.2 $mL \cdot kg^{-1} \cdot min^{-1}$ and 367.0 ± 31.6 W, respectively. All participants had been cycling >8 $h \cdot wk^{-1}$ for at least the previous 12 months. Participants read the information sheet, were screened for contraindications, were fully informed of the purpose and risks of the procedure, and were provided written consent before commencing the study. The study protocol was approved by the Human and Disability Ethics Committee, Ministry of Health, New Zealand.

Experimental Design

A randomized double-blind four-way crossover was used to determine the effect of drink composition on outcomes. Each participant made nine visits to the laboratory for 6 wk. The first visit on week 1 consisted of an incremental test to establish $\dot{V}O_{2max}$ and W_{max} followed by familiarization of the performance test. Subjects modified and recorded their cycle training and repeated this on a weekly basis according to the defined experimental weekly block: day 1, long duration ride (3–4 h); days 2 and 3, medium-duration ride (2–3 h); day 4, controlled standardized ride in the laboratory (2 h at 50% W_{max}; day 5, rest day; day 6, experimental trial; and day 7, recovery ride (1–2 h). Each of the four experimental trials was separated by 7 d, and for each subject, trials were conducted at the same time of day to control for circadian variance. To reduce background ^{13}C enrichment, subjects were provided with extensive food lists and instructed not to eat foods with components derived from plants naturally enriched with ^{13}C (i.e., maize, sugar cane) for at least 10 d before the first experimental trial and for the duration of the study. To standardize diet, all subjects recorded their food intake for the 2 d before the first experimental trial and repeated this intake on the second day before each of the three subsequent trials. To assist in standardizing energy intake and hydration, subjects were provided with a prepackaged pasta meal (per kilogram body mass, 55 kJ; 1.85 g CHO, 0.63 g protein, 0.62 g fat) and a 600-mL drink bottle full of water, all to be consumed the evening before each experimental trial.

Protocols

Preliminary testing and familiarization. $\dot{V}O_{2max}$ and W_{max} were measured using a progressive exercise protocol on an electronically braked cycle ergometer (VeloTron Racer Mate, Seattle, WA) and a calibrated Moxus MaxII Metabolic System (AEI Technologies, Naperville, IL) as described elsewhere (24). After the incremental test, participants rested for 10 min then completed a full familiarization of the experimental trial including the repeated-sprint performance test.

Experimental trial. Subjects reported overnight fasted to the laboratory between 0500 and 0630 h on day 6 of each weekly block. No strenuous activity was undertaken or

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alcohol consumed in the previous 24 h. On arrival, a 20-GA cannula (Becton Dickinson Medical Pte Ltd., Singapore) was inserted into an antecubital vein. A two-way stopcock valve (Becton Dickinson Medical Pte Ltd.) was connected to the cannula to allow for blood sampling at this point and during exercise and was maintained patent with regular saline flushes. After a resting blood sample, subjects toileted and had their body mass recorded then were seated next to the Velotron cycle ergometer to complete resting psychometric scales and expired breath sampling. Breath sampling procedures comprised ~1 min of breathing through the mouthpiece and a two-way valve (Hans Rudolph, Shawnee, KS) to stabilize respiration followed by the collection of expired breath for 90 s directed through a 5-L mixing chamber connected in series to a 150-L Douglas bag and a 6-L anesthetic bag. Near the end of the 90-s collection, expired breath samples were drawn into 2 × 10 mL evacuated tubes (Exetainer, Labco Ltd., High Wycombe, UK) from a 20-GA needle positioned at the distal end of the mixing chamber; these were later used to quantify breath ^{13}C enrichment and exogenous-glucose oxidation rate. Further expired breath was saved in the anesthetic bag and used for the analysis of $^{14}CO_2$ activity for quantification of EFO (16). Breath held in the anesthetic bag was bubbled through a CO_2 trapping solution until the pink-colored solution became clear, at which point exactly 1 mmol of CO_2 was trapped. The trapping solution was contained in a 20-mL scintillation vial comprising 1 mL of hyamine hydroxide in 1 M of methanol (Fisher Scientific, Fair Lawn, NJ), 2 mL of 96% ethanol (VWR International Ltd., Poole, England), and one to two drops of phenolphthalein (Ajax Finechem, Auckland, NZ). Once 1 mmol of CO_2 was trapped, 17 mL scintillation cocktail (Ultima Gold XR; Perkin Elmer, Waltham, MA) was added to the trapping solution. $^{14}CO_2$ radioactivity (disintegrations per minute [dpm]; later converted to $dpm \cdot mmol^{-1}$) was determined by 10-min triplicate counts in a liquid scintillation counter (Wallac 1409 LS, Turku, Finland).

After resting sampling, subjects rode for 2 h at 57.5% W_{max} as the highest steady-state intensity participants were likely to complete before the performance test. Outcome variables were collected at rest and every 15 min during exercise in the order of ratings of exertion, expired breath samples, drink ingestion, and rating of drink sweetness. Blood samples were drawn at rest and at 28, 40, 50, 60, 70, 80, and 90 min during exercise. At the completion of the 2-h cycle, subjects dismounted, had a final blood sample taken, and had their cannula removed. Subjects then toileted and had their body mass recorded before remounting the cycle ergometer to complete the performance test. This test consisted of 10 maximal sprint efforts taking approximately 2 min each to complete, interspersed and beginning with a recovery interval at 40% W_{max} taking 5.5 min; full procedures and calculations for power output are described elsewhere (29,31,36). No breath or blood samples were collected during the performance test. During all rides, environmental conditions were maintained at 20°C (1.0) and 43.8% (6.1) relative humidity by air conditioning with a standardized airflow maintained over the subjects by a fan.

CHO Drinks

Immediately before exercise, subjects ingested a 400-mL bolus of experimental drink, followed by a further 200 mL at 15-min intervals throughout the 2-h ride. During the performance test, the drinks continued to be ingested on a per serving basis (200 mL) provided at the beginning of the trial and again at the beginning of each second subsequent recovery blocks (every ~16 min). Four different drinks were prepared for ingestion during exercise. The three experimental drinks comprised fructose and maltodextrin and/or glucose, at ratios of 0.5:1 (0.5 ratio), 0.8:1 (0.8 ratio), and 1.25:1 (1.25 ratio) (fructose–maltodextrin–glucose). The quantity of maltodextrin in all three experimental drinks was fixed at 0.67 $g \cdot min^{-1}$, with glucose added to the 0.5 ratio (0.33 $g \cdot min^{-1}$) and 0.8 ratio (0.67 $g \cdot min^{-1}$) drinks to balance drink osmolality (419–429 $mOsm \cdot kg^{-1}$) against fructose content while maintaining isocaloricity and CHO concentration (11.25%). A summary of the CHO drinks is provided in Supplemental Digital Content 1, http://links.lww.com/MSS/A267 (table defining the drink composition). The control drink was water (46 $mOsm \cdot kg^{-1}$) containing 2.1 $g \cdot L^{-1}$ artificial sweetener (Sucaryl, Hansells, Masterton, NZ). Included in each drink was NaCl (1.17 $g \cdot L^{-1}$, 20 $mmol \cdot L^{-1}$ Na^+), citric acid (2.11 $g \cdot L^{-1}$), and lime juice (16 $g \cdot L^{-1}$). Both the maltodextrin (Star-Dri 100; Tate & Lyle, Decatur, IL) and glucose (National Starch, Auckland, NZ) were maize derived with ^{13}C enrichment of −10.40 δ‰ and −10.78 δ‰ (respectively) versus Vienna Pee Dee Bellemnitella (v-PDB). The fructose (Fructofin C, Danisco, Manukau, NZ) was sourced from beetroot (−26.87 δ‰). Drinks consumed between 0 and 105 min (1.8 L) were labeled with a total of 6.75 kBq (calculated effective dose, 0.3915 mSv) of U-$^{14}C_6$-fructose (American Radiolabeled Chemicals, St. Louis, MO). The U-$^{14}C_6$-fructose was omitted from drinks ingested during the performance test to minimize unnecessary exposure. Drinks consumed during the 2-h training rides on day 4 contained maltodextrin derived from tapioca (Briess Malt & Ingredients, Chilton, WI), NaCl, citric acid, and lime juice at the same total concentrations as in the experimental drinks. At exactly 30 min into the 2-h cycle, 5 g of 99.8% deuterium oxide (D_2O; Cambridge Isotope Laboratories, Andover, MA) was ingested to allow measurement of water absorption (19).

Psychometric Scales

The effect of drink CHO ratio on physical exertion (exertion, muscle soreness, and leg tiredness during sprints only), gastrointestinal comfort (nausea and abdominal cramping), and drink sweetness were recorded at rest, every 15 min during the 2-h ride, and after sprints 1, 4, 7, and 10 of the performance test. On each scale, the following levels of

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perception were represented: 0 (nothing), 1 (extremely mild), 2 (mild), 4 (moderate), 6 (high), and 8 (maximal). Subjects were instructed to make a pen mark on a continuous scale rating the strength of their perception of the measure.

Blood Treatment

Blood was deproteinated using the perchloric acid extraction according to Moore et al. (23). D_2O enrichment of the extract was determined by continuous-flow isotope-ratio mass spectrometry (Finnigan DeltaV; Thermo Electron Corporation, Bremen, Germany). The isotopic enrichment is expressed as δ‰ against the international water standard Vienna Standard Mean Ocean Water.

Expired Breath

Analysis. Fractions of oxygen and carbon dioxide in expired gas from Douglas bag collections were measured through calibrated gas analyzers of the Moxus. Expired gas volume was measured using PowerLab 4/20 spirometer and software (ADInstruments, Bella Vista, NSW). Volume calibration was carried out before sampling using a known volume (90 L) and verified again at the end of each testing session. Any drift was assumed to be linear, and raw volumes were adjusted accordingly. Expired breath samples captured in the evacuated tubes were analyzed for $^{13}C/^{12}C$ by gas chromatography continuous flow isotope-ratio mass spectrometry (Finnigan Delta XP; Thermo Electron Corporation).

Calculations. Total fat and CHO oxidation rates ($g \cdot min^{-1}$) were calculated using the nonprotein respiratory quotient (18): CHO oxidation ($g \cdot min^{-1}$) = $4.210\dot{V}CO_2 - 2.962\dot{V}O_2$; fat oxidation ($g \cdot min^{-1}$) = $1.695\dot{V}O_2 - 1.701\dot{V}CO_2$. Energy potentials of 15.64 $kJ \cdot g^{-1}$ for CHO and 40.81 $kJ \cdot g^{-1}$ for fat oxidation were used to estimate the contribution to energy expenditure (18). Endogenous CHO oxidation was assumed to be 100% from muscle glycogen; therefore, an energy potential of 17.36 $kJ \cdot g^{-1}$ for endogenous CHO (glycogen) oxidation was used to estimate the contribution to energy expenditure.

Isotopic enrichment of expired CO_2 was expressed according to the following formula: $\delta^{13}C = [(^{13}C/^{12}C \text{ ratio sample} / ^{13}C/^{12}C \text{ ratio standard}) - 1] \times 10^3‰$, where $^{13}C/^{12}C$ standard = 0.0112372 (7). The exogenous-CHO oxidation rate ($g \cdot min^{-1}$) was calculated from = $\dot{V}CO_2[(\delta_{Exp} - \delta_{bkg}) / (\delta_{Ing} - \delta_{bkg})] / k$, in which δ_{bkg} is the ^{13}C enrichment of expired air in the control condition, δ_{Exp} is the ^{13}C enrichment of expired CO_2 during the 2-h ride with ^{13}C-enriched CHO ingestion, δ_{Ing} is the ^{13}C enrichment of the CHO, and k is the volume of CO_2 (L) produced via the oxidation of 1 g of glucose (k = 0.7467). The oxidation rate of ingested maltodextrin was given as grams of glucose equivalents oxidized, assuming that 1.00 g of maltodextrin provides 1.11 g of glucose, owing to the property of dehydration of the maltodextrin (28). Therefore, with a dextrose equivalent of 10.4, this conversion was applied to only 89.6% of the maltodextrin in drinks, whereas the remaining 10.4% was calculated gram for gram as glucose.

The rate of EFO was calculated according to the formula: EFO = $\dot{V}CO_2[(^{14}CO_2 \cdot 6) / (\text{SA Fruc})](1 / k)$, where $^{14}CO_2$ is the radioactivity of 1 mmol of expired CO_2 ($dpm \cdot mmol^{-1}$) multiplied by 6 because there are six carbon atoms per molecule of [U-^{14}C]fructose, SA Fruc is the specific activity of the ingested fructose ($dpm \cdot mmol^{-1}$), and k is the volume of CO_2 (L) produced by the oxidation of 1 g of fructose (k = 0.7467) (30). The percentage efficiency of exogenous-glucose and exogenous-fructose metabolism was oxidation/ingestion rate × 100. The exogenous-CHO outcome measures were reported from 60 to 120 min of exercise due to the delay in equilibration of $^{13}CO_2$ and $^{14}CO_2$ with the large endogenous HCO_3^- pool (24,26).

Statistical Analysis

Sample size. The typical error measurement (coefficient of variation [CV]) for sprint mean power was 3.1% (31). Using the clinical likelihood sample size method of Hopkins et al. (14) and an anticipated moderate within-subject effect size 0.9 CV (2.79%), a sample size of 10 was calculated. A Latin square (Williams design) was used leaving four nonsequential orders of application. Therefore, to balance n = 12.

General method. The effects of fructose–maltodextrin–glucose ingestion ratio on outcomes were estimated with mixed models (Proc Mixed, SAS Version 9.1, SAS Institute, Cary, NC). Dependent variables, except psychometric parameters and raw data expressed as a percent, were analyzed after natural log-transformation to reduce effects of nonuniformity of error and to express changes as percentages (14). Fixed effects were treatment and the order term, which accounts for familiarization, adaptation, or fatigue effects between consecutive trials. For time-series data, the x-axis variable was grand-mean centered for linear modeling (as in regression analysis). Subject was the random effect. To graphically summarize exogenous-CHO oxidation rate and efficiency data to aid inference, linear quadratics were fitted in a mixed model to the appropriately transformed mean between-subject value for the independent variable, with CHO ratio the x-axis parameter, and subject the random effect.

Mechanisms analysis of the relationship between CHO metabolism and gastrointestinal distress on power. The modifying effect of metabolic and gastrointestinal distress outcomes on the effect of treatment on performance was determined via singular addition of the standardized mechanism covariate to a linear model for sprint mean power. The covariates were the average value of the data (metabolic data log-transformed) derived from the time series standardized to the within-subject standard deviation for the parameter. In this analysis, the corresponding qualified magnitude of change in treatment effect on power by the covariate indicates the extent to which change in

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power was attributable to change in the covariate. Covariate-adjusted effects that likely substantially altered the magnitude of the mean unadjusted effect thereby altering the magnitude-based inference were prioritized in reporting.

Precision of estimation and statistical inference. Inference was by magnitude-based inference as described recently (14,24). Precision was 90% confidence intervals for mechanistic variables and 99% confidence intervals on the harm side of uncertainty for performance. Interpretation of uncertainty was in relation to effect-size magnitude thresholds (14,24). The magnitudes of effects on performance were decided via a novel rationale (4): the performance test was assumed to simulate the physical demands of a typical intermittent high-intensity cycle race, where the threshold for the smallest worthwhile change was the CV for sprint power × 0.3 (0.93%) (14). Further effect magnitudes were accordingly qualified using the thresholds: moderate, ×0.9 (2.8%); large, ×1.6 (5.0%); very large ×2.4 (7.5%), and extremely large, ×4 (12.4%) (14). For other outcomes, we chose the standardized (Cohen) change of 0.20 times the between-subject standard deviation for the baseline measure in the control condition as the substantial threshold; a modified classification system (trivial, 0.0–0.2; small, 0.2–0.6; moderate, 0.6–1.2; large, 1.2–2.0; very large, >2.0) was used to interpret the magnitude of the standardized change (14). An effect was unclear if the confidence interval overlapped both the upper and the lower thresholds for substantiveness. Otherwise, the likelihood of a substantial increase or decrease was calculated from the two-tailed Student's *t* distribution and classified as follows: <0.5%, almost certainly not; 0.5%–5%, very unlikely; 5%–25%, unlikely; 25%–75%, possible; 75%–95%, likely; 95%–99.5%, very likely; and >99.5%, almost certain. When the majority (>50%) of the confidence interval lies between the threshold for substantially positive and negative effects, the likelihood of the effect being trivial (negligible) was qualified.

RESULTS

Performance

The overall sprint mean power was 294, 303, 296, and 275 W (between-subject CV 18%) for 0.5, 0.8, and 1.25 ratio and water, respectively (Figs. 1A and 1B). Relative to both the 0.5 and the 1.25 ratio drinks, higher sprint mean power was very likely with the 0.8 ratio (likelihoods of harm/trivial/benefit relative to the threshold for a small worthwhile effect size of 0.93% (24): 0.0/3.3/96.7 and 0.0/1.7/98.3, respectively); accordingly, the 0.4% (99% CI, ±2.8%) difference between 0.8 and 1.25 ratios was unclear (likelihoods: 29.2/59.4/11.4). Relative to water, a large to extremely large overall increase in sprint mean power in all CHO conditions was almost certain (likelihoods: 0.0/0.0/100). Reduction in mean power output (slope effect) for the 10 repeated sprints was 13.2% (±3.4%), 11.4% (±3.5%), 13.0% (±3.5%), and 14.5% (±3.4%) for 0.5, 0.8, and 1.25 ratio and water, respectively; only the 3.6% (±5.8%) attenuation in fatigue between the 0.8 ratio drink and water was likely substantial.

Substrate Oxidation

Breath $^{14}CO_2$ radioactivity and ^{13}C enrichment during the 2-h ride are shown in as figures (see Supplemental Digital Content 2, http://links.lww.com/MSS/A268), figure plates A and B, breath and blood isotope enrichments during exercise). Oxidation rates are shown in Figures 2A–2E, and exogenous-CHO oxidation efficiency is presented in Figures 3A–3C. A statistical summary for average substrate oxidation rates and exogenous-CHO oxidation efficiency is in Table 1, with the corresponding treatment effect estimates in Table 2.

Exogenous CHO oxidation. The oxidation rate of exogenous fructose increased with dose, but EFO efficiency was highest with the 0.8 ratio (Tables 1 and 2). Despite the

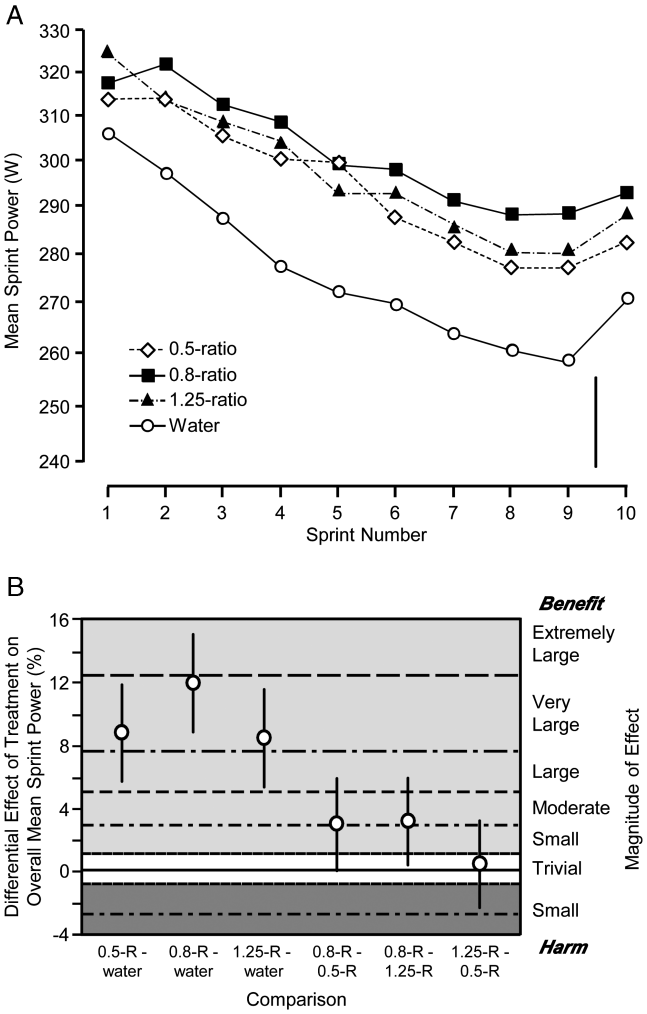

FIGURE 1—Effect of CHO ratio on sprint mean power during the performance test. (A) Plot of mean sprint power by sprint number. Bar represents the composite between-subject standard deviation derived from the analysis. (B) Point data are the overall average sprint mean power with the bars showing the statistical uncertainty represented as the 99% confidence interval. Full description of the probabilities associated with the magnitude-based inferences is provided in the Methods section. R, ratio.

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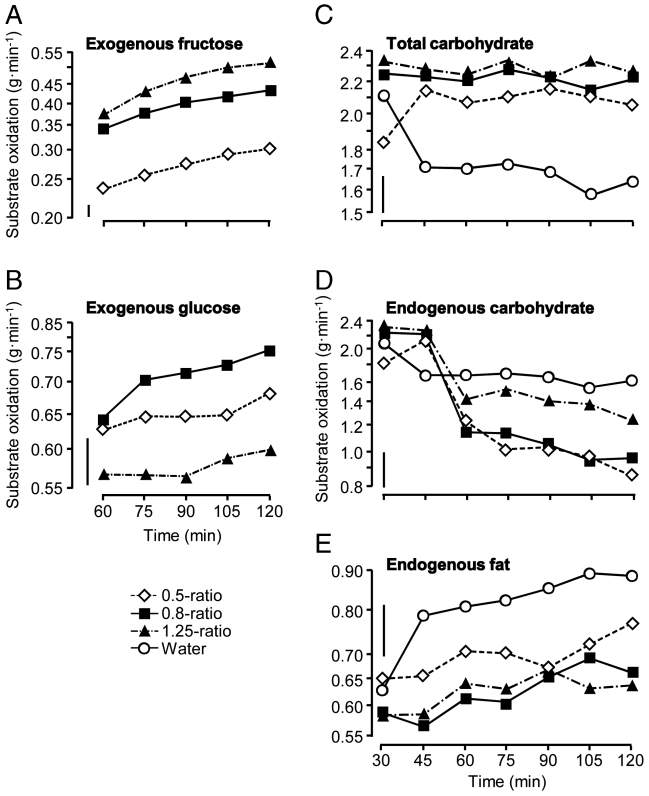

FIGURE 2—The effect of CHO ratio on pattern of (A) exogenous fructose, (B) exogenous glucose, (C) total CHO, (D) endogenous CHO, and (E) endogenous fat oxidation rates during the 2-h ride. Bars represent the respective back-transformed composite between-subject CV.

lower ingestion rate, a small increase in exogenous-glucose oxidation rate was possible with the 0.8 ratio compared with the 0.5 ratio. Exogenous-glucose oxidation efficiency was almost certainly lowest with the 0.5 ratio compared with the other CHO conditions. The total exogenous-CHO oxidation rate and oxidation efficiency was higher with the 0.8 ratio relative to the 0.5 and 1.25 ratio conditions (Tables 1 and 2).

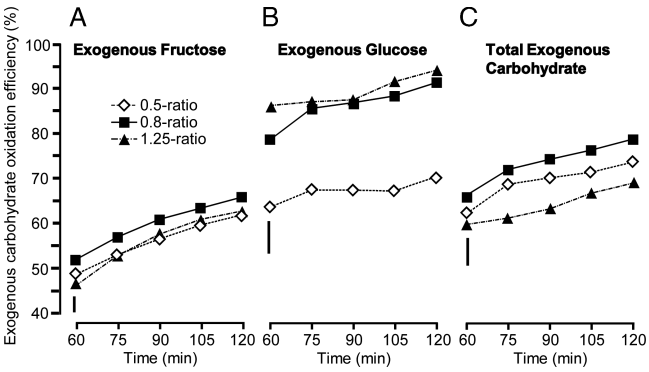

FIGURE 3—The effect of CHO ratio on the efficiency of the oxidation of (A) exogenous fructose, (B) exogenous glucose, and (C) total exogenous CHO ingested during the 2-h ride. Bars represent the back-transformed composite between-subject CV.

Fat and endogenous and total CHO oxidation. CHO ratio had no clear effect on endogenous-fat oxidation rate (Fig. 2; Table 2); and as expected, fat oxidation was substantially higher with water relative to all CHO conditions. Relative to water, only the 0.5 and the 0.8 ratio conditions lowered endogenous-CHO oxidation rate (Table 2), whereas there were very likely moderate and large increases with the 1.25 ratio condition relative to the 0.5 and 0.8 ratio conditions, respectively (Fig. 2; Table 2). Although no clear differences in the rate of total-CHO oxidation were observed between any of the CHO conditions, oxidation rates were likely higher than water (Table 2). Differences in slope between the CHO conditions were unclear (comparison not shown).

D_2O Appearance

Blood deuterium enrichments rose during the 28- to 90-min period of the 2-h ride after ingestion of D_2O at 30 min (Supplemental Digital Content 2, http://links.lww.com/MSS/A268), plate C, breath and blood isotope enrichments during exercise). The initial rise in blood deuterium enrichment was faster with water compared with the CHO drinks; but overall, contrasts were unclear (standardized differences in blood enrichment: 0.8 minus 0.5 ratio, −0.34 [90% CI, ±0.78]; 1.25 minus 0.8 ratio, 0.27 [±0.93]; 1.25 minus 0.5 ratio, −0.12 [±0.84]). All slope contrasts were also unclear.

Gastrointestinal Comfort and Exertion

Gastrointestinal comfort. The perception of nausea during exercise was below mild (<2 scale units) for all conditions, with trivial effects of treatment. The rate at which nausea perception increased (slope effect) during the 2-h ride was very likely moderately faster with water compared with all CHO conditions, and possibly faster with 0.5 ratio relative to the

TABLE 1. Oxidation rates of exogenous and endogenous substrates during the 60th to the 120th min of the 2-h ride.

	Drink			
Substrate	**Water**	**0.5 ratio**	**0.8 ratio**	**1.25 ratio**
Oxidation rate				
Exogenous fructose ($g \cdot min^{-1}$)	—	0.27 (46)	0.39 (56)	0.46 (53)
Exogenous glucose ($g \cdot min^{-1}$)	—	0.65 (30)	0.71 (14)	0.58 (28)
Total exogenous CHO ($g \cdot min^{-1}$)	—	0.94 (21)	1.10 (9)	1.03 (23)
Endogenous CHO ($g \cdot min^{-1}$)	1.64 (30)	1.01 (28)	1.04 (39)	1.39 (27)
Total CHO ($g \cdot min^{-1}$)	1.64 (30)	2.06 (21)	2.19 (27)	2.27 (19)
Endogenous fat ($g \cdot min^{-1}$)	0.85 (35)	0.71 (41)	0.64 (60)	0.64 (36)
Oxidation Efficiency				
Exogenous fructose (%)	—	54 (24)	59 (12)	55 (19)
Exogenous glucose (%)	—	65 (26)	85 (14)	86 (25)
Total exogenous CHO (%)	—	62 (12)	74 (7)	69 (13)

Oxidation rate ($g \cdot min^{-1}$) data are the back log-transformed least-squares mean. Values in parentheses are the between-subject CV (%).

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TABLE 2. Statistical estimates for the effect of drink composition on substrate oxidation rate and efficiency from the 60th to the 120th min period of the 2-h ride.

Substrate	Mean Effect Comparisons (%),[a] with ±90% CI[b] and Qualitative Inference[c]					
	0.5—water	0.8—water	1.25—water	0.8–0.5	0.8–1.25	1.25–0.5
Oxidation rate						
Exogenous fructose	—	—	—	45; ±27 moderate****	−16; ±21 unclear	67; ±31 large****
Exogenous glucose	—	—	—	8.8; ±7.2 small*	17.0; ±5.7 small****	−9.7; ±6.2 small**
Total exogenous CHO	—	—	—	18; ±5 moderate****	5.2; ±4.6 small*	12; ±5 small***
Endogenous CHO	−36; ±13 moderate****	−33; ±14 moderate****	−12; ±18 unclear	5; ±21 unclear	−31; ±27 moderate***	37; ±28 large***
Total CHO	25; ±23 moderate**	33; ±25 moderate***	38; ±26 large***	6; ±20 unclear	−4; ±19 unclear	11; ±27 unclear
Endogenous fat	−14; ±16 likely*	−24; ±14 moderate***	−24; ±14 moderate***	−11; ±16 unclear	0.4; ±18.1 unclear	−12; ±16 unclear
Oxidation efficiency						
Exogenous fructose	—	—	—	8.6; ±4.2 small**	7.8; ±4.2 small**	0.8; ±4.2 trivial**
Exogenous glucose	—	—	—	27; ±7 moderate****	−3.7; ±6.9 trivial*	30; ±7 moderate****
Total exogenous CHO	—	—	—	17; ±5 moderate****	5.3; ±4.8 small*	12; ±5 small****

[a]Data are the percent effect of treatment relative to the reference condition on substrate oxidation rate ($g \cdot min^{-1}$); 0.5, 0.8, and 1.25 refer to the 0.5, 0.8, and 1.25 ratio drinks.
[b]±90% CI: add and subtract this number to the mean to obtain the 90% confidence interval for the true difference.
[c]Qualified thresholds are described in the Methods section. Magnitude descriptors are *possible, **likely, ***very likely, ****most likely.

0.8 and 1.25 ratio conditions during the sprints (see Supplemental Digital Content 3, http://links.lww.com/MSS/A269), figure plates A and B, for the perceptual response during exercise in response to drink condition). Abdominal cramping was extremely mild (<1 scale unit) during exercise. During the 2-h ride, small increases in abdominal cramps were likely with water compared with 0.8 and 1.25 ratio conditions, but all other comparisons were trivial. During the sprints, there were likely small increases in overall ratings of abdominal cramps and in the rate of rise with the 0.8 ratio relative to all other conditions; however, absolute differences remained <1 scale unit.

Perceived exertion, muscle tiredness, and soreness. The perception of muscle soreness and exertion rose (slope effect) throughout the 2-h ride from extremely mild at time 0 to moderately severe at time 120 min. It continued to rise (slope effect) during the 10 repeated sprints, finishing between severe and very severe after the 10th sprint, but differences between treatments were trivial (not shown). Relative to 0.5 ratio, there was an overall very likely small reduction in muscle tiredness during the sprints compared with the 0.8 and 1.25 ratios and almost certainly moderate higher response relative to water. Tiredness increased (very large) in all conditions between the 1st and the 10th sprint (slope effect), but only the moderate increase with the 0.8 ratio drink relative to the 0.5 ratio drink was likely.

Drink sweetness. The perception of sweetness during exercise was moderately large lower with water compared with all CHO drinks (Supplemental Digital Content 3, http://links.lww.com/MSS/A269), figure plate 2C, perceptual response to drink composition during exercise). During the 2-h ride, sweetness was most certainly lower with the 0.5 ratio relative to 0.8 and 1.25 ratio. During the sprints, only the small increase in sweetness with the 1.25 versus 0.8 ratio and the moderate decline with water versus the increase with 0.5 ratio were likely. Other comparisons were unclear or trivial.

Mechanisms analysis. Total- and endogenous-CHO oxidation rates, total exogenous-CHO oxidation efficiency, abdominal cramp and nausea, and drink sweetness were likely substantial modifiers of sprint mean power; meanwhile, the exogenous-glucose and EFO rate and oxidation efficiency were possible substantial modifiers (a full summary of the mechanisms analysis is in Supplemental Digital Content 4, http://links.lww.com/MSS/A270), Tables 1–3.

Increased abdominal cramp was associated with the largest effect on sprint mean power of any of the covariates with a large (~7%) attenuating effect across the CHO conditions (SDC 4 (Supplemental Digital Content 1, http://links.lww.com/MSS/A270), Table 2, tables summarizing the statistical analysis for the effect of mechanism covariates on sprint mean power); however, abdominal cramp was unlikely to be a pure independent predictor. The metabolic covariates that clearly altered the magnitude-based inference to the effect of treatment on power (see Supplemental Digital Content 4, http://links.lww.com/MSS/A270), Tables 2 and 3, tables summarizing the statistical analysis for the effect of mechanism covariates on sprint mean power) were as follows:

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increased EFO rate in the 1.25 versus 0.5 ratio and 0.8 versus 1.25 ratio contrasts, increased exogenous-glucose and total exogenous-CHO oxidation rate in the 0.8 versus 1.25 ratio contrast, and increased exogenous-CHO oxidation efficiency in the 0.8 versus 1.25 ratio contrast. In addition, altered endogenous-CHO oxidation rate was a substantial effector of the effect of ratio on power in all ratio contrasts, whereas total-CHO oxidation rate increased power in the 0.8 versus 0.5 ratio contrast but decreased power in the 0.8 versus 1.25 ratio contrast.

DISCUSSION

By clamping drink energy content and osmolality, we showed the greatest benefits to exogenous CHO delivery as measured by end point oxidation and high-intensity endurance performance with fructose–maltodextrin–glucose formulated to a ratio of 0.8 compared with the 0.5 and 1.25 ratio formulations. Total exogenous-CHO oxidation rate and net exogenous-CHO oxidation efficiency were highest with a 0.8 ratio drink versus 0.5 and 1.25 ratio drinks; these effects were characterized by higher EFO efficiency and a moderate enhancement of high-intensity endurance performance. Despite the higher exogenous-CHO oxidation outcome implying faster intestinal absorption, the effect of fructose–maltodextrin–glucose ratio on fluid absorption rate was inconclusive. All fructose–maltodextrin–glucose drinks were ingested at a rate marginally above that estimated to be optimal for performance (34) and produced very low level gut discomfort, including less nausea and abdominal cramps than the noncaloric control. The leading qualifying mechanisms to explain the effect of CHO ratio on power were abdominal cramps, total and endogenous CHO oxidation rate, and drink sweetness.

The dual isotope approach and control of drink osmolality and CHO concentration allowed inference to the monosaccharide most responsible for the combined benefits of the 0.8 ratio drink over the other the lower and higher fructose–maltodextrin–glucose ratio drinks. Increases both in exogenous fructose oxidation efficiency and exogenous glucose oxidation rate with the 0.8 ratio drink resulted in 6%–13% higher total exogenous-CHO oxidation rates compared with the other CHO drinks (Fig. 4). Examining the metabolism of the individual monosaccharides revealed higher exogenous-glucose oxidation rate and efficiency with the 0.8 ratio drink despite the 0.5 ratio glucose-ingestion rate being 20% higher. Also, only a small possible decline in glucose oxidation efficiency relative to the 1.25 ratio was evident. These data suggest enhanced synergistic glucose absorption and metabolism coupled to higher EFO efficiency with a 0.8 ratio drink. The clear dose–response saturation of exogenous-glucose oxidation rate without evidence for a similar saturation phenomena with exogenous fructose suggests mucosal glucose absorption processes as the most important mechanism defining peak total exogenous-CHO oxidation efficiency

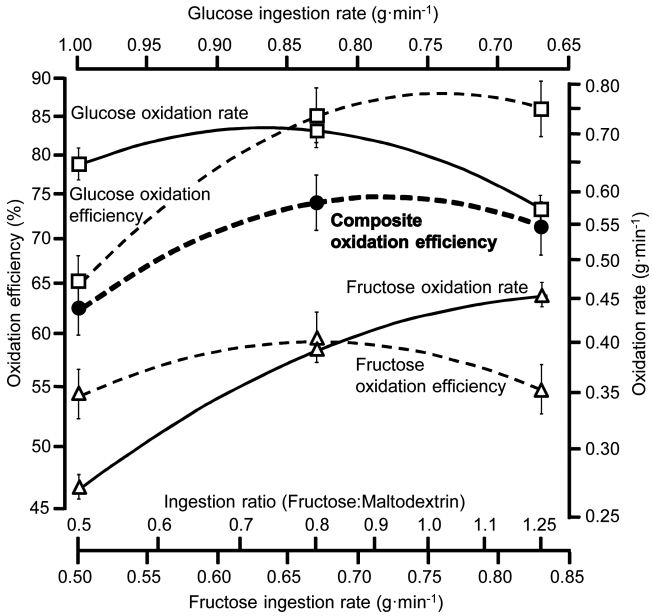

FIGURE 4—Integrated model for the mean oxidation rate and the mean efficiency of the oxidation of exogenous fructose, exogenous glucose, and the composite (combined) total exogenous CHO ingested in the three experimental fructose–maltodextrin–glucose ratio drinks during endurance exercise. Curves are quadratics derived from the solution of a mixed model of the within-subject mean value for each level of treatment by independent variable.

and absorption (Fig. 4). Inference would therefore suggest that interventions on mucosal glucose-specific absorption mechanisms offer the best chance for enhancing epithelial total CHO absorption.

The average peak oxidation rate of a single CHO ingested at high rates during exercise is no more than 1.0–1.1 g·min^{-1} (16) (although mean oxidation rates are typically 0.2–0.4 g·min^{-1} lower), which is within the range of SGLT1 glucose transporter saturation estimated from intubation studies of between 0.81 (27) and 1.7 g·min^{-1} (11). Both the 0.8 and the 1.25 ratios, therefore, may have approximated average maximal saturation conditions for active glucose transport, whereas the 0.5 ratio may have eclipsed or come close to the average absorption maximum, thereby providing a plausible explanation for the moderate sized drop in glucose oxidation efficiency (Figs. 3B and 4). However, it does not explain the small increase in absolute glucose oxidation rate with the 0.8 ratio, suggesting that factors more influential than mucosal transporter saturation determine the effect of fructose–maltodextrin–glucose ratio on rates of independent sugar oxidation. Indeed, Figure 4 illustrates a leveling of mean exogenous-glucose oxidation efficiency at ~0.75 g·min^{-1} ingestion, indicating that with further glucose ingestion, no further increase in oxidation efficiency occurred. Second, the ratio-related maxima for fructose oxidation efficiency suggests a fructose absorption (or metabolic) synergism associated with reaching the exogenous-glucose oxidation maxima. Therefore, the current data imply that the optima for

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fructose–maltodextrin–glucose ingestion ratio lies within the ratio range ~0.8–1.0 (Fig. 4). This phenomenon is consistent whether interpreted in absolute oxidation rate or in oxidation efficiency terms. Importantly, the optima is physiologically robust and supported by previous findings (24,30) and by the classic triple-lumen experiments of Shi et al. (33). Regarding fructose absorption, the saturation point for facilitative diffusion of fructose across the brush border of the enterocyte by GLUT5 is undefined, but the present fructose oxidation efficiency peak occurring with the 0.8 ratio is consistent with the dose-dependent glucose-stimulated fructose uptake reported by Rumessen and Gudmand-Høyer (32). Synergistic absorption between fructose and glucose was illustrated elsewhere by Truswell et al. (38), who found that glucose coingested with fructose as sucrose or as fructose + glucose eliminated fructose malabsorption. Although the synergistic effect of glucose on fructose absorption has been proposed to be associated with increased solvent drag or concentration gradient (6,33), experimental evidence rather favors direct stimulation of fructose transport with increased expression and membrane content of the facilitative transporter GLUT2 presenting as the best candidate for the synergistic effect (20,22).

In addition to intestinal mechanisms of fructose absorption, events at the liver might play a role in fructose metabolism that could at least partially explain the higher total exogenous-CHO oxidation rate and oxidation efficiency with the 0.8 ratio drink. For example, Lecoultre et al. (21) examined the metabolic fate of fructose coingested with glucose at a ratio of ~0.7 during exercise and found that ~29% of ingested fructose was released into the systemic circulation as glucose, and presumably subsequently oxidized in active skeletal muscle. Furthermore, the increased oxidation of lactate derived from the ingested fructose accounted for approximately half of the ingested fructose oxidation (21). Lactate released into the systemic circulation is formed from ingested fructose in the liver (33), but also potentially from the enterocyte after absorption (3); some gluconeogenesis in the gut is also thought to occur (35). Intravenous fructose delivery shows that some three quarters of the fructose is converted to lactate, pyruvate, and glucose, while the remaining ~20% of fructose is metabolized directly in working or resting muscle (2). Therefore, assuming intestinal fructose absorption is not limited by luminal fructose concentration, the effect of fructose–maltodextrin–glucose ratio on intestinal absorption will differentially affect hepatic metabolism of fructose by controlling concentrations of this hexose and associated metabolites in the portal vein.

In contrast to our previous observations (24,36), gastrointestinal comfort was largely unaffected by drink composition during exercise, suggesting at first sight that gut comfort was an unlikely mechanism to explain the observed performance outcome. The absence of any clear treatment differences could be the result of less residual CHO remaining in the gut with all test drinks compared with the previous study (24), where ingestion of 0.5 ratio fructose–maltodextrin drink at 1.8 $g \cdot min^{-1}$ was associated with higher gut discomfort ratings compared with the 0.8 ratio drink. Similarly, severe gastrointestinal distress reported with the ingestion of 2.4 $g \cdot min^{-1}$ of single-CHO drinks associated with a large reduction in power (15,37) was likely also due to reduced gastric emptying and increased distension from high fluid secretion (25). However, in the present performance test, the small mild increase in abdominal cramping with the 0.8 ratio aligned with better performance; a similar observation was recently reported (29). Indeed, abdominal cramping during the sprints was a clear and substantial mechanism variable affecting power, with the adjusted effect of treatment accentuated with removal of the covariate (Supplemental Digital Content 4, http://links.lww.com/MSS/A270), Table 2, tables summarizing the statistical analysis for the effect of mechanism covariates on sprint mean power). In other words, increased abdominal cramping associated with increased sprint power output appears to attenuate the metabolic or other benefit associated with the higher exogenous- and total-CHO oxidation rates arising from the physiological response to 0.8 ratio fructose–maltodextrin–glucose.

Despite predicting faster fluid absorption with the 0.8 ratio drink secondary to higher osmotic draw after hypothetically higher rates of epithelial CHO transport, we detected mostly inconclusive effects of treatment. These results might be explained simply as excessive noise introduced by multiple pipetting steps in the analysis of D_2O blood accumulation or insufficient sensitivity (too low tracer–tracee ratio). Alternatively, the true effect of CHO ratio on intestinal fluid absorption could be trivial, suggesting that the site for the mechanism of action to explain the increased exogenous-CHO oxidation rate with 0.8 ratio ingestion could be due to undefined metabolic events in the liver.

Previously, Rowlands et al. (30) observed an effect of fructose–glucose ratio on sweetness and palatability, leading to the suggestion that drink sweetness might positively affect performance via the activation of brain mechanisms and brain centers associated with reward and motivation (5). In the present study, sweetness was a substantial candidate mechanism affecting sprint mean power. In addition, the higher sweetness perception rating with the 0.8 ratio drink relative to the 0.5 ratio drink during the 2-h preload signifies an associative mechanism, although this association was trivial in the sprints. Further, the likely small increase in sweetness perception with the 1.25 ratio drink during the sprints implies a negative association with performance. The current data suggest a mechanism; a carefully designed future study is required to verify whether there is a meaningful effect of drink sweetness due to fructose–maltodextrin–glucose ratio on performance.

To conclude, using a dual isotope approach, we showed that the ingestion of a 0.8:1 ratio fructose–maltodextrin–glucose energy-hydration beverage during prolonged intense exercise increases exogenous-CHO oxidation efficiency and total CHO oxidation relative to 0.5 and 1.25 ratio beverages and lowers endogenous-CHO oxidation rate, relative to the

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1.25 ratio beverages. Modeling suggested fructose–maltodextrin–glucose ratios of between 0.8 to unity are oxidized with highest efficiency relative to the ingestion rate. The CHO metabolic responses were associated with a very likely moderate enhancement of mean sprint power with total- and endogenous-CHO oxidation rate, abdominal cramps, and drink sweetness presenting as candidate explanatory mechanisms. Therefore, oral CHO-hydration formulations containing fructose–maltodextrin–glucose at a ratio of around 0.8–1.0 may provide the most practical benefit for endurance athletes.

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