Pectin-Alginate Does Not Further Enhance Exogenous Carbohydrate Oxidation in Running

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ABSTRACT

BARBER, J. F. P., J. THOMAS, B. NARANG, A. HENGIST, J. A. BETTS, G. A.WALLIS, and J. T. GONZALEZ. Pectin-Alginate Does Not Further Enhance Exogenous Carbohydrate Oxidation in Running. *Med. Sci. Sports Exerc.*, Vol. 52, No. 6, pp. 1376–1384, 2020. **Purpose:** Maximizing carbohydrate availability is important for many endurance events. Combining pectin and sodium alginate with ingested maltodextrin–fructose (MAL + FRU + PEC + ALG) has been suggested to enhance carbohydrate delivery via hydrogel formation, but the influence on exogenous carbohydrate oxidation remains unknown. The primary aim of this study was to assess the effects of MAL + FRU + PEC + ALG on exogenous carbohydrate oxidation during exercise compared with a maltodextrin–fructose mixture (MAL + FRU). MAL + FRU has been well established to increase exogenous carbohydrate oxidation during cycling compared with glucose-based carbohydrates (MAL + GLU). However, much evidence focuses on cycling, and direct evidence in running is lacking. Therefore, a secondary aim was to compare exogenous carbohydrate oxidation rates with MAL + FRU versus MAL + GLU during running. **Methods:** Nine trained runners completed two trials (MAL + FRU and MAL + FRU + PEC + ALG) in a double-blind, randomized crossover design. A subset ($n = 7$) also completed a MAL + GLU trial to address the secondary aim, and a water trial to establish background expired $^{13}CO_2$ enrichment. Participants ran at 60% $\dot{V}O_2$peak for 120 min while ingesting either water only or carbohydrate solutions at a rate of 1.5 g carbohydrate per minute. **Results:** At the end of 120 min of exercise, exogenous carbohydrate oxidation rates were 0.9 (SD 0.5) g·min^{-1} with MAL + GLU ingestion. MAL + FRU ingestion increased exogenous carbohydrate oxidation rates to 1.1 (SD 0.3) g·min^{-1} ($P = 0.038$), with no further increase with MAL + FRU + PEC + ALG ingestion (1.1 (SD 0.3) g·min^{-1}; $P = 1.0$). No time–treatment interaction effects were observed for plasma glucose, lactate, insulin, or nonesterified fatty acids, or for ratings of perceived exertion or gastrointestinal symptoms (all, $P > 0.05$). **Conclusion:** To maximize exogenous carbohydrate oxidation during moderate-intensity running, athletes may benefit from consuming glucose(polymer)–fructose mixtures over glucose-based carbohydrates alone, but the addition of pectin and sodium alginate offers no further benefit. **Key Words:** FRUCTOSE, GLUCOSE, HYDROGEL, METABOLISM, SPORTS NUTRITION

Carbohydrate availability is a key determinant of endurance exercise performance. Low muscle and liver glycogen concentrations are strongly associated with fatigue during prolonged, moderate-to-high intensity exercise (1,2). The ingestion of carbohydrate during exercise provides an additional (exogenous) source of carbohydrate, which can prevent or attenuate the decline in liver (3), and sometimes muscle (4,5), glycogen contents. Increasing exogenous carbohydrate oxidation via altering the dose or type of carbohydrates ingested can improve endurance performance (6–9). Strategies to maximize the ability to digest, absorb, and oxidize ingested carbohydrate are therefore a priority for endurance athletes during competition.

One well-established strategy for increasing exogenous carbohydrate oxidation rates during exercise is the coingestion of glucose–fructose mixtures (10–12). When compared with glucose-based carbohydrates alone, isocaloric coingestion of fructose with glucose-based carbohydrates typically increases peak exogenous carbohydrate oxidation rates from ~1 to ~1.7 g·min^{-1} (13), which is thought to be (in part) due to fructose being absorbed by an additional intestinal transport route (GLUT5), and thereby bypassing the limiting step of intestinal glucose transport (primarily SGLT1) (14). A recent innovation in commercial carbohydrate sports drinks is the inclusion of pectin and sodium alginate alongside maltodextrin and fructose (15). When combined with water, this mixture can create a hydrogel upon exposure to a low pH environment such as the stomach (16). It is hypothesized that the hydrogel will allow for greater rates of gastric emptying via a reduction in nutrient sensing and thus increase intestinal carbohydrate delivery and absorption, thereby facilitating improvements in endurance performance (15). Although some evidence does indicate that the addition of pectin could accelerate gastric emptying during

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enteral feeding (17), other studies that have added either pectin to a meal (18) or alginate to meal preloads (19) demonstrate that each of these can *delay* gastric emptying at rest.

To date, only two studies have been conducted in which ingesting carbohydrate hydrogel has been compared with typical carbohydrate ingestion during exercise. These recent studies indicate no benefit to preloaded incremental time-to-exhaustion during running, or preloaded repeated sprint cycling performance with the ingestion of a maltodextrin–fructose–hydrogel, over maltodextrin–fructose alone (16,20). It is possible, however, for hydrogels to only be relevant in specific contexts, such as when gastric emptying and carbohydrate availability are both contributing to limiting performance. This scenario may occur with high exercise intensities (>80% $\dot{V}O_{2peak}$), combined with a prolonged duration (>90 min), such as elite marathon racing. Methodological limitations mean that it is not yet possible to accurately assess exogenous carbohydrate oxidation at such intensities. Therefore, the current best approach to understand the physiology of carbohydrate hydrogels is likely to be to understand the metabolic responses at moderate-intensity exercise, combined with performance and gut comfort responses at race pace. This approach has been historically fruitful, as the primary principles of glucose–fructose mixtures were developed with data collected at moderate-intensity exercise (12) and have translated well into performances during high-intensity exercise (21). It is yet to be established whether a maltodextrin–fructose–hydrogel can enhance exogenous carbohydrate oxidation during exercise. It is also interesting to note that direct comparisons of exogenous carbohydrate oxidation from glucose plus fructose ingestion versus glucose alone have, to date, only been made during cycling-based exercise (13,22). Given the substantial metabolic differences and the potential for differences in gastrointestinal function with the mechanical action of running compared with cycling (23), evidence derived from cycling cannot necessarily be extrapolated to running.

Therefore, the primary aim of the present study was to assess whether the addition of sodium alginate and pectin to a maltodextrin–fructose mixture enhances exogenous carbohydrate oxidation rates during running. A secondary aim was to assess whether a maltodextrin–fructose mixture enhances exogenous carbohydrate oxidation rates during running, when compared with isocaloric ingestion of glucose-based carbohydrates alone. It was hypothesized that a maltodextrin–fructose mixture would enhance exogenous carbohydrate oxidation rates compared with maltodextrin–glucose ingestion, and that the addition of sodium alginate and pectin to a maltodextrin–fructose mixture would further increase exogenous carbohydrate oxidation rates.

METHODS

Study design. All participants completed preliminary testing followed by two main trials to address the primary aim, in a randomized, double-blind, crossover design separated by 7–14 d (n = 9). During main trials, participants ingested a maltodextrin–fructose mixture either without (MAL + FRU) or with pectin and sodium alginate to create a hydrogel (MAL + FRU + PEC + ALG). Trials were conducted at the University of Bath, in accordance with the latest version of the Declaration of Helsinki and following institutional ethical approval (MSES 18/19-001). Participants provided informed written consent before participation. Randomization was performed by J.T.G. with online software (https://www.randomizer.org). Blinding and preparation of the test drinks was performed by an assistant who was not involved in the exercise tests.

Two subgroups of participants (n = 7) also completed an additional trial with the ingestion of either glucose-based carbohydrates alone (MAL + GLU) or water alone (WATER) to address the secondary aim and to determine background $^{13}CO_2$ breath enrichment for calculation of exogenous carbohydrate oxidation rates, respectively.

Participants. Ten trained male runners were recruited to the study (>1-yr training in endurance running), but because of dropouts, nine participants completed the two main trials (MAL + FRU and MAL + FRU + PEC + ALG), and seven participants completed the MAL + GLU and the WATER trial, respectively (Table 1). Exclusion criteria included the following: metabolic or gastrointestinal disorders, smokers, or failure to pass a physical activity readiness questionnaire. Women were excluded on the rationale of studying a homogenous population because there are potential sex differences in gastric emptying (24).

Preliminary testing. Participants' height (Leicester Height Measure; Seca GmbH, Hamburg, Germany) and mass (Tanita, Tokyo, Japan) were measured. To determine running economy and peak oxygen consumption ($\dot{V}O_{2peak}$), participants completed a graded exercise test to exhaustion on a motorized treadmill (Ergo ELG70; Woodway, Weil am Rhein, Germany). Participants initially ran for 4 × 4 min on a 0% gradient to establish the relationship between O_2 uptake and running speed (8–12 km·h^{-1}) on a flat treadmill. After a 5-min rest, participants then began the exhaustive test, whereby the treadmill speed was fixed (at a speed based on participants perception in the 4-min stages) and the gradient was increased by 3% every 3 min, starting from a 1% gradient, until volitional exhaustion. The running speed, which elicited 60% $\dot{V}O_{2peak}$, was interpolated and used for prescribing running velocity during the experimental visits.

Replication of usual diet and physical activity. The approach to replication of usual diet and physical activity was based on the balance between reducing day-to-day variability while minimizing participant burden (25). Participants recorded diet and exercise for 2 d before the first experimental

TABLE 1. Participant characteristics.

	Characteristics
Age	22 (18–30) yr
Body mass	69 (61–74) kg
Height	1.82 (1.74–1.88) m
$\dot{V}O_{2peak}$	63 (56–72) mL·min^{-1}·kg^{-1}
Running speed to elicit 60% $\dot{V}O_{2peak}$	10.7 (9.3–11.8) km·h^{-1}

Data are means (ranges).

trial and replicated these before subsequent trials. During this time, participants refrained from consuming foods with a high natural abundance of ^{13}C to minimize background shifts in ^{13}C enrichment of expired gas arising from endogenous carbohydrate stores being oxidized during exercise. For 24 h before each visit, participants refrained from strenuous exercise, caffeine, and alcohol. Participants also fasted for 8 h before each experimental visit. Participants were reminded of these protocols 5 and 3 d before trials. Participants were also reminded of the fasting period 24 h before trials. Adherence to these protocols was confirmed verbally with participants before each trial. This relatively modest method was thought to be appropriate for the current study design, as the primary outcome measure (exogenous carbohydrate oxidation) has been shown to be unaffected by preexercise glycogen status (26), which would be influenced by dietary carbohydrate intake and physical activity levels.

Main trials. Participants arrived at the laboratory after pretrial standardization (confirmed by verbal questioning) and at a similar time of day within participants (±1 h). After a 5-min flush period (to washout dead space in tubing and familiarize participants), a 5-min sample of expired breath was taken using the Douglas bag method, and an additional breath sample was collected into an exetainer for analysis of ^{13}C enrichment. A cannula was then inserted into an antecubital vein, and a resting blood sample was drawn. Participants then ran for 2 h at a speed eliciting 60% $\dot{V}O_{2peak}$. The run was performed in standard environmental conditions (17°C–22°C dry bulb temperature, 40%–65% relative humidity), and participants were fan cooled throughout.

Carbohydrate drinks. On all trials other than the WATER trial, participants ingested 140 mL of a 16% w/v solution upon initiating running, and then every 15 min until 105 min providing an average intake of 1.5 g carbohydrate per minute. The rate of carbohydrate intake was chosen to align with guidelines for prolonged exercise. Because the solution concentration may affect the ability to form a hydrogel in the stomach, this meant that fluid intake could not be tailored to expected sweat losses. This may have resulted in a slight hypohydration on all trials. The MAL + GLU drink provided 0.87 g maltodextrin per minute and 0.63 g dextrose per minute, whereas both the MAL + FRU and MAL + FRU + PEC + ALG drinks provided 0.87 g maltodextrin per minute and 0.63 g fructose per minute. The ratio of fructose/glucose to maltodextrin was dictated by that present in the commercially available product at the time of testing. Systematic review indicates that a ratio closer to unity might be more optimal for balancing exogenous oxidation, gut comfort, and performance (14). MAL + GLU and MAL + FRU had 1 g sodium chloride per liter added to match the MAL + FRU + PEC + ALG drink. Consistent with the manufacturer's instructions, all drinks were made with low-calcium water (<40 $mg \cdot L^{-1}$).

To quantify exogenous carbohydrate oxidation, carbohydrates with a high natural abundance of ^{13}C were used. The natural ^{13}C abundance of the MAL + GLU, MAL + FRU, and MAL + FRU + PEC + ALG were −11.37, −11.20 and −11.86 δ‰ versus Pee Dee Bellemnitella, respectively. Maltodextrin, dextrose (both MyProtein, Cheshire, United Kingdom), and fructose (PeakSupps, Bridgend, United Kingdom) were purchased as raw materials and mixed accordingly, whereas the MAL + FRU + PEC + ALG was purchased as a commercially available finished product (Maurten, Gothenburg, Sweden).

Expired breath analysis. Expired breath samples were analyzed using the Douglas bag method to establish rates of oxygen consumption and carbon dioxide production. At rest, a 5-min sample was collected after a 5-min equilibration period. During exercise, 1-min samples were taken after 1-min equilibration periods. Concurrently, ambient O_2 and CO_2 concentrations were measured to account for changes in inspired gas concentrations (27). Concentrations of O_2 and CO_2 were measured in a known volume of sample (Mini MP 5200, Servomex Ltd., Crowborough, United Kingdom), and the total volume of expired gas was determined by evacuation using a dry gas meter (Harvard Apparatus, Holliston, MA). To determine ^{13}C enrichment of expired CO_2, breath samples were collected in 10-mL exetainers (Labco Ltd, Lampeter, United Kingdom), filled in duplicate by 10-s exhalation into a discard bag (Quintron Inc, Milwaukee, WI). At rest, participants exhaled for 20 s to ensure sufficient collection of expired gas.

Whole-body substrate oxidation was calculated from $\dot{V}O_2$ and $\dot{V}CO_2$ according to stochiometric equations (28,29). The $^{13}C/^{12}C$ ratio of expired CO_2 was determined by continuous flow isotope ratio mass spectrometry, and the enrichment was expressed as δ per mil difference between the $^{13}C/^{12}C$ ratio of the sample and a known standard (30). The $\delta^{13}C$ was related to an international standard from which exogenous carbohydrate oxidation was calculated according to the following equation (31):

$$\text{exogenous carbohydrate oxidation} = \dot{V}CO_2 \left(\frac{\delta Exp - \delta EXP_{bkg}}{\delta Ing - \delta Exp_{bkg}} \right) \left(\frac{1}{k} \right)$$

where δExp is the ^{13}C enrichment of expired CO_2, δIng is the ^{13}C enrichment of the drink, and δEXP_{bkg} is the ^{13}C enrichment of expired CO_2 during the WATER trial. For participants who did not complete a WATER trial, the group mean of the other participants was used for δEXP_{bkg}. k is the $\dot{V}CO_2$ with the oxidation of 1 g of glucose (0.7467 L $CO_2 \cdot g^{-1}$).

Some ^{13}C can be trapped within the bicarbonate pool with implications for the quantification of exogenous carbohydrate oxidation. However, during exercise, the increase in CO_2 production results in a rapid equilibration of expired $^{13}CO_2$ with the $^{13}CO_2/H^{13}CO_3^-$ pool and recovery of $^{13}CO_2$ from oxidation approaches 100% after 20 min of exercise at ~60 $\%\dot{V}O_{2peak}$ (unpublished observations). Therefore, calculations on substrate oxidation were performed on data from 30 min of exercise onwards.

Blood sampling and analysis. Venous blood samples (10 mL) were taken at rest and at 15, 30, 60, 90, and 120 min

of exercise. Samples were collected into EDTA-containing tubes (Sarstedt, Nümbrecht, Germany) and centrifuged for 10 min at 4000*g* and 4°C. Aliquots of plasma were stored at −80°C before analysis. Because of cost implications, only blood samples from the trials that related to the primary aim were analyzed (MAL + FRU and MAL + FRU + PEC + ALG trials). Plasma was analyzed for glucose and lactate using an automated analyzer (RX Daytona, Randox, United Kingdom). Insulin (IBL International, Hamburg, Germany) and nonesterified fatty acid (NEFA) concentrations (WAKO Diagnostics, Richmond, VA) were analyzed by enzyme-linked immunosorbent assay and colorimetric assays, respectively. For all analyses, intra-assay and interassay coefficients of variation were less than 10%.

Subjective ratings. Ratings of gastrointestinal distress were measured on a 7-point scale adapted from the Gastrointestinal Symptoms Rating Scale (32). Four questions related to upper, three to central, and two to lower gastrointestinal symptoms. The Gastrointestinal Symptoms Rating Scale has adequate internal consistence ($\alpha > 0.61$), construct, and discriminant validity, and is suitable for comparisons over 6 wk (32). Because these ratings are subjective and cannot therefore be readily compared between groups of people, only data for the primary comparison (MAL + FRU vs MAL + FRU + PEC + ALG) are presented.

Statistical analysis. An *a priori* sample size estimate was performed based on the effect size (Cohen's *d*) of exogenous carbohydrate oxidation rates in response to glucose–fructose coingestion compared with glucose alone based on the following equations:

$$d = \frac{\text{mean}_{\text{experimental}} - \text{mean}_{\text{control}}}{\text{SD}_{\text{pooled}}}$$

where

$$\text{SD}_{\text{pooled}} = \sqrt{\frac{(n_{\text{control}} - 1)\text{SD}^2_{\text{control}} + (n_{\text{experimental}} - 1)\text{SD}^2_{\text{experimental}}}{n_{\text{control}} + n_{\text{experimental}} - 2}}$$

Peak exogenous carbohydrate oxidation rates from glucose ingestion alone have been reported to be 1.06 (SD 0.11) g·min^{-1} compared with 1.75 (SD 0.31) g·min^{-1} with glucose–fructose coingestion ($n = 8$, in a crossover design) (12). Using this effect size ($d = 2.49$), five participants should provide power > 95% to detect a difference with a two-tailed test and an α level of 0.05. To ensure adequate power with the potential for dropouts, we aimed to recruit at least seven participants.

Data were analyzed using Prism (v 8.2.1; GraphPad, San Diego, CA) and SPSS (v24; IBM, Armonk, NY). Data expressed over time (e.g., expired $^{13}CO_2$ enrichment, exogenous carbohydrate oxidation rates, $\dot{V}O_2$, VCO_2, RER, plasma metabolite and hormone concentrations, RPE, and gastrointestinal symptom ratings) were analyzed by repeated-measures ANOVA or mixed-effects model as appropriate. Summary statistics (e.g., peak exogenous carbohydrate oxidation rates, the percentage contribution of substrates to total energy expenditure) were analyzed by one-way ANOVA or two-tailed, paired *t*-tests with Bonferroni correction, as appropriate. An exploratory analysis was performed to assess whether baseline differences in NEFA concentrations were driving differences in whole-body substrate use by analysis of covariance on whole-body fat oxidation rates with baseline plasma NEFA concentrations as the covariate. Furthermore, data were checked for order effects by repeated-measures ANOVA (trial order–time interaction) and one-way ANOVA (trial order) as appropriate. All data are expressed as means (SD) in the text and tables, and as means ± 95% confidence interval (CI) in figures, other than subjective data, which are presented as medians ± 95% CI. Differences were considered significant if $P \leq 0.05$.

RESULTS

Substrate oxidation and gas exchange. No order effects were detected for either expired $^{13}CO_2$ enrichments (trial order: $P = 0.59$; trial order–time interaction effect: $P = 1.0$) or exogenous carbohydrate oxidation rates (trial order: $P = 0.61$; trial order–time interaction effect: $P = 1.0$). Furthermore, no order effects were detected for the total amount of fat ($P = 0.62$), endogenous carbohydrate ($P = 0.38$), or exogenous carbohydrate oxidized ($P = 0.93$). Expired $^{13}CO_2$ enrichments increased during exercise (time effect, $P < 0.001$) and were higher during MAL + FRU compared with MAL + GLU (treatment effect, $P < 0.001$; *post hoc* comparison, $P < 0.001$), with no further increase seen with MAL + FRU + PEC + ALG compared with MAL + FRU ($P = 0.11$; Fig. 1A). Differences across time were detected between the WATER trial and the carbohydrate drink treatments (time–treatment interaction, $P < 0.001$). Exogenous carbohydrate oxidation rates increased over time (time effect, $P < 0.001$), and to a greater extent with both of the fructose-containing drinks compared with MAL + GLU (time–treatment interaction, $P < 0.001$; Fig. 1B). At the end of exercise, exogenous carbohydrate oxidation rates were higher with MAL + FRU compared with MAL + GLU ($P = 0.04$), but not further increased by MAL + FRU + PEC + ALG ($P = 1.0$). The exogenous oxidation rate expressed relative to ingestion rate at this time point equated to 59% (SD 19%), 70% (SD 19%), and 71% (SD 21%) with MAL + GLU, MAL + FRU, and MAL + FRU + PEC + ALG, respectively. Peak exogenous carbohydrate oxidation rates were 0.92 (SD 0.29), 1.08 (SD 0.26), and 1.11 (SD 0.31) g·min^{-1} with MAL + GLU, MAL + FRU, and MAL + FRU + PEC + ALG, respectively (all, $P > 0.05$).

During MAL + GLU and MAL + FRU trials, fat oxidation was 234 (SD 50) and 165 (SD 83) kcal·h^{-1}, respectively ($P = 0.14$). Fat oxidation was 255 (SD 120) kcal·h^{-1} during the MAL + FRU + PEC + ALG trial, which was higher than that during the MAL + FRU trial ($P = 0.04$). During the MAL + GLU and MAL + FRU trials, endogenous carbohydrate oxidations were 525 (SD 89) and 530 (SD 99) kcal·h^{-1}, respectively ($P = 0.93$). During MAL + FRU + PEC + ALG trial, endogenous carbohydrate oxidation was lower compared with MAL + FRU (434 (SD 112) kcal·h^{-1}, $P = 0.05$). During MAL + GLU, exogenous carbohydrate oxidation was 165 (SD 60) kcal·h^{-1}. MAL + FRU increased exogenous carbohydrate

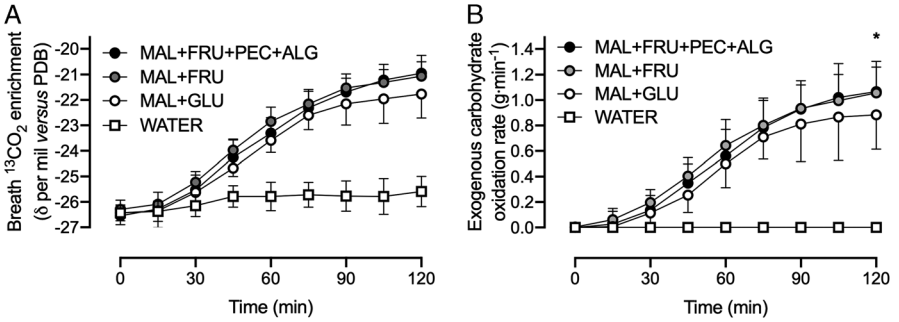

FIGURE 1—Breath $^{13}CO_2$ enrichment (A) and exogenous carbohydrate oxidation rates (B) during 120 min of running at 60% $\dot{V}O_{2peak}$ with the ingestion of water (WATER; n = 7) or 1.5 g·min^{-1} of carbohydrate in the form of maltodextrin plus glucose (MAL + GLU; n = 7), maltodextrin plus fructose (MAL + FRU; n = 9), or maltodextrin plus fructose with pectin and sodium alginate (MAL + FRU + PEC + ALG; n = 9). Data are means (error bars: 95% CI). *P < 0.05 for MAL + GLU vs MAL + FRU.

oxidation to 201 (SD 66) kcal·h^{-1} (P = 0.05), with no further increase from MAL + FRU + PEC + ALG ingestion (193 (SD 66) kcal·h^{-1}; P = 0.66).

When expressed as the contribution to total energy expenditure, fat oxidation contributed ~20%–25% of total energy expenditure during the MAL + GLU and MAL + FRU trials and increased to ~30% of total energy expenditure during the MAL + FRU + PEC + ALG trial (P = 0.02; Fig. 2). However, this increase in fat oxidation as a contribution to total energy expenditure between MAL + FRU and MAL + FRU + PEC + ALG (mean difference, 10.7%; 95% CI, 0.2% to 21.1%) did not remain after baseline NEFA concentrations were added as a covariate (adjusted mean difference, 7.8%; 95% CI, −0.6% to 16.1%; P = 0.07). Endogenous carbohydrate oxidation contributed ~60% of total energy expenditure during the MAL + GLU and MAL + FRU trials, and decreased to ~50% of total energy expenditure during the MAL + FRU + PEC + ALG trial (P = 0.03; Fig. 2). Exogenous carbohydrate oxidation contributed ~18% of total energy expenditure during MAL + GLU and increased to ~22% of total energy expenditure during MAL + FRU (P = 0.05; Fig. 2). Exogenous carbohydrate oxidation was not further increased with MAL + FRU + PEC + ALG compared with MAL + FRU (P = 0.71; Fig. 2).

$\dot{V}O_2$, $\dot{V}CO_2$, and RER all displayed main effects of time (all, P < 0.05), but no treatment effects were detected (all, P > 0.29; P = 0.08 for RER), and no differences over time were detected (time–treatment interaction effects; all, P > 0.45; Fig. 3).

Plasma insulin and metabolite concentrations. Plasma glucose, lactate, and insulin concentrations all rose slightly at the onset of exercise (time effect for all, P < 0.01), to a similar extent across time in both MAL + FRU and MAL + FRU + PEC + ALG trials (treatment effect and time–treatment interaction; all, P > 0.20; Figs. 4A–C, respectively). Plasma NEFA concentrations were ~0.13 mmol·L^{-1} higher at baseline in the MAL + FRU + PEC + ALG trial compared with the MAL + FRU trial (P = 0.03; Fig. 4D). During exercise, plasma NEFA concentrations declined (time effect, P < 0.001), to a similar level across time in both trials (treatment effect and time–treatment interaction; both, P = 0.12).

Subjective ratings. RPE, and upper, central, and lower gastrointestinal symptom ratings all increased throughout exercise (time effect; all, P < 0.01), to a similar extent across time in both trials (treatment effect and time–treatment interaction; all, P > 0.07; Figs. 5A–D, respectively).

DISCUSSION

The present data demonstrate that, when ingesting carbohydrates at 90 g·h^{-1} during running, the addition of pectin and sodium alginate to ingested glucose–fructose does not further enhance exogenous carbohydrate oxidation rates when compared with a glucose–fructose mixture alone. However, ingestion of glucose–fructose mixture can enhance exogenous carbohydrate oxidation during running when compared with isocaloric ingestion of glucose-based carbohydrates alone.

Maximizing carbohydrate availability during exercise is a key goal for many endurance athletes (22). A novel nutrient blend of sodium alginate and pectin, combined with a

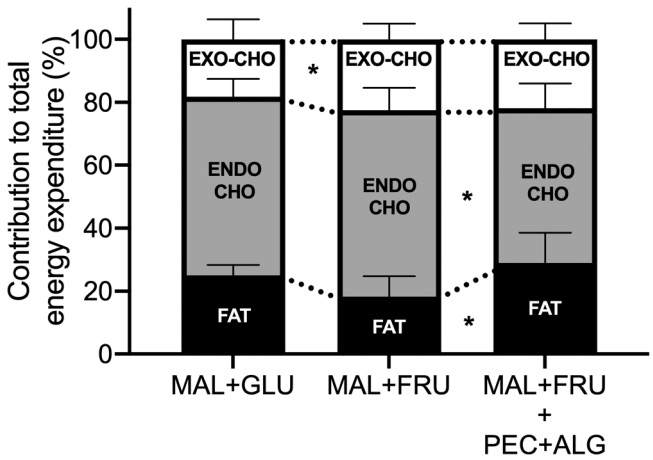

FIGURE 2—Whole-body fat (FAT), endogenous carbohydrate (ENDO CHO), and exogenous carbohydrate oxidation rates (EXO CHO) during 120 min of running at 60% $\dot{V}O_{2peak}$ with the ingestion of 1.5 g·min^{-1} of carbohydrate in the form of maltodextrin plus glucose (MAL + GLU; n = 7), maltodextrin plus fructose (MAL + FRU; n = 9), or maltodextrin plus fructose with pectin and sodium alginate (MAL + FRU + PEC + ALG; n = 9). Data are means (error bars: 95% CI). *P < 0.05 for differences between treatments. Data were calculated from minutes 30–120 of exercise.

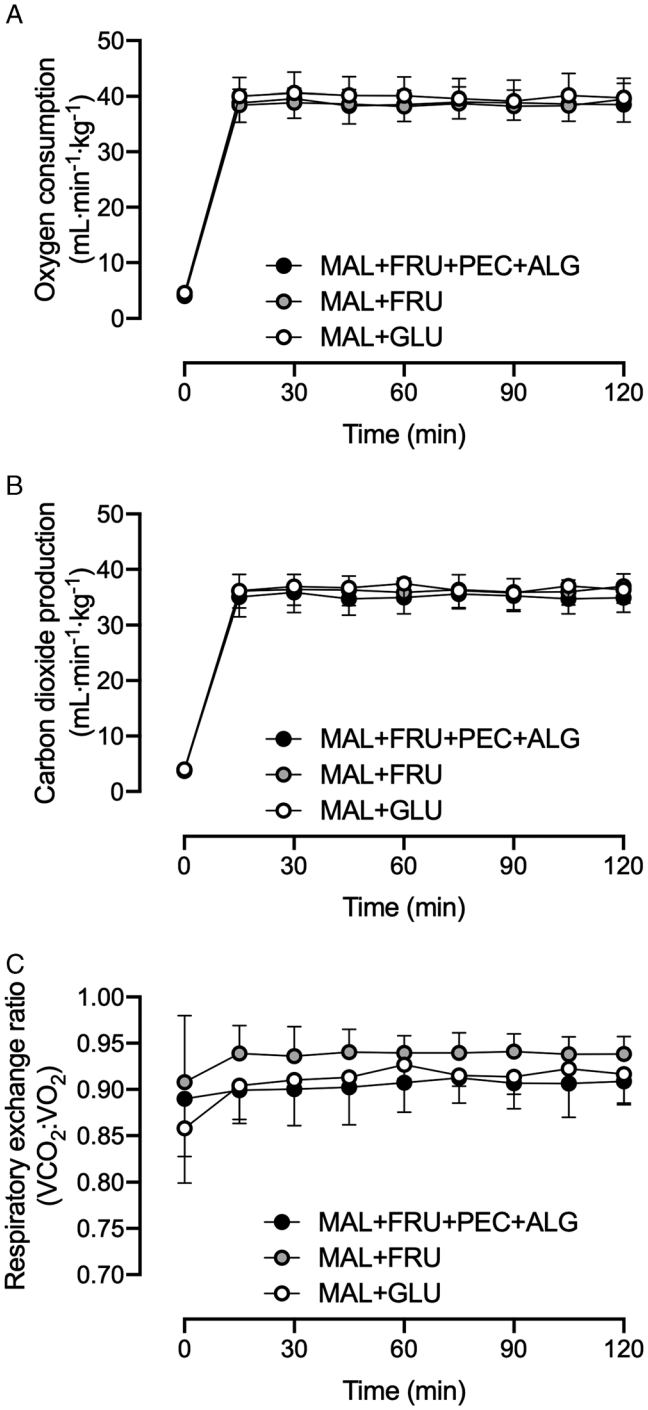

FIGURE 3—Oxygen consumption (A), carbon dioxide production (B), and respiratory exchange ratio (C) during 120 min of running at 60% $\dot{V}O_{2peak}$ with the ingestion of 1.5 g·min^{-1} of carbohydrate in the form of maltodextrin plus glucose (MAL + GLU; *n* = 7), maltodextrin plus fructose (MAL + FRU; *n* = 9), or maltodextrin plus fructose with pectin and sodium alginate (MAL + FRU + PEC + ALG; *n* = 9). Data are means (error bars: 95% CI).

maltodextrin–fructose mixture, has recently been developed and has been proposed to further enhance exogenous carbohydrate oxidation during exercise (15). This combination purports to produce a hydrogel when exposed to the acidic environment of the stomach, thereby encapsulating the carbohydrate (15). It is expected that this hydrogel may attenuate the reduction in gastric-emptying rates seen with large amounts of carbohydrate ingestion, thereby facilitating high exogenous carbohydrate oxidation rates during exercise. To the best of the authors' knowledge, there are currently only two randomized, controlled trials that have examined the effects of coingesting pectin and sodium alginate with carbohydrates during exercise. Both of these studies demonstrated no changes in whole-body metabolism, ratings of gut discomfort or perception of effort, or performance during running (16) or cycling (20). Consistent with this, we also observed no differences in ratings of gut discomfort or perception of effort. However, it is possible that increased exogenous carbohydrate availability above that seen with maltodextrin–fructose mixtures only enhances performance during very specific contexts. Therefore, further insight into the potential for this nutritional strategy to influence performance could be gained from establishing whether pectin and sodium alginate coingestion with carbohydrate affects exogenous carbohydrate oxidation.

In the present study, exogenous carbohydrate oxidation rates were not further increased by the coingestion of pectin and sodium alginate with a maltodextrin–fructose mixture compared with a maltodextrin–fructose mixture alone. If the mechanism by which pectin and alginate are proposed to enhance carbohydrate delivery is via accelerating gastric emptying, then the lack of effect on exogenous carbohydrate oxidation is perhaps not surprising, as gastric-emptying rates are not thought to be limiting to exogenous carbohydrate oxidation when large amounts of carbohydrate are ingested during exercise (33). These data demonstrate that there is no increase in exogenous carbohydrate availability with the coingestion of alginate and pectin with a maltodextrin–fructose mixture, and thereby can explain why recent studies have demonstrated a lack of effect on endurance performance (16,20).

It is well established that the coingestion of fructose with glucose can enhance exogenous carbohydrate oxidation rates during cycling-based exercise when compared with the coingestion of glucose-based carbohydrates alone (13,34). However, the ability to extrapolate findings from cycling to other modes of exercise is uncertain. When compared with cycling, running typically results in higher rates of fat oxidation and a concomitant decrease in whole-body carbohydrate oxidation rates (35,36). Furthermore, running is thought to pose a greater mechanical stress on the gastrointestinal system, potentially altering the capacity for intestinal absorption and thus limiting the rate of digestion, absorption, and oxidation of exogenous carbohydrate (35). Nevertheless, the only direct comparison to date of prolonged running versus cycling reported equivalent exogenous carbohydrate oxidation rates with the ingestion of a glucose–fructose mixture (35). However, in that study, participants exercised at the same relative intensity during both trials (60% $\dot{V}O_{2peak}$), resulting in a ~5% higher absolute exercise intensity (based on oxygen consumption and energy expenditure) with running versus cycling (35). The higher absolute energy cost of exercise could have driven a higher exogenous carbohydrate oxidation rate in the running trial and offset any

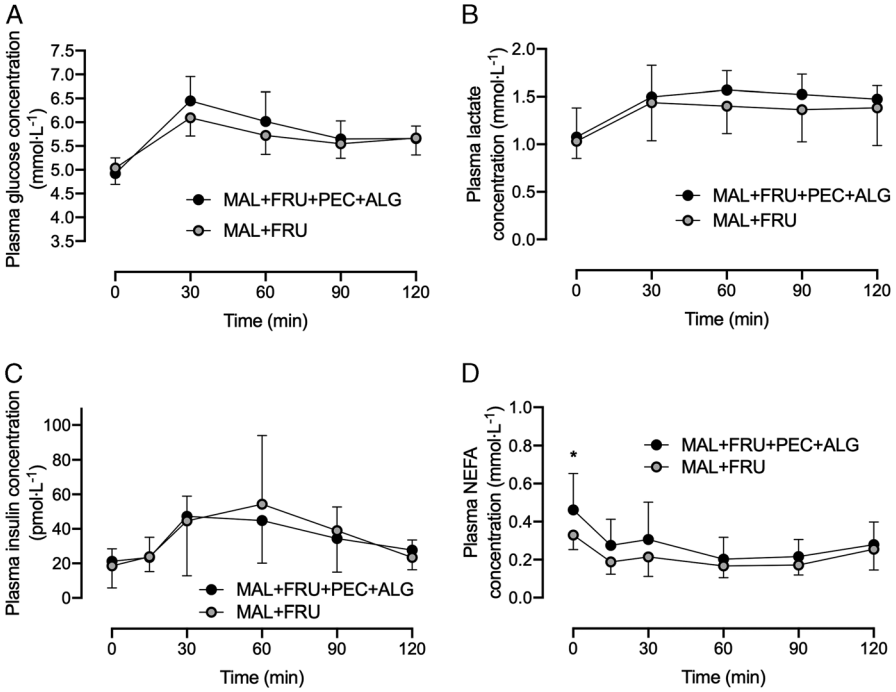

FIGURE 4—Plasma glucose (A), lactate (B), insulin (C), and NEFA (D) concentrations during 120 min of running at 60% $\dot{V}O_{2peak}$ with the ingestion of 1.5 g·min^{-1} of carbohydrate in the form of maltodextrin plus fructose (MAL + FRU; $n = 9$) or maltodextrin plus fructose with pectin and sodium alginate (MAL + FRU + PEC + ALG; $n = 9$). Data are means (error bars: 95% CI). *$P < 0.05$ for MAL + FRU vs MAL + FRU + PEC + ALG.

potential reduction in exogenous carbohydrate oxidation rates seen with running. Therefore, although the present data demonstrate that a glucose–fructose mixture can increase exogenous carbohydrate oxidation during running, it remains to be established whether running versus cycling alters the efficiency or capacity for digestion, absorption, and oxidation of exogenous carbohydrate.

Unexpectedly, during the trial where pectin and sodium alginate were coingested with a maltodextrin–fructose mixture, we observed a higher rate of fat oxidation compared with

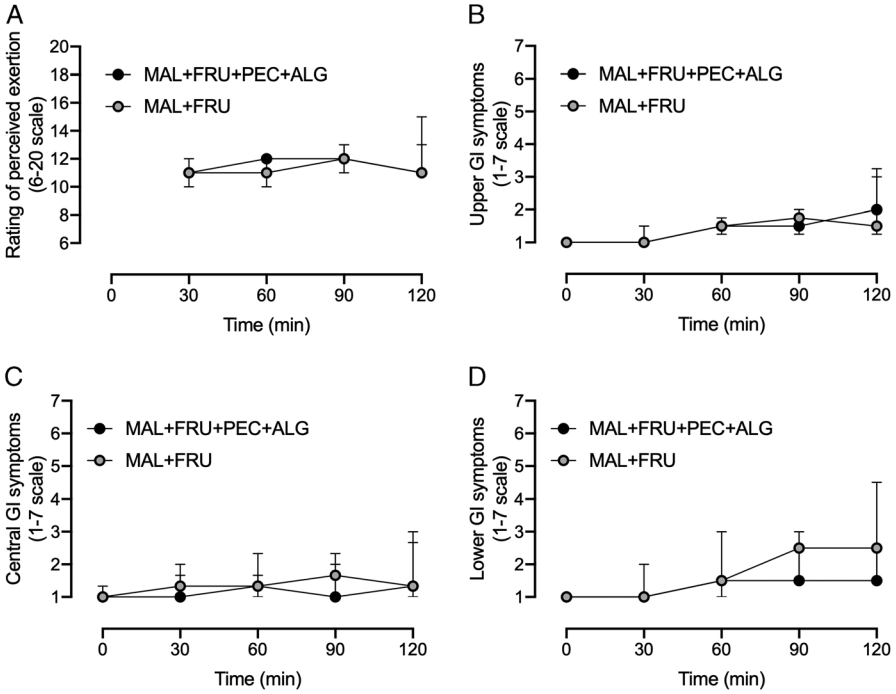

FIGURE 5—Ratings of perceived exertion (A), upper (B), central (C), and lower (D) gastrointestinal (GI) symptoms during 120 min of running at 60% $\dot{V}O_{2peak}$ with the ingestion of 1.5 g·min^{-1} of carbohydrate in the form of maltodextrin plus fructose (MAL + FRU; $n = 9$) or maltodextrin plus fructose with pectin and sodium alginate (MAL + FRU + PEC + ALG; $n = 9$). Data are medians (error bars: 95% CI).

ingestion of a maltodextrin–fructose mixture alone. Because there was no change in exogenous carbohydrate oxidation, this resulted in a reduction in endogenous carbohydrate oxidation. It is tempting to speculate that this could be a direct effect of the test drink. For example, it has been suggested that hydrogels may attenuate nutrient sensing in the proximal gastrointestinal tract (15), which would result in higher gastric-emptying rates and lower insulin secretion (37). However, plasma insulin concentrations were unaffected by the addition of pectin and sodium alginate to carbohydrate in the present study. In addition, a baseline difference was observed in plasma NEFA concentrations, which was higher in the MAL + FRU + PEC + ALG trial. Elevated baseline NEFA is one possible explanation for the higher whole-body fat oxidation in that trial (38). Indeed, when baseline NEFA concentrations are added as a covariate, the difference in fat oxidation between trials is no longer statistically significant. The reasons for this baseline difference in NEFA concentrations are not clear. Although participants were asked to replicate diet and activity in the days before trials, this was only checked by verbal confirmation, and it is possible that this was not fully adhered to. Differences in carbohydrate intake and/or physical activity levels could have caused baseline glycogen concentrations to be lower in the MAL + FRU + PEC + ALG trial. Fortunately, this is unlikely to have implications for our primary and secondary aims, as exercising with low glycogen contents does not alter exogenous carbohydrate oxidation rates (26). This highlights the importance of considering pretrial standardization with respect to the specific aims and methods of a study. If a study design requires tighter control of preexercise carbohydrate availability, then researchers should consider requesting participants to report back on the accuracy of diet and physical activity replication and/or provide food packages to facilitate adherence (25).

A potential limitation with the present study is that it was not confirmed whether the addition of pectin and sodium alginate to carbohydrate resulted in hydrogel formation within the stomach or therefore altered gastric emptying. Nevertheless, the product was made accordingly to the manufacturer's instructions, and this method has been recently shown to produce a hydrogel within a low pH environment *in vitro* (16). Furthermore, the measurement of exogenous carbohydrate oxidation encapsulates the integrated sum of gastric emptying, intestinal absorption, and oxidation of the ingested carbohydrate. Therefore, if a carbohydrate hydrogel is to enhance carbohydrate delivery and thereby performance, an increase in exogenous carbohydrate oxidation is most likely a requirement. Although the study was powered for the outcome of exogenous carbohydrate oxidation with the specified comparisons, the relatively small sample size has the potential to be underpowered for some of our other outcome measures reported. Inadequate power for some outcomes has the potential to result in either a type II error (false negative), but also overestimate the true effect size when an effect is detected. It should also be acknowledged that the exercise intensity used in the present study is not relevant to elite-level marathon running, which occurs at ~90% $\dot{V}O_{2peak}$ (39). Given the differences in gastric-emptying rates at high- versus moderate-intensity exercise (40), it is not possible to directly extrapolate the findings of the present study to exercise intensities above ~80% $\dot{V}O_{2peak}$. However, the measurement of exogenous carbohydrate oxidation also becomes problematic at high exercise intensities, and therefore, it is unlikely that measurements of exogenous carbohydrate oxidation can be made at elite-level marathon race with the current methods available.

In conclusion, when carbohydrates are ingested at rates recommended for prolonged endurance-type exercise (i.e., 90 $g \cdot h^{-1}$), maltodextrin–fructose mixtures increase exogenous carbohydrate oxidation compared with the ingestion of glucose-based carbohydrates alone. The additional ingestion of pectin and sodium alginate with a maltodextrin–fructose mixture does not further increase exogenous carbohydrate oxidation, or alter the perception of effort or ratings of gastrointestinal symptoms during moderate-intensity running. Given the technical difficulties in assessing exogenous carbohydrate oxidation at exercise intensities reflective of elite marathon racing, decisions on the use of hydrogels in elite sport should be based on the total balance of evidence from mechanistic studies at moderate-intensity exercise and performance studies at race pace, combined with careful observations in elite athletes during hard training and racing.

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