Human Kinetics

Postexercise Glucose–Fructose Coingestion Augments Cycling Capacity During Short-Term and Overnight Recovery From Exhaustive Exercise, Compared With Isocaloric Glucose

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During short-term recovery, postexercise glucose–fructose coingestion can accelerate total glycogen repletion and augment recovery of running capacity. It is unknown if this advantage translates to cycling, or to a longer (e.g., overnight) recovery. Using two experiments, the present research investigated if postexercise glucose–fructose coingestion augments exercise capacity following 4-hr (short experiment; $n = 8$) and 15-hr (overnight experiment; $n = 8$) recoveries from exhaustive exercise in trained cyclists, compared with isocaloric glucose alone. In each experiment, a glycogen depleting exercise protocol was followed by a 4-hr recovery, with ingestion of 1.5 or 1.2 $g \cdot kg^{-1} \cdot hr^{-1}$ carbohydrate in the short experiment (double blind) and the overnight experiment (single blind), respectively. Treatments were provided in a randomized order using a crossover design. Four or fifteen hours after the glycogen depletion protocol, participants cycled to exhaustion at 70% W_{max} or 65% W_{max} in the short experiment and the overnight experiment, respectively. In both experiments there was no difference in substrate oxidation or blood glucose and lactate concentrations between treatments during the exercise capacity test (trial effect, $p > .05$). Nevertheless, cycling capacity was greater in glucose + fructose versus glucose only in the short experiment (28.0 ± 8.4 vs. 22.8 ± 7.3 min, $d = 0.65$, $p = .039$) and the overnight experiment (35.9 ± 10.7 vs. 30.6 ± 9.2 min, $d = 0.53$, $p = .026$). This is the first study to demonstrate that postexercise glucose–fructose coingestion enhances cycling capacity following short-term (4 hr) and overnight (15 hr) recovery durations. Therefore, if multistage endurance athletes are ingesting glucose for rapid postexercise recovery then fructose containing carbohydrates may be advisable.

Keywords: carbohydrates, endurance, metabolism, nutrition, sugars

Carbohydrate is the dominant energy source during moderate- to high-intensity exercise (Romijn et al., 1993). Human carbohydrate stores are finite, with skeletal muscle and liver storing ~500 g (Jensen et al., 2011) and ~100 g of glycogen (Taylor et al., 1996), respectively. During moderate- to high-intensity exercise, total carbohydrate oxidation exceeds maximum exogenous carbohydrate oxidation. Therefore, during prolonged exercise, low glycogen availability is inevitable (King et al., 2018; Tsintzas et al., 2001). Low glycogen availability is associated with the onset of fatigue, demonstrated by the strong relationship between muscle (Bergstrom et al., 1967) and liver (Casey et al., 2000) glycogen content and exercise capacity. Consequently, for optimal performance, endurance athletes should aim to maximize preexercise glycogen content. Moreover, in repeated endurance bouts with short-recovery duration, the rate of glycogen repletion is the principal determinant of recovery time (Alghannam et al., 2016; Casey et al., 2000).

To maximize glycogen repletion, guidelines recommend ingesting 1.0–1.2 $g \cdot kg^{-1} \cdot hr^{-1}$ carbohydrate of moderate- to high-glycemic index during the first 4 hr of recovery, before resuming daily fuel needs (Burke et al., 2011). These guidelines are primarily based on evidence relating to maximizing muscle glycogen repletion (Betts & Williams, 2010). However, liver glycogen is also important, as it contributes ~17% to total glycogen stores and contributes to the maintenance of blood glucose availability during

exercise, a lack of which is associated with the onset of fatigue (Coyle & Coggan, 1984). In contrast to muscle glycogen, liver glycogen synthesis is potently increased by coingestion of the low-glycemic index carbohydrate, fructose, with glucose (or sucrose ingestion; a glucose–fructose disaccharide), when compared with isocaloric glucose alone (Decombaz et al., 2011; Fuchs et al., 2016, 2019). Moreover, glucose–fructose coingestion does not compromise muscle glycogen repletion (Wallis et al., 2008), resulting in greater total glycogen storage, compared with isocaloric glucose (Fuchs et al., 2016). Maunder et al. (2018) is the only study to investigate if this translates into improved exercise capacity when ingesting large quantities of carbohydrates (>1.2 $g \cdot kg^{-1} \cdot hr^{-1}$), reporting 90 g/hr fructose–maltodextrin (glucose polymer) coingestion augments 4-hr recovery of running capacity compared with maltodextrin alone.

Muscle glycogen depletion is associated with fatigue in running, whereas hypoglycemia (indicative of liver glycogen depletion) is more implicated in fatigue during cycling (Tsintzas & Williams, 1998). Therefore, it cannot be assumed that accelerated recovery of running capacity with postexercise maltodextrin–fructose coingestion (Maunder et al., 2018) translates to cycling. Furthermore, the relatively short recovery period in the prior study (4 hr) may have resulted in the ingested carbohydrate being unabsorbed at the start of exercise. Indeed, the greater exercise capacity with maltodextrin–fructose coingestion may be partially attributed to faster intestinal absorption of glucose–fructose versus glucose alone (Jentjens & Jeukendrup, 2005) enabling greater short-term carbohydrate availability. Consequently, it is unknown

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if enhanced exercise capacity is generalizable to an overnight recovery, relevant to multiday events (e.g., Grand Tours). Therefore, the aim of the present experiments was to determine whether postexercise glucose–fructose coingestion improves exercise capacity following 4-hr (short experiment) and 15-hr (overnight experiment) recoveries from exhaustive exercise in trained cyclists. We hypothesized that, compared with glucose alone, isocaloric glucose–fructose coingestion would augment cycling capacity in short and overnight experiments.

Methods

Study Design

The present research comprised two randomized, crossover experiments conducted at the University of Bath, investigating the effect of postexercise glucose–fructose coingestion versus glucose alone on subsequent cycling capacity. Experiment 1 (double blind) employed a 4-hr recovery (short experiment), whereas, Experiment 2 (single blind) used a 15-hr overnight recovery (overnight experiment). Experimental procedures were approved by the University of Bath Research Ethics Approval Committee for Health (short experiment: MSES 17/18-007; overnight experiment: MSES 17/18-006), conducted according to the Declaration of Helsinki, and participants provided informed, written consent prior to participation.

Participants

Eight trained male cyclists participated in the short experiment (Table 1). Three female and five male trained cyclists participated in the overnight experiment. Volunteers were recruited from local cycling and triathlon clubs. Inclusion criteria included: weekly aerobic training >3 hr and peak oxygen consumption ($\dot{V}O_2$peak) > 45 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}.

Preliminary Testing

The $\dot{V}O_2$peak and peak power (W_{max}) were determined on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). Power output began at 100 W (50 W for females), increasing by 1 W every 3 s until exhaustion. Expired gas was measured continuously using a breath-by-breath analyzer (ParvoMedics TrueOne® 2400, Salt Lake City, UT). $\dot{V}O_2$peak was defined as the highest 30-s $\dot{V}O_2$ rolling average.

Experimental Trials

The short experiment used a full protocol familiarization between preliminary testing and experimental trials. The overnight experiment used an exercise capacity test familiarization following the $\dot{V}O_2$peak test after a 30-min rest.

Table 1   Participant Characteristics

	Short experiment	Overnight experiment
Age (years)	24 ± 7	24 ± 5
Mass (kg)	73.2 ± 7.6	69.1 ± 9.1
Height (m)	1.81 ± 0.08	1.78 ± 0.09
$\dot{V}O_2$peak (ml$\cdot$kg^{-1}$\cdot$min^{-1})	66.9 ± 6.1	62.9 ± 9.8
W_{max} (W)	403 ± 34	378 ± 74

Note. Data are presented as mean ± *SD*. W_{max} = peak power.

All visits were separated by ≥4 days. For females, visits were standardized to the same menstrual cycle phase (by self-report), controlling for the influence of hormonal variance on performance (Julian et al., 2017). Participants refrained from high-intensity exercise for 48 hr, and moderate-intensity exercise, caffeine and alcohol for 24 hr, before each trial.

The short experiment began with a glycogen depletion protocol (GDP). Participants alternated between 2-min efforts at 90% W_{max} and 50% W_{max}. When 90% efforts could no longer be sustained, the high-intensity effort was reduced to 80% and the alternating process continued. When 80% efforts could not be maintained, the alternating process was repeated until exhaustion was reached at 70% W_{max}. The overnight experiment began between 14:30 and 16:30 with a similar GDP; however, when 70% W_{max} could not be maintained, participants continued cycling to complete exhaustion at 50% W_{max}. Both protocols are efficacious at inducing liver and muscle glycogen depletion (Decombaz et al., 2011; Fuchs et al., 2016). Participants were fan cooled and provided water ad libitum.

The GDP was followed by 4 hr ingesting of 1.5 or 1.2 g·kg^{-1}·hr^{-1} carbohydrate in the short experiment and the overnight experiment, respectively. In the short experiment, after 4 hr of recovery, participants cycled to exhaustion at 70% W_{max}. In the overnight experiment, participants cycled to exhaustion at 65% W_{max} the following morning, 15 hr after GDP cessation. Exercise capacity time was recorded upon first attainment (i.e., no three strike rule) of volitional exhaustion. Verbal encouragement was not provided.

During the exercise capacity test, 1-min expired gas samples were collected every 15 min, and at exhaustion. At these time points, capillary fingertip measurements of blood glucose (Free-Style Optium; Abbott Diabetes Care, Witney, UK) and lactate (Lactate Pro; Arkray, Kyoto, Japan) concentrations were taken using portable analyzers (typical error ± 0.2 mmol/L). In the short experiment, this was complemented with a 10 cm, 16-item visual analog scale GDP of upper, lower, and central gastrointestinal symptoms. In the short experiment, all measures were also taken hourly during the 4-hr recovery.

Carbohydrate Test Drinks and Dietary Intake

In the short experiment, a self-selected breakfast was replicated prior to each trial. In the overnight experiment, participants replicated a low-carbohydrate diet (1.3 ± 0.8 g/kg) prior to the GDP.

In the short experiment, participants ingested 1.5 g·kg^{-1}·hr^{-1} of either maltodextrin or sucrose (glucose–fructose disaccharide) in 20% solutions every 30 min during the 4-hr recovery. In the overnight experiment, participants ingested 1.2 g·kg^{-1}·hr^{-1} of either maltodextrin or maltodextrin–fructose (1:1) in hourly 20% solutions for 4-hr post-GDP. Hydrolysis of maltodextrin and sucrose is rapid; hence, these carbohydrates are not digested or absorbed at different rates to their constituent monosaccharides (Gray & Ingelfinger, 1966; Hawley et al., 1991). Therefore, hereafter treatments are referred to as glucose only and glucose + fructose.

In the overnight experiment, participants consumed a standardized meal during the first hour after the GDP (energy: 892 kJ, fat: 11.7 g, carbohydrate: 19.4 g, and protein: 5.8 g) and the following morning at least 1 hr before exercise (energy: 1,784 kJ, fat: 32.8 g, carbohydrate: 7.8 g, and protein: 25.0 g). Thus, participants ingested 359 ± 44 g of carbohydrate between the GDP and the exercise capacity test. Ingesting a low-carbohydrate meal

before exercise removed the influence of exogenous carbohydrate on exercise capacity.

Substrate Oxidation

Expired gas was collected using the Douglas bag technique (Hans Rudolph, Shawnee, KS; Servomex MiniMP 5200; Servomex, Crowborough, UK). During sampling, concurrent measures of inspired air were made to correct for changes in ambient O_2 and CO_2 (Betts & Thompson, 2012). Before each trial, the Servomex was calibrated to two gases of known concentrations, zero gas (100% nitrogen) and a mixed gas (16.12% O_2 and 5.06% CO_2). Rates of substrate oxidation were calculated using the moderate- to high-intensity indirect calorimetry equations of Jeukendrup and Wallis (2005).

Statistical Analysis

A priori sample size was calculated using the running capacity data ($d = 1.84$) from Maunder et al. (2018). $N = 5$ would provide 95% statistical power with $\alpha = .05$. Therefore, eight participants were recruited in each experiment to ensure ample data sets.

In the short and overnight experiments exit interviews indicated one and three participants, respectively, were not successfully blinded to treatment. In each experiment, one participant was metabolically unstable during the exercise capacity test (blood lactate concentration >10 mmol/L) and excluded from metabolite and substrate oxidation data, as indirect calorimetry is not accurate above lactate threshold (Jeukendrup & Wallis, 2005). Experimental data were processed and analyzed using Microsoft Excel (Microsoft, Redmond, WA) and SPSS (version 22; IBM, Armonk, NY). Differences in gastrointestinal discomfort, blood glucose, lactate and rates of carbohydrate, and fat oxidation were assessed between treatments and across time using a two-way repeated-measures analysis of variance. Following a significant interaction, Bonferroni post hoc tests were used to locate

differences. For all comparisons, α was set at .05. The magnitude of differences between variables was assessed using Cohen d effect sizes (d). Data are presented as mean $\pm$ SD alongside 95% confidence intervals.

Results

Short Experiment

No differences were detected in GDP duration between glucose only and glucose + fructose (61.8 ± 13.5 vs. 64.1 ± 10.0 min; $p = .44$). No trial order effects were observed for exercise capacity ($p = .83$). Exercise capacity was greater in glucose + fructose compared with glucose only (28.0 ± 8.4 vs. 22.8 ± 7.3 min; $p = .039$; Figure 1a). This difference was robust to the inclusion ($\Delta 5.1$ min; $d = 0.65$) or exclusion ($\Delta 4.4$ min; $d = 0.54$) of the participant, who correctly identified the trial order.

Blood metabolites. During recovery, blood glucose concentrations increased to a peak at ~60 min before decreasing to baseline concentrations (time effect, $p < .01$), with no differences between trials during recovery or during the exercise capacity test (trial effect, $p = .37$; Time × Trial interaction effect, $p = .54$; Figure 2a).

Blood lactate concentrations were higher in glucose + fructose, compared with glucose only (trial effect, $p = .01$), which was most apparent in the early phase of recovery (Time × Trial interaction effect, $p < .01$; Figure 2b). No differences in blood lactate concentrations were detected during the exercise capacity test (Figure 2b).

Substrate metabolism. Rates of carbohydrate oxidation during the entire recovery period were higher in glucose + fructose versus glucose only (trial effect, $p = .02$; Time × Trial interaction effect, $p = .99$; Figure 2c). However, no differences in carbohydrate oxidation between trials were apparent during the exercise capacity
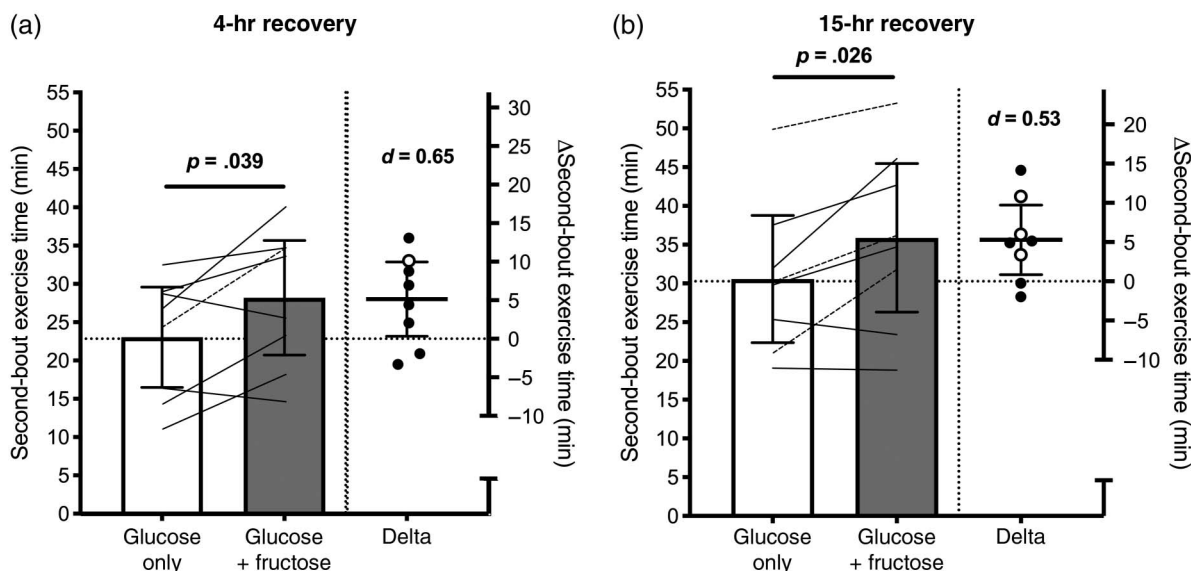

Figure 1 — Exercise capacity following 4-hr short experiment (a) and 15-hr overnight experiment (b) recovery from exhaustive exercise, ingesting glucose alone (glucose only) or isocaloric glucose plus fructose coingestion (glucose + fructose), in trained cyclists. Bars and horizontal lines represent means ± 95% confidence interval ($n = 8$ in both experiments). Lines and circles show individual responses, with those who were unsuccessfully blinded to treatment displayed as dashed lines or open circles, respectively.
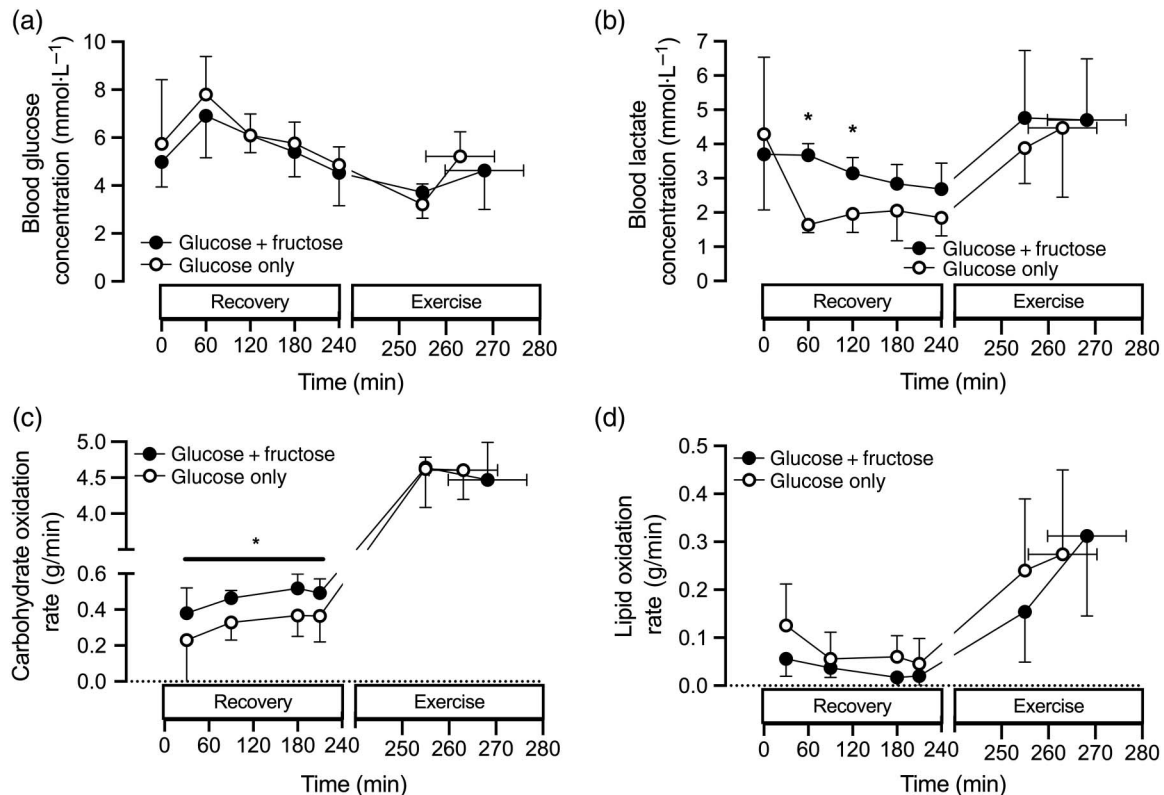
Figure 2 — Blood glucose concentrations (a), blood lactate concentrations (b), carbohydrate oxidation (c), and fat oxidation (d) responses during 4-hr recovery and a subsequent exercise capacity test with ingestion of glucose alone (glucose only) or isocaloric glucose plus fructose coingestion (glucose + fructose) in trained cyclists ($n = 7$). Data are presented as mean ± 95% confidence interval. *A significant trial effect ($p < .05$).

test (Figure 2c), nor were any differences detected in rates of lipid oxidation during recovery or during the exercise capacity test (trial effect, $p = .20$; Figure 2d). Although a Time × Trial interaction effect was detected for lipid oxidation rates, no post hoc comparison remained significant following correction for multiple comparisons (all $ps > .05$).

Gastrointestinal symptoms. Ratings of upper (Figure 3a), lower (Figure 3b), and central/other gastrointestinal symptoms (Figure 3c) all remained low and did not differ between trials (time effect, $p > .05$ for all; trial effect, $p > .05$ for all; Trial × Time interaction effect, $p > .05$ for all).

Overnight Experiment

There was no difference in GDP duration between glucose only and glucose + fructose (96.0 ± 24.2 vs. 92.2 ± 26.8 min; $p = .44$). No trial order effects were detected for exercise capacity ($p = .48$). Exercise capacity was greater in glucose + fructose compared with glucose only (35.9 ± 10.7 vs. 30.6 ± 9.2 min; $p = .026$; Figure 1b). This difference was similar in participants successfully blinded to treatment ($n = 5$; Δ 4.4 min; $d = 0.51$) and those who were not ($n = 3$; Δ 6.7 min; $d = 0.63$).

Blood metabolites. Blood glucose concentrations rose across the exercise capacity test (time effect, $p = .004$), with no differences between trials (trial effect, $p = .11$; Time × Trial interaction effect, $p = .74$; Figure 4a). This response was mirrored in blood lactate concentrations (time effect, $p = .004$), with no differences between trials (trial effect, $p = .12$; Time × Trial interaction effect, $p = .84$; Figure 4b).

Substrate oxidation. Rates of carbohydrate (Figure 4c) and fat oxidation (Figure 4d) were stable across time (time effect, $p > .05$), with no differences between trials (trial effect, $p > .05$; Time × Trial interaction effect, $p > .05$).

Discussion

The present experiments demonstrate that postexercise glucose–fructose coingestion, compared with isocaloric glucose alone, augments cycling capacity following short (4 hr) and overnight (15 hr) recovery periods in trained cyclists. These data provide functional relevance to previous work, showing that enhancement of postexercise liver glycogen repletion and short-term recovery of running capacity, translate into improved cycling capacity over 4 hr and overnight recovery durations.

Previous research exploring postexercise glucose–fructose coingestion versus glucose alone has investigated relatively short recovery periods <5 hr (Casey et al., 2000; Decombaz et al., 2011; Fuchs et al., 2016; Maunder et al., 2018). Relevant to multiday endurance athletes, we demonstrate that postexercise glucose–fructose coingestion augments cycling capacity after an overnight (15 hr) recovery. Exercise capacity tests have been criticized for poor reliability and ecological validity (Currell & Jeukendrup, 2008). However, in races, the intensity is often set by the fastest athlete with others maintaining this pace until it becomes unsustainable; hence, exercise capacity tests are relevant to real-world endurance racing (Alghannam et al., 2014).

The improvement in exercise capacity in the short experiment (27.2 ± 27.5%; $d = 0.65$) and the overnight experiment
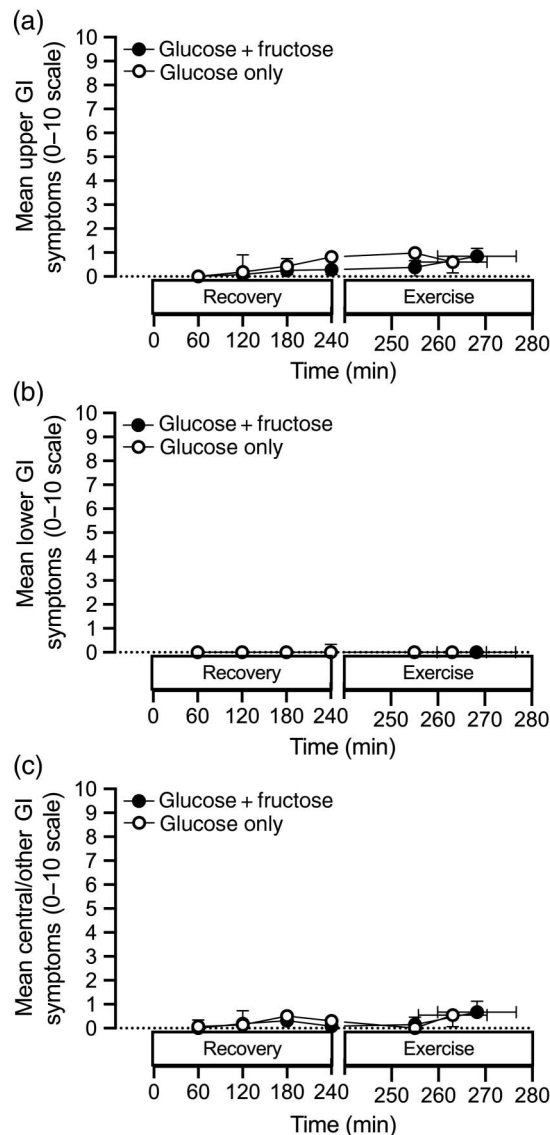
Figure 3 — Upper (a), lower (b), and central/other (c) gastrointestinal symptoms during 4-hr recovery and a subsequent exercise capacity test with ingestion of glucose alone (glucose only) or isocaloric glucose plus fructose coingestion (glucose + fructose) in trained cyclists (*n* = 7). Data are presented as mean ± 95% confidence interval. GI = gastrointestinal.

(18.0 ± 19.4%; *d* = 0.53) was smaller than that observed by Maunder et al. (2018; 32.4 ± 19.9%; *d* = 1.84). The difference in the magnitude of improvement could be explained by differences in exercise modality or intensity. Debilitating gastrointestinal symptoms are more common in runners than cyclists (Peters et al., 2000). Indeed, in the short experiment, we observed no differences in gastrointestinal symptoms between treatments, whereas Maunder et al. (2018) report trends toward greater gastrointestinal discomfort in the glucose treatment. Even mild gastrointestinal distress is associated with impaired endurance performance (O'Brien & Rowlands, 2011; Rowlands et al., 2012). Thus, differences in gastrointestinal comfort could explain the smaller difference in exercise capacity compared with Maunder et al. (2018). Furthermore, the present experiments utilized a higher exercise intensity than Maunder et al. (2018; ~80% vs. 70% $\dot{V}O_2$peak), which may have altered the cause of

fatigue. Indeed, we found that carbohydrate oxidation rates were maintained at exhaustion, whereas, Maunder et al. (2018) report declining rates of carbohydrate oxidation. Thus, in Maunders' research carbohydrate availability (as a fuel) being a more direct cause of fatigue could also partly explain the greater difference in exercise capacity.

The greater improvement in exercise capacity with glucose–fructose coingestion in Maunder et al. (2018) compared with the overnight experiment could further be explained by differences in recovery duration (4 vs. 15 hr, respectively). It has been suggested that a higher liver glycogen concentration stimulates glycogenolysis (Roden et al., 2001). Therefore, a potential increase in liver glycogen content with glucose–fructose coingestion may have diminished overnight. Furthermore, contributing to the smaller difference in exercise capacity in the overnight experiment could be removal of the temporary advantage provided by residual carbohydrate in the gut at exercise initiation. Indeed, intestinal absorption of glucose occurs at ~1 g/min (Jeukendrup, 2004); hence, the carbohydrate ingested in the overnight experiment (359 ± 44 g) would have been absorbed in <6 hr, glucose–fructose being faster still (Jentjens et al., 2004). Thus, unlike research employing a short recovery, exercise capacity in the overnight experiment is more likely to only be influenced by endogenous carbohydrate availability, reducing any metabolic advantage provided through enhanced exogenous carbohydrate availability following glucose–fructose coingestion.

In cycling, hypoglycemia is frequently observed at exhaustion (Casey et al., 2000; Coyle et al., 1986), commonly attributed to a reduction in hepatic glucose output due to liver glycogen depletion (Gonzalez & Betts, 2018). In contrast, in all treatments in the present experiments, blood glucose concentrations rose at exhaustion. This could be explained by the high-intensity exercise (~80% $\dot{V}O_2$peak). During high-intensity exercise, epinephrine is the most potent regulator of hepatic glucose output (Marliss et al., 1992; Sigal et al., 1996). Epinephrine increases hepatic glucose output, while reducing muscle glucose uptake (Howlett et al., 1999). Consequently, increased epinephrine concentrations at exhaustion (Kreisman et al., 2000) could explain the increase in blood glucose concentration. In the present experiments, high rates of carbohydrate oxidation were maintained at exhaustion, which contrasts with prior work in running (Maunder et al., 2018). Thus, in addition to the lower total carbohydrate utilized during the exercise capacity tests in the present experiments versus that of Maunder et al. (2018; <130 vs. >157 g), suggests that low carbohydrate availability may have been less important as a fatigue mechanism in the present experiments. Nevertheless, it is plausible that even a relatively small decrease in carbohydrate oxidation rates could have contributed to fatigue, due to the high-intensity exercise. Furthermore, it is possible that enhanced liver glycogen content after glucose–fructose coingestion facilitated the greater exercise capacity via mechanisms other than providing fuel. Indeed, greater carbohydrate availability is associated with a reduction in perceived effort for a given exercise intensity (Marquet et al., 2016; Noakes, 2004). However, it was not possible to measure liver glycogen content in the present experiments, and therefore, it cannot be determined directly if there was greater liver glycogen availability after glucose–fructose coingestion. Nevertheless, we observed increases in carbohydrate oxidation and blood lactate concentrations at rest following glucose–fructose coingestion versus glucose alone, which is consistent with the hypothesis of greater liver glycogen repletion and hepatic conversion of fructose to lactate (Fuchs et al., 2016).
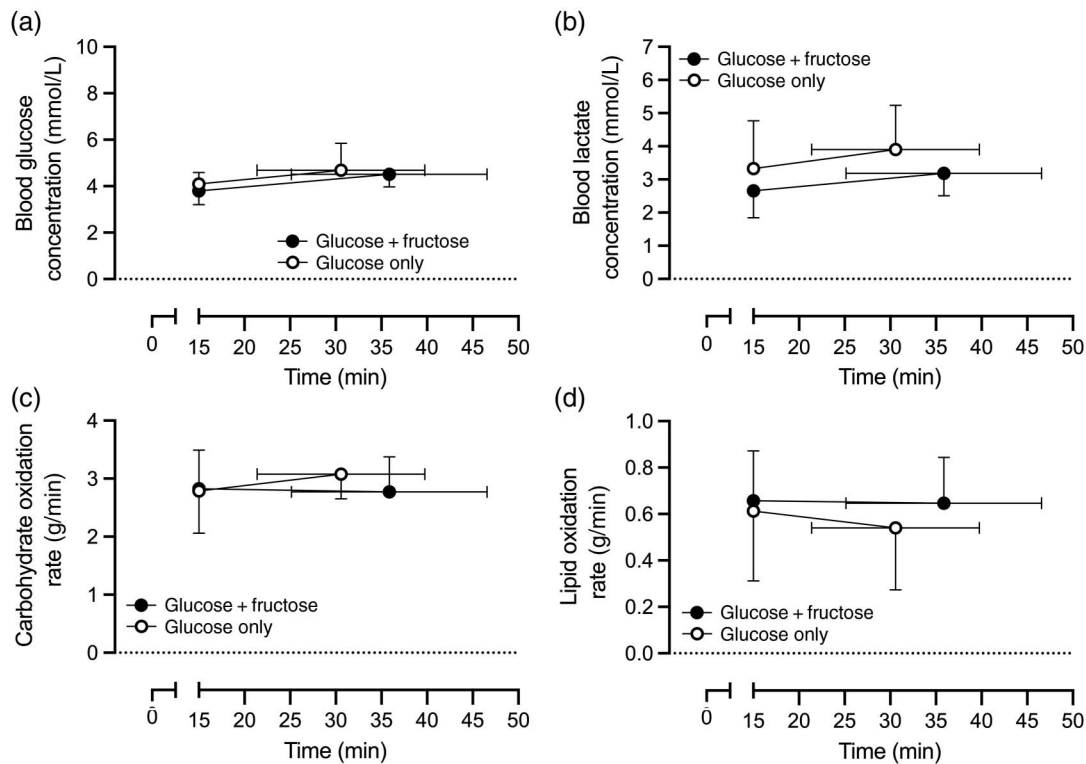
Figure 4 — Blood glucose concentrations (a), blood lactate concentrations (b), carbohydrate oxidation (c), and fat oxidation (d) responses after 15-hr recovery during the exercise capacity test with ingestion of glucose alone (glucose only) or isocaloric glucose plus fructose coingestion (glucose + fructose) in trained cyclists ($n = 7$). Data are presented as mean ± 95% confidence interval.

In conclusion, postexercise glucose–fructose coingestion augments cycling capacity following short (4 hr) and overnight (15 hr) recoveries from exhaustive exercise, compared with isocaloric glucose alone. Therefore, endurance athletes competing in multistage events where recovery time is limited may benefit from consuming fructose alongside glucose in their postexercise nutritional strategies.

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