

# Post-exercise fructose-maltodextrin ingestion enhances subsequent endurance capacity

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1 **ARTICLE TYPE:** Original investigation

2

3 **TITLE:** Post-exercise fructose-maltodextrin ingestion enhances subsequent  
4 endurance capacity

5

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16

17 **ABSTRACT**

18 *Purpose:* Restoring skeletal muscle and hepatic glycogen content during short-term  
19 (<6 h) recovery from prolonged exercise is pertinent for athletes seeking to maximize  
20 performance in repeated exercise bouts. Previous research suggests co-ingestion of  
21 fructose-glucose carbohydrate sources augments hepatic and has equivalent effects on  
22 skeletal muscle glycogen storage during short-term recovery from prolonged exercise  
23 compared to isocaloric glucose ingestion. The aim of the present investigation was to  
24 determine if this has a discernible effect on subsequent exercise capacity. *Methods:*  
25 Eight trained endurance runners and triathletes performed two experimental trials in a  
26 single-blind, randomised, and counterbalanced cross-over design. Trials involved  
27 treadmill running to exhaustion at 70 % $\dot{V}O_{2max}$ , a four-hour recovery with 90 g.h<sup>-1</sup> of  
28 glucose-maltodextrin (GLU+MAL) or fructose-maltodextrin (FRU+MAL) ingestion  
29 (1:1.5 ratio), and a second bout of treadmill running to exhaustion at 70 % $\dot{V}O_{2max}$ .  
30 *Results:* Exercise capacity in bout two was significantly greater with FRU+MAL  
31 (81.4 ± 22.3 vs. 61.4 ± 9.6 min,  $P = 0.02$ ), a *large* magnitude effect (ES = 1.84 ± 1.12,  
32 32.4 ± 19.9 %). Total carbohydrate oxidation rates were not significantly different  
33 during bout one or two between-trials, although total carbohydrate oxidized in bout  
34 two was significantly greater with FRU+MAL (223 ± 66 vs. 157 ± 26 g,  $P = 0.02$ ).  
35 Ingested carbohydrate oxidation rates were greater during bout two with FRU+MAL  
36 ( $P = 0.001$ ). Plasma glucose and non-esterified fatty acid concentrations were not  
37 significantly different between-trials. Plasma lactate concentrations were significantly  
38 greater during recovery prior to bout two with FRU+MAL ( $P = 0.001$ ). Self-reported  
39 nausea and stomach fullness during bout two were marginally in favour of  
40 FRU+MAL. *Conclusion:* Short-term recovery of endurance capacity was significantly  
41 enhanced with FRU+MAL vs. GLU+MAL ingestion during recovery.

42 **KEY WORDS:** Nutrition, carbohydrates, prolonged exercise, substrate oxidation.

43 **INTRODUCTION**

44 Humans have capacity to store finite amounts of carbohydrate energy as glycogen,  
45 predominantly in skeletal muscle (1) and the liver (2). Carbohydrates provide  
46 quantitatively the most important metabolic substrate for fuel metabolism during  
47 exercise of moderate-to-high intensities (3). Most research has focused on the role of  
48 muscle glycogen and it has long been known that exercise of sufficient length and  
49 intensity will eventually deplete these stores to very low concentrations (1),  
50 implicating endogenous carbohydrate availability as a limiting factor during  
51 prolonged, thermoneutral exercise. This hypothesis is supported by recent suggestions  
52 that preferential, accelerated depletion of glycogen stored in the intramyofibrillar  
53 compartment has deleterious effects on muscle function and therefore elicits fatigue  
54 (4).

55

56 Maximizing recovery of muscle glycogen content in the post-exercise period is  
57 pertinent to athletes seeking to optimize performance in repeated bouts of prolonged  
58 exercise with limited recovery time. Indeed, recent evidence suggests the muscle  
59 glycogen-mediated limitation to prolonged exercise capacity holds true for repeated  
60 bouts (5). Current guidelines recommend ingestion of moderate-to-high-glycaemic  
61 index carbohydrates such as glucose-based sources at rates of 1-1.2 g.kg<sup>-1</sup>.h<sup>-1</sup>,  
62 beginning as soon as logistically possible following exercise, when recovery duration  
63 is short (<4 h) (6). This nutritional strategy should facilitate rapid and sufficient  
64 substrate availability to maximize insulin-dependent muscle glycogen synthesis (7),  
65 and take advantage of the insulin-independent contraction-mediated muscle glucose  
66 uptake and glycogen synthesis that occurs in the initial post-exercise period (8).

67

68 The liver functions as a carbohydrate reservoir for release into the circulation and  
69 resultant oxidation by working skeletal muscle, as well as in maintenance of  
70 euglycaemia (9). Whilst glucose is the primary carbohydrate substrate for muscle  
71 glycogen synthesis (10), fructose exerts a superior effect on hepatic glycogen  
72 synthesis (11). Indeed, some studies have now observed superior hepatic and equal  
73 muscle glycogen synthesis with co-ingestion of large amounts of fructose-glucose  
74 carbohydrate sources during acute recovery from prolonged exercise compared to  
75 isocaloric glucose ingestion (12–14). This may have implications for subsequent  
76 exercise capacity through increased whole-body carbohydrate availability.

77

78 To-date, no study has investigated if the apparent metabolic advantage ascertained  
79 through co-ingestion of fructose-glucose carbohydrate sources during short-term  
80 recovery from prolonged exhaustive exercise translates into a discernible effect on  
81 subsequent exercise performance or capacity. The purpose of the present investigation  
82 was to elucidate if such an effect exists, and to determine how any differences  
83 manifest metabolically and perceptually. It was hypothesized that fructose-  
84 maltodextrin co-ingestion during short-term recovery from prolonged exhaustive  
85 exercise would result in superior subsequent exercise capacity compared to isocaloric  
86 glucose-maltodextrin ingestion.

87

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93 **METHODS**

94 *Participants*

95 Eight (six male, two female) healthy, trained endurance runners and triathletes  
96 participated in the present investigation (age,  $31 \pm 6$  y; height,  $176 \pm 6$  cm; mass,  $68.4$   
97  $\pm 5.6$  kg;  $\dot{V}O_{2\max}$ ,  $3.76 \pm 0.47$  l.min<sup>-1</sup>). The sample size was chosen based on  
98 pragmatic reasons, balancing logistical, financial and recruitment-related  
99 considerations for such an arduous experimental protocol, as well as reflecting the  
100 number of participants used in previous similar studies (15,16). All participants were  
101 engaging in training for endurance running events, habitually covering a self-reported  
102  $63 \pm 19$  km.week<sup>-1</sup>. Experimental procedures were approved by the University of  
103 Birmingham (United Kingdom) Ethics Committee, and all participants provided  
104 written informed consent.

105

106 *Study design*

107 The present investigation adopted a single-blinded, randomized, and counterbalanced  
108 cross-over design involving four laboratory visits. In the first laboratory visit, a  
109 maximal treadmill test (*Preliminary test*) was performed to determine maximum  
110 oxygen uptake ( $\dot{V}O_{2\max}$ ). The second visit consisted of full familiarization to  
111 procedures performed in the subsequent experimental trials, without venous  
112 cannulation and blood collection. Thereafter, two experimental trials were conducted  
113 4-16 d apart in a random, counterbalanced order ([www.random.org](http://www.random.org)). Participants  
114 were blinded to trial order. Experimental trials consisted of treadmill running to  
115 exhaustion at 70 % $\dot{V}O_{2\max}$ , a four-hour recovery with 90 g.h<sup>-1</sup> maltodextrin and  
116 glucose (MAL+GLU) or maltodextrin and fructose (MAL+FRU) ingestion (1.5:1  
117 ratio), and a second bout of treadmill running to exhaustion at 70 % $\dot{V}O_{2\max}$ .

118

119 *Preliminary test*

120 To determine  $\dot{V}O_{2\max}$ , an incremental step test to volitional exhaustion was performed  
121 on a motorized treadmill (quasar, h/p cosmos, GER) during the first laboratory visit  
122 ( $18 \pm 1^\circ\text{C}$ ,  $48 \pm 14\%$  rH). Height (Model 220, Seca, GER) and body mass (Champ II,  
123 OHAUS, SWI) of each participant was measured prior to the test. Participants then  
124 completed 4-min stages at 7, 9, 11, and 13  $\text{km}\cdot\text{h}^{-1}$  against a 1 % gradient to simulate  
125 the energetic cost of level-gradient outdoor running (17). Heart rate (HR; Polar  
126 Electro, FIN) and perceived exertion (RPE) according to Borg's 6-20 scale (18) were  
127 recorded in the final 60 s of each stage. Oxygen uptake ( $\dot{V}O_2$ ) was measured  
128 continuously using an automated analyzer (JAEGER<sup>®</sup> Vyntus CPX, CareFusion,  
129 GER), and calculated at each velocity as the average value during the final 30 s.  
130 Following completion of the 13  $\text{km}\cdot\text{h}^{-1}$  stage, treadmill velocity was reduced to 11  
131  $\text{km}\cdot\text{h}^{-1}$  and subsequently increased by 0.5  $\text{km}\cdot\text{h}^{-1}$  every 30 s until attainment of  
132 volitional exhaustion. Maximum oxygen uptake ( $\dot{V}O_{2\max}$ ) was accepted as the highest  
133  $\dot{V}O_2$  15-breath rolling average if two of the following three criteria were met:  
134 respiratory exchange ratio  $\geq 1.10$ , HR  $\pm 10 \text{ b}\cdot\text{min}^{-1}$  of age-predicted maximum ( $205.8$   
135  $- 0.685[\text{age (y)}]$ ) (19), and attainment of volitional exhaustion. Simple regression  
136 equations were used to estimate the speed required to elicit 70 %  $\dot{V}O_{2\max}$  for use in the  
137 subsequent trials.

138

139 *Experimental trials*

140 At least five days following the familiarization trial, participants reported to the  
141 laboratory at ~08:00hrs having fasted overnight and refrained from caffeine, alcohol,  
142 and vigorous exercise for 24 h, and having completed a three-day pre-trial diet diary



143 to be repeated in the run-up to the final experimental trial. Participants were fitted  
144 with an antecubital venous cannula (BD Venflon<sup>TM</sup>, Helsingborg, SWE), and a 5-ml  
145 baseline venous blood sample was drawn. A treadmill run to volitional exhaustion at  
146 70 % $\dot{V}O_{2max}$  then commenced (quasar, h/p cosmos, GER), with venous blood samples  
147 obtained after 30 min, 60 min, and at exhaustion from an extension line to minimise  
148 impact on running gait and technique. Expired gas samples were collected every 15  
149 min and at exhaustion and analyzed for  $\dot{V}O_2$  and  $\dot{V}CO_2$  using an automated analyser  
150 (JAEGER<sup>®</sup> Vyntus CPX, CareFusion, GER). Water was consumed *ad libitum*. On  
151 attainment of volitional exhaustion, treadmill speed was reduced to 4.4 km.h<sup>-1</sup> for two  
152 minutes. Treadmill speed was then restored to that eliciting 70 % $\dot{V}O_{2max}$  and the  
153 participant was again asked to run to volitional exhaustion. This process was repeated  
154 so only at the third attainment of volitional exhaustion was the test terminated. This  
155 protocol has lower coefficient of variation for exercise capacity compared to  
156 traditional single-exhaustion protocols (5.4 %, 1.4-9.6 [95 % confidence intervals])  
157 (20).

158

159 Participants then passively rested for four hours, during which sedentary activities  
160 such as reading and use of laptops were permitted. Participants immediately ingested  
161 a 300-ml beverage containing 18 g glucose (GLU+MAL) or fructose (FRU+MAL)  
162 with 27 g maltodextrin, and therefore in a 1:1.5 ratio. Carbohydrates ingested during  
163 recovery were of high <sup>13</sup>C natural abundance (-11.36 and -11.39  $\delta^{13}C_{V-PDB}$  ‰; The  
164 Hut Group, Cheshire, UK; Sports Supplements Ltd., Essex, UK). Identical beverages  
165 were ingested every 30 min throughout recovery ending 30 min prior to the end of the  
166 recovery period, such that 2.4 l of fluid and 90 g.h<sup>-1</sup> of carbohydrate was ingested over  
167 the four-hour period. Venous blood samples and scales for gastrointestinal comfort

168 (GC) (21) were obtained every hour during recovery. The GC scales assessed nausea,  
169 stomach fullness, and abdominal cramping using 1-10 point Likert scales.

170

171 Following the four-hour recovery period, participants commenced a second treadmill  
172 run to volitional exhaustion at 70 %  $\dot{V}O_{2max}$  as before. Venous blood samples, GC  
173 scales, RPE, and a scale for lower-limb muscle soreness (22) were obtained every 15  
174 min and at exhaustion. Expired gas samples were also obtained for 4 min every 15  
175 min and at exhaustion, and analysed for  $\dot{V}O_2$  and  $\dot{V}CO_2$  using an automated analyzer  
176 (JAEGER<sup>®</sup> Vyntus CPX, CareFusion, GER). The exhaustion time-point expired gas  
177 sample was collected during a period of running between the first and final claim of  
178 volitional exhaustion. At these time-points, and also immediately following the first  
179 exercise bout, breath samples were collected into 10-ml evacuated tubes (Exetainer<sup>®</sup>  
180 Breath Vial, Labco Ltd., UK).

181

#### 182 *Blood analyses*

183 Venous blood samples were aliquoted into ~5 ml pre-chilled  
184 ethylenediaminetetraacetic acid tubes, centrifuged for 10 min at 4°C and 3500  
185 revs.min<sup>-1</sup>, and stored at -25°C. Plasma glucose, non-esterified fatty acid (NEFA), and  
186 lactate concentrations were later determined through duplicate colorimetric assays  
187 using a semi-automatic analyser (ILab 650, Instrumentation Laboratory, Bedford,  
188 MA, USA) and commercially available kits (Randox Laboratories Ltd., County  
189 Antrim, UK).

190

#### 191 *Gas analyses*

192  $\dot{V}O_2$  and  $\dot{V}CO_2$  were calculated using an automated analyzer (JAEGER<sup>®</sup> Vyntus  
193 CPX, CareFusion, GER). This allowed for calculation of whole-body rates of fat and  
194 carbohydrate ( $CHO_{tot}$ ) oxidation at each time-point during the first and second bouts  
195 of the experimental trials using the following equations, which assume a negligible  
196 contribution of protein oxidation to metabolism (Eq. 1-2) (23):

197

$$198 \quad \text{Fat oxidation} = (1.695 \times \dot{V}O_2) - (1.701 \times \dot{V}CO_2)$$

199

200 **Eq. 1**

201

$$202 \quad CHO_{tot} \text{ oxidation} = (4.210 \times \dot{V}CO_2) - (2.962 \times \dot{V}O_2)$$

203

204 **Eq. 2**

205

206 Additionally, non-linear modelling software was used (Microsoft Excel 2011,  
207 Redmond, WAS) such that  $CHO_{tot}$  could be compared between-trials at the point of  
208 exhaustion in the second bout of the shorter duration trial (Eq. 3). For example, if  
209 exhaustion occurred for one individual at 55 min in the second bout of the  
210 GLU+MAL trial, and 70 min in the FRU+MAL trial, the curve for  $CHO_{tot}$  vs. time in  
211 the FRU+MAL trial was non-linearly modelled such that  $CHO_{tot}$  could be estimated at  
212 55 min.

213

$$214 \quad \text{Modelled } CHO_{tot} = a + (b/t) + c \ln(t)$$

215

216 **Eq. 3** where  $t$  = time-point (min) and  $a$ ,  $b$ , and  $c$  were solved such that the  
217 modelled equation produced the lowest cumulative deviation from known  
218 values in each individual.

219

220 The isotopic enrichment of breath samples collected into 10-ml evacuated tubes  
221 (Exetainer® Breath Vial, Labco Ltd., UK) at each time-point was determined by gas  
222 chromatography isotope ratio mass spectrometry (IsoAnalytical Ltd., Crewe, UK)  
223 using the following equation (24) (Eq. 4):

224

$$225 \quad \delta^{13}\text{C} = [({}^{13}\text{C}:{}^{12}\text{C} \text{ sample}/{}^{13}\text{C}:{}^{12}\text{C} \text{ standard}) - 1] \times 10^3 \text{ ml}^{-1}$$

226

227 **Eq. 4** where isotopic enrichment was expressed as  $\delta.\text{ml}^{-1}$  and related to an  
228 international standard (PDB).

229

230 Subsequently, the oxidation rate of carbohydrate ingested during recovery ( $\text{CHO}_{\text{ing}}$ ) at  
231 each time-point during the second bout could then be calculated according to the  
232 following equation (Eq. 5):

233

$$234 \quad \text{CHO}_{\text{ing}} = \dot{V}\text{CO}_2 \times [(\delta\text{Exp} - \text{Exp}_{\text{bkg}})/(\delta\text{Ing} - \text{Exp}_{\text{bkg}})] \times (1/0.7467)$$

235

236 **Eq. 5** where  $\delta\text{Exp}$  =  $^{13}\text{C}$  enrichment of expired gas sample,  $\delta\text{Ing}$  =  $^{13}\text{C}$   
237 enrichment of ingested carbohydrate,  $\text{Exp}_{\text{bkg}}$  =  $^{13}\text{C}$  enrichment of expired gas  
238 sampled following the first exercise bout, and  $0.7467 = \dot{V}\text{CO}_2$  of 1 g glucose  
239 oxidation.

240

241 A consideration when attempting to measure specific oxidation of ingested high  
242 natural abundance  $^{13}\text{C}$  carbohydrates in expired breath is temporary retention of the  
243  $^{13}\text{C}$  label in the body's endogenous bicarbonate pool as  $^{13}\text{CO}_2$  during the initial 60  
244 min of moderate-intensity exercise, resulting in underestimation of calculated ingested  
245 carbohydrate oxidation rates (25). As this underestimation is likely to be systematic  
246 between-trials, no arbitrary correction factor was deemed necessary given the cross-  
247 over design of the present investigation. Nonetheless, the ingested carbohydrate  
248 oxidation rates presented here should be considered minimal estimates.

249

#### 250 *Statistical analyses*

251 Data was analyzed using commercially available software (SPSS Statistics, v22, SPSS  
252 Inc., Chicago, IL). Data collected in the first exercise bout, recovery period, and  
253 second exercise bout was considered separately. Sample distribution data is expressed  
254 mean  $\pm$  standard deviation (SD). Statistical significance was inferred when  $P \leq 0.05$ .

255

256 Between- and within-trial time-point specific substrate oxidation rate comparisons  
257 ( $\text{CHO}_{\text{tot}}$ ,  $\text{CHO}_{\text{ing}}$ , fat oxidation), as well as those for plasma glucose, NEFA, and  
258 lactate concentrations, and psychometric scales, were made using two-way repeated  
259 measures analyses of variance. Non-spherical data was corrected using the  
260 Greenhouse-Geisser (epsilon  $< 0.75$ ) or Huynh-Feldt (epsilon  $> 0.75$ ) adjustment.  
261 Where a significant effect was indicated for these variables, Holm-Bonferroni  
262 stepwise correction was made for location of variance post-hoc, and these  $P$  values  
263 are reported.

264

265 Total substrate oxidation, *i.e.* CHO<sub>tot</sub>, CHO<sub>ing</sub>, and fat oxidation in grams, was  
266 estimated for the second exercise bout in each trial through manually calculated area  
267 under the curve (g.min<sup>-1</sup> vs. time). These variables, and exercise capacity, were  
268 compared between-trials using paired t-tests or Wilcoxon-signed rank tests, dependent  
269 on normality. The magnitude of statistically significant effects in these variables was  
270 determined through within-subject Cohen's *d* effect sizes (ES) computed using a  
271 purpose-built spreadsheet (26). ES, presented ± 90 % confidence limit, was  
272 interpreted according to Cohen's criteria: 0.2-0.5, small; 0.5-0.8, moderate; >0.8,  
273 large (27). Where appropriate, percent changes are presented ± confidence limit, and  
274 post-hoc calculation of achieved power was made using the ES, sample size, and *P*  
275 value (G\*Power 3.1, Universität Düsseldorf, DEU).

276

## 277 **RESULTS**

### 278 *Exercise capacity*

279 Exercise intensity was matched between-trials in bout one (69.4 ± 2.5 vs. 69.3 ± 2.4  
280 %  $\dot{V}O_{2max}$  in GLU+MAL and FRU+MAL, respectively, *P* = 0.91) and two (69.6 ± 1.3  
281 vs. 69.3 ± 1.9 %  $\dot{V}O_{2max}$  in GLU+MAL and FRU+MAL, respectively, *P* = 0.64). Bout  
282 one exercise capacity was not significantly different between-trials (131.3 ± 36.1 vs.  
283 134.6 ± 34.6 min in GLU+MAL and FRU+MAL, respectively, *P* = 0.38). The within-  
284 subject SD for bout one exercise capacity was 6.4 ± 3.4 min, with a coefficient of  
285 variation of 5.5 ± 3.2 %. No order effect was observed (*P* = 0.41).

286

287 Second bout exercise capacity was significantly greater in the FRU+MAL trial (81.4  
288 ± 22.3 vs. 61.4 ± 9.6 min, *P* = 0.02, Figure 1), a *large* magnitude effect (ES = 1.84 ±  
289 1.12, 32.4 ± 19.9 %). This effect was observed in seven of the eight participants. Post-

290 hoc analysis revealed the study had 95% statistical power to reveal an enhanced  
291 exercise capacity based on the sample size used and effect size observed. No order  
292 effect was observed ( $P = 0.69$ ).

293

294 \*\*\*INSERT FIGURE 1 ABOUT HERE\*\*\*

295

### 296 *Substrate metabolism*

297 CHO<sub>tot</sub> oxidation rates were not significantly different between-trials during bout one  
298 ( $P = 0.96$ ). CHO<sub>tot</sub> oxidation rates at 15 min, 30 min and exhaustion in bout two were  
299 not significantly different between-trials ( $P = 0.171$ , Table 1), but were significantly  
300 reduced at exhaustion vs. 15 and 30 min ( $P < 0.005$ ). The modelled CHO<sub>tot</sub> oxidation  
301 rate in bout two of the trial with superior exercise capacity at the point of exhaustion  
302 in the trial with inferior exercise capacity was significantly greater than the CHO<sub>tot</sub>  
303 oxidation rate at the point of exhaustion in the trial with inferior exercise capacity  
304 ( $2.74 \pm 0.52$  vs.  $1.88 \pm 0.52$  g.min<sup>-1</sup>,  $P = 0.002$ ). This effect was consistent in all eight  
305 participants and was of *large* magnitude (ES =  $1.46 \pm 0.89$ ,  $58 \pm 28$  %). In the seven  
306 participants who had greater second bout exercise capacity with FRU+MAL, the  
307 modelled CHO<sub>tot</sub> oxidation rate in the FRU+MAL trial at the point of exhaustion in  
308 the GLU+MAL trial was significantly greater than the CHO<sub>tot</sub> oxidation rate at  
309 exhaustion in the GLU+MAL trial ( $2.71 \pm 0.55$  vs.  $1.84 \pm 0.55$  g.min<sup>-1</sup>,  $P = 0.03$ ).  
310 This effect was consistent in all seven participants and was of *large* magnitude (ES =  
311  $1.36 \pm 0.97$ ,  $60 \pm 34$  %). The absolute amount of CHO<sub>tot</sub> oxidised during bout two  
312 was significantly greater with FRU+MAL, a *large* magnitude effect (Table 1).

313

314 CHO<sub>ing</sub> oxidation rates were significantly greater after 15 and 30 min in the  
315 FRU+MAL vs. GLU+MAL trial ( $P < 0.002$ , Table 1). CHO<sub>ing</sub> oxidation rate at  
316 exhaustion was significantly decreased vs. 15 and 30 min in both trials ( $P < 0.005$ ). In  
317 the GLU+MAL trial, CHO<sub>ing</sub> was also significantly lower at 30 vs. 15 min ( $P =$   
318 0.002). The absolute amount of CHO<sub>ing</sub> oxidised during bout two was significantly  
319 greater with FRU+MAL, a *large* magnitude effect (Table 1).

320

321 \*\*\*INSERT TABLE 1 ABOUT HERE\*\*\*

322

### 323 *Plasma variables*

324 Due to clotting, blood data sets in bout two are available for six participants (bout one  
325 and recovery, N = 8). Plasma variables were not significantly different between-trials,  
326 with the exception of plasma glucose concentration at the point of exhaustion in bout  
327 two ( $6.3 \pm 1.0$  vs.  $5.3 \pm 0.7$  mmol.l<sup>-1</sup>, in GLU+MAL and FRU+MAL, respectively,  $P$   
328 = 0.003), and plasma lactate concentrations after 60, 120, and 180 min of recovery  
329 (FRU+MAL > GLU+MAL,  $P < 0.02$ , see Figure, SDC1, plasma metabolite responses  
330 to the experimental protocols).

331

### 332 *Perceptual responses*

333 Bout two RPE was significantly lower with FRU+MAL vs. GLU+MAL after 30 min  
334 ( $13 \pm 1$  vs.  $14 \pm 2$  AU,  $P = 0.02$ ). Muscle soreness in bout two was not significantly  
335 different between-trials ( $P = 0.31$ ), but was significantly elevated in bout two at  
336 exhaustion vs. all other time-points in both trials ( $P < 0.05$ ).

337



338 Nausea, stomach fullness, and abdominal cramping were not significantly different  
339 between-trials during recovery ( $P > 0.27$ ). Between-trial differences in nausea ( $P =$   
340 0.04) and stomach fullness ( $P = 0.03$ ) during bout two were not significant at any  
341 time-point after post-hoc analysis ( $P > 0.10$ ). Stomach fullness was significantly  
342 lower at each time-point vs. all previous time-points during bout two with FRU+MAL  
343 ( $P = 0.05$ , Figure 2).

344

345 \*\*\*INSERT FIGURE 2 ABOUT HERE\*\*\*

346

347

348 **DISCUSSION**

349 The aim of the present investigation was to determine if the previously observed  
350 metabolic advantages ascertained through co-ingestion of fructose-glucose  
351 carbohydrate sources during short-term recovery from prolonged exercise translate  
352 into a discernible effect on subsequent exercise capacity. The main finding was that  
353 short-term recovery of endurance exercise capacity was significantly augmented with  
354 co-ingestion of fructose and maltodextrin during recovery compared to isocaloric  
355 glucose and maltodextrin ingestion by  $32.4 \pm 19.9$  %. Using a conversion described  
356 previously (28), it can be estimated that this equates to an  $\sim 5.9$  % improvement in  
357 time-trial performance. This novel finding provides functional relevance to previous  
358 metabolic investigations demonstrating enhanced hepatic and equivalent effects on  
359 skeletal muscle glycogen storage during acute recovery from prolonged exhaustive  
360 exercise with such nutritional regimens (12–14,29–32).

361

362 In the present investigation, improved recovery of exercise capacity was observed  
363 with FRU+MAL in seven of eight participants. This contradicts a previous study  
364 adopting a similar experimental design (12). Casey *et al.* (12) had participants ingest a  
365 single  $1 \text{ g}\cdot\text{kg}^{-1}$  glucose or sucrose bolus at the start of a four-hour recovery period.  
366 This amounted to  $\sim 19 \text{ g}\cdot\text{h}^{-1}$  of carbohydrate on average, compared to  $90 \text{ g}\cdot\text{h}^{-1}$   
367 throughout recovery in the present investigation. The dosing provided by Casey *et al.*  
368 (12) was substantially lower than those demonstrating enhanced hepatic, with similar  
369 muscle, glycogen synthesis during short-term recovery from prolonged exercise with  
370 fructose-glucose carbohydrate sources ( $\sim 69\text{--}116 \text{ g}\cdot\text{h}^{-1}$ ) (13,14,31). Indeed, Casey and  
371 colleagues observed no significant differences in muscle or hepatic glycogen  
372 synthesis between the sucrose and glucose trials. Therefore, whilst no measure of

373 glycogen synthesis was made presently, it is possible that the failure to observe an  
374 effect on subsequent exercise capacity by Casey *et al.* (12) occurred due to the lower  
375 carbohydrate doses and similar metabolic recovery provided between-conditions. The  
376 use of exercise capacity protocols has been questioned regarding issues of reliability  
377 (33), but a strong coefficient of variation for bout one exercise capacity was observed  
378 presently, replicating recent work (20). The larger carbohydrate doses used in the  
379 present investigation may have facilitated a metabolic advantage with FRU+MAL,  
380 and this may be required to ascertain the observed *large* beneficial effect on  
381 subsequent exercise capacity.

382

383 In the present investigation, there is some indication second bout exercise capacity  
384 was limited by carbohydrate availability for oxidation, presumably in skeletal muscle,  
385 as the CHO<sub>tot</sub> oxidation rate significantly declined at exhaustion in both trials. This  
386 reduction in carbohydrate oxidation rate is in line with some (5,15,34,35), but not all  
387 (36,16,37), previous investigations adopting similar repeated exercise capacity  
388 protocols. While speculative, carbohydrate oxidation rates during the second bout in  
389 the present study may have become unsustainable to fuel the exercise intensity. It is  
390 possible the enhanced second bout exercise capacity observed with FRU+MAL is  
391 attributable to an ability to maintain whole-body carbohydrate oxidation rates for  
392 longer prior to the reduction seemingly associated with fatigue. Accordingly, the  
393 absolute CHO<sub>tot</sub> oxidised in bout two was significantly greater with FRU+MAL  
394 (Table 1), although this could be an artefact of the enhanced bout two exercise  
395 duration. The existence of this effect is supported by the modelled relationship  
396 between CHO<sub>tot</sub> oxidation rate and time with FRU+MAL. That is, CHO<sub>tot</sub> in  
397 FRU+MAL was estimated to be significantly greater at the point of exhaustion in

398 GLU+MAL in the seven participants who performed better in the FRU+MAL trial.  
399 This suggests the augmented bout two exercise capacity seen with FRU+MAL might  
400 be attributed to enhanced ability to sustain whole-body carbohydrate oxidation at the  
401 rate required to support the exercise intensity. In further support of a metabolic  
402 explanation for the observed effect is that the one participant who demonstrated  
403 reduced bout two exercise capacity with FRU+MAL exhibited poorer maintenance of  
404 carbohydrate oxidation rate in that trial.

405

406 As compared to GLU+MAL, greater CHO<sub>ing</sub> oxidation rates were observed at 15 and  
407 30 min with FRU+MAL, alongside similar declines at exhaustion. This supports to  
408 the suggestion that enhanced carbohydrate availability facilitated the greater second  
409 bout exercise capacity with FRU+MAL. Greater CHO<sub>ing</sub> oxidation rates may reflect  
410 augmented hepatic, and similar muscle, glycogen synthesis with FRU+MAL, an  
411 effect observed previously with similar dosing regimens (13,14,31). Greater whole-  
412 body glycogen synthesis, derived from the ingested carbohydrate, may therefore  
413 facilitate greater carbohydrate availability for oxidation by working skeletal muscle  
414 during bout two. It must also be acknowledged that the source of the additional  
415 oxidised ingested carbohydrates cannot be discerned in the present investigation. That  
416 is, it is not possible to determine what proportion of the oxidised ingested  
417 carbohydrate was first stored in muscle, in liver, or oxidised directly after absorption.  
418 There is a wealth of literature describing the more rapid intestinal absorption and  
419 oxidation of glucose-fructose carbohydrate sources ingested during exercise compared  
420 to glucose alone (10), which could plausibly contribute to the observed effect on  
421 CHO<sub>ing</sub> oxidation rates if participants began bout two with any unabsorbed  
422 carbohydrate residing in the gut. Furthermore, greater plasma lactate concentrations

423 were observed during the recovery period with FRU+MAL, a finding in line with  
424 previous investigations (14,30–32). This likely reflects augmented hepatic lactate  
425 production derived from ingested fructose (10). Lactate is a glycogenic precursor (10)  
426 and carbohydrate substrate that can be oxidised directly (38). The observed greater  
427 plasma lactate concentrations during recovery with FRU+MAL may therefore be  
428 derived from ingested fructose-derived hepatic lactate production, and provide  
429 substrate for whole-body glycogen synthesis or direct oxidation in the early stages of  
430 bout two, thereby supporting the greater CHO<sub>ing</sub> oxidation rates with FRU+MAL.  
431 Further mechanistic work is required in order to establish the metabolic route by  
432 which carbohydrate ingested during recovery is oxidized during bout two.

433

434 Similar to previous investigations adopting similar repeated exercise capacity  
435 protocols (5,15,34–36,16,37), it does not appear that hypoglycaemia limited exercise  
436 in the present investigation, as evidenced by the absence of low plasma glucose  
437 concentrations at exhaustion in both trials (Suppl. Figure 1). However, there is now  
438 acknowledgement that differences in gut comfort can impact prolonged exercise  
439 performance (39). In the present investigation, no clear significant differences  
440 between-trials were observed for gut comfort during recovery, which is in  
441 contradiction to previous investigations reporting greater self-reported symptoms of  
442 gastrointestinal distress with glucose ingestion alone, although the severity of these  
443 symptoms was unclear, and a second bout of exercise was not performed (14,31). Any  
444 differences during bout two were of small numeric magnitude (Figure 2). The mean  
445 value for nausea in the GLU+MAL trial at exhaustion ( $3.1 \pm 2.2$  AU) reflects  
446 symptoms between “slight” and “moderate”. Interestingly, during bout two, stomach  
447 fullness progressively, and significantly, declined with FRU+MAL, but this was not

448 observed with GLU+MAL. Greater stomach fullness with MAL+GLU might be  
449 explained by accumulation of carbohydrate in the gut, given the more rapid intestinal  
450 absorption of fructose-glucose sources (40). Again, stomach fullness at exhaustion  
451 with GLU+MAL was less than “moderate” ( $3.8 \pm 2.4$  AU). However, whilst these  
452 values appear of small magnitude, it is not possible to discern the threshold nausea  
453 and stomach fullness values likely to impact exercise cessation, and so between-trial  
454 differences in gastrointestinal comfort cannot be dismissed as an explanation for the  
455 observed effect on exercise capacity.

456

457 In conclusion, the present investigation has for the first time demonstrated  
458 maltodextrin-fructose co-ingestion enhances short-term recovery of endurance  
459 exercise capacity. Secondly, accompanying data suggests some of the effect may be  
460 explained by increased carbohydrate availability, although a contribution from  
461 improved gastrointestinal comfort cannot be dismissed. If verified in future work,  
462 these results have implications for endurance athletes aiming to optimize performance  
463 in repeated bouts of prolonged exhaustive exercise with limited recovery duration.

464

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469

#### 470 **CONFLICT OF INTEREST**

471 The authors declare no conflicts of interest. The results of the present study are  
472 presented clearly, honestly, and without fabrication, falsification, or inappropriate  
473 data manipulation, and do not constitute endorsement by the ACSM.

474

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- 590
- 591

592 **FIGURE HEADINGS**

593

594 **Fig. 1.** Mean responses for second bout exercise capacity (min) in the GLU+MAL  
595 and FRU+MAL trials (N = 8). \* denotes  $P = 0.02$  between-trials.

596

597 **Fig. 2.** Self-reported (a) nausea, (b) stomach fullness, and (c) abdominal cramping (1-  
598 10, AU) during recovery and bout two of the GLU+MAL and FRU+MAL trials (N =  
599 8).

600

601 **Suppl. Fig. 1.** Plasma concentrations of (a) glucose, (b) NEFA, and (c) lactate  
602 ( $\text{mmol.l}^{-1}$ ) throughout bout one (N = 8), recovery (N = 8), and bout two (N = 6) of the  
603 GLU+MAL and FRU+MAL trials. “PreEx1” refers to samples obtained immediately  
604 prior to bout one, “Ex1-30” refers to samples obtained after 30 min of bout one, “Ex1-  
605 ex” refers to samples obtained at exhaustion of bout one, “Rec-60” refers to samples  
606 obtained after 60 min of recovery, *etc.*, \* denotes  $P < 0.05$  between-trials, # denotes  $P$   
607  $< 0.01$  between-trials.

608