Sucrose ingestion after exhaustive exercise accelerates liver, but not muscle glycogen repletion compared with glucose ingestion in trained athletes

Cas J. Fuchs,1,2 Javier T. Gonzalez,3 Milou Beelen,1 Naomi M. Cermak,1 Fiona E. Smith,4,5 Pete E. Thelwall,4,5 Roy Taylor,4,5 Michael I. Trenell,4 Emma J. Stevenson,2,4 and Luc J. C. van Loon1

1NUTRIM School of Nutrition and Translational Research in Metabolism, Maastricht University Medical Centre, Maastricht, The Netherlands; 2Department of Sport, Exercise, and Rehabilitation, Northumbria University, Newcastle upon Tyne, United Kingdom; 3Department for Health, University of Bath, Bath, United Kingdom; 4Institute of Cellular Medicine, Newcastle University, Newcastle upon Tyne, United Kingdom; and 5Newcastle Magnetic Resonance Centre, Newcastle University, Newcastle upon Tyne, United Kingdom

Submitted 3 December 2015; accepted in final form 18 March 2016

Fuchs CJ, Gonzalez JT, Beelen M, Cermak NM, Smith FE, Thelwall PE, Taylor R, Trenell MI, Stevenson EJ, van Loon LJ. Sucrose ingestion after exhaustive exercise accelerates liver, but not muscle glycogen repletion compared with glucose ingestion in trained athletes. J Appl Physiol 120: 1328–1334, 2016. First published March 24, 2016; doi:10.1152/japplphysiol.01023.2015. The purpose of this study was to assess the effects of sucrose vs. glucose ingestion on postexercise liver and muscle glycogen repletion. Fifteen well-trained male cyclists completed two test days. Each test day started with glycogen-depleting exercise, followed by 5 h of recovery, during which subjects ingested 1.5 g·kg−1·h−1 sucrose or glucose. Blood was sampled frequently and 13C magnetic resonance spectroscopy and imaging were employed 0, 120, and 300 min postexercise to determine liver and muscle glycogen concentrations and liver volume. Results were as follows: Postexercise muscle glycogen concentrations increased significantly from 85 ± 27 (SD) mmol/l to 140 ± 23 vs. 136 ± 26 mmol/l following sucrose and glucose ingestion, respectively (no differences between treatments; P = 0.673). Postexercise liver glycogen concentrations increased significantly from 183 ± 47 vs. 167 ± 65 mmol/l to 280 ± 72 vs. 234 ± 81 mmol/l following sucrose and glucose ingestion, respectively (time × treatment, P = 0.051). Liver volume increased significantly over the 300-min period after sucrose ingestion only (time × treatment, P = 0.001). As a result, liver glycogen content increased during postexercise recovery to a greater extent in the sucrose treatment (from 53.6 ± 16.2 to 86.8 ± 29.0 g) compared with the glucose treatment (49.3 ± 25.3 to 65.7 ± 27.1 g; time × treatment, P < 0.001), equating to a 3.4 g/l (95% confidence interval: 1.6–5.1 g/l) greater repletion rate with sucrose vs. glucose ingestion. In conclusion, sucrose ingestion (1.5 g·kg−1·h−1) further accelerates postexercise liver, but not muscle glycogen repletion compared with glucose ingestion in trained athletes.

13C magnetic resonance spectroscopy; carbohydrate; recovery; fructose; endurance exercise

NEW & NOTEWORTHY

This is the first study to assess both muscle and liver glycogen repletion postexercise after ingesting different types of carbohydrates in large amounts. We observed that sucrose ingestion accelerates postexercise liver glycogen repletion compared with glucose ingestion in spite of lower insulinemia and reduced gut discomfort. Therefore, when rapid recovery of endogenous carbohydrate stores is a goal, ingestion of sucrose at 1.5 g·kg−1·h−1 would be more appropriate than glucose.

CARBOHYDRATES ARE A MAIN substrate source used during prolonged moderate- to high-intensity exercise (35, 42). Both exogenous and endogenous carbohydrate stores can contribute to carbohydrate oxidation during exercise. Endogenous carbohydrate stores include liver and skeletal muscle glycogen, which can provide sufficient energy to sustain 45–60 min of high-intensity exercise (8, 10). However, at longer exercise durations (>60 min) endogenous glycogen stores may become depleted, causing early fatigue (1, 4–6, 9, 16, 20, 39). Because of the apparent relationship between glycogen depletion and exercise capacity (1, 4–6, 9, 12, 19, 20), the main factor determining the time needed to recover from exhaustive exercise is the rate of glycogen repletion. This is particularly relevant when exercise performance needs to be regained within 24 h, for example during tournament-style competitions or in between stages in races such as during the Tour de France.

Previous studies have shown that muscle glycogen repletion rates can reach maximal values when glucose (polymer) are ingested in an amount of 1.2 g·kg−1·h−1 (2, 43), with no further improvements at higher glucose ingestion rates (18). It has been speculated that postexercise muscle glycogen synthesis rates may be further increased when ingesting multiple transportable carbohydrates (i.e., mix of glucose and fructose). Glucose and fructose are absorbed by several similar (GLUT2, GLUT8, and GLUT12) as well as different intestinal transporters (SGLT1 and GLUT5, respectively) (24, 37). Hence the combined ingestion of both glucose and fructose may augment intestinal carbohydrate uptake and accelerate their subsequent delivery into the circulation (24, 37). To date, only one study investigated this hypothesis, showing no further improvements in postexercise muscle glycogen repletion rates after the ingestion of ~1.2 g·kg−1·h−1 (or 90 g/h) of multiple transportable carbohydrates compared with an equivalent dose of glucose (44).

The use of multiple transportable carbohydrates is potentially more relevant for liver glycogen repletion, as fructose is preferentially metabolized and retained in the liver (30). Factors that contribute to this are the high first-pass extraction of fructose by the liver and the high hepatic expression of fructokinase and triokinase, which are essential enzymes for the metabolism of fructose (30). Furthermore, it has been shown that intravenously administered fructose leads to greater increases in liver glycogen content when compared with intravenous glucose administration (33). Yet few studies have tried to assess the effects of carbohydrate ingestion on postexercise.
liver glycogen repletion (9, 14, 15, 31). This is mainly due to obvious methodological limitations, as liver biopsies are not considered appropriate for measuring liver glycogen concentrations for research purposes in vivo in humans (17). With the introduction of $^{13}$C magnetic resonance spectroscopy ($^{13}$C MRS), a noninvasive measurement to study changes in liver and muscle glycogen (40, 41), it has been demonstrated that postexercise liver glycogen resynthesis is stimulated by carbohydrate ingestion (9, 14, 15). Only two studies assessed the effects of fructose ingestion on postexercise liver glycogen resynthesis rates. Décombaz et al. (14) reported elevated liver glycogen resynthesis rates when coningesturing fructose with maltodextrin ($-0.93$ g·kg$^{-1}$·h$^{-1}$), whereas Casey et al. (9) reported no differences in postexercise liver glycogen resynthesis following ingestion of $-0.25$ g·kg$^{-1}$·h$^{-1}$ glucose vs. sucrose (9). No study has assessed the impact of ingesting multiple transportable carbohydrates on both liver and muscle glycogen resynthesis when optimal amounts of carbohydrate are ingested during postexercise recovery.

We hypothesize that ingestion of large amounts of sucrose leads to higher liver and muscle glycogen resynthesis rates compared with the ingestion of the same amount of glucose. To test this hypothesis, 15 well-trained cyclists completed glycogen-depleting exercise, after which we applied $^{13}$C MRS to compare liver and muscle glycogen resynthesis rates following ingestion of $1.5$ g·kg$^{-1}$·h$^{-1}$ sucrose or $1.5$ g·kg$^{-1}$·h$^{-1}$ glucose during $5$ h of postexercise recovery.

**METHODS**

**Subjects.** Fifteen well-trained male cyclists participated in this study [age $22\pm4$ (SD) yr, body weight $74.4\pm7.5$ kg, body mass index $22.6 \pm 1.8$ kg/m$^2$, maximal workload capacity ($W_{\text{max}}$) $350\pm30$ W, peak oxygen uptake ($V_{\text{O}_2\text{peak}}$) $61.5\pm5.2$ ml·kg$^{-1}$·min$^{-1}$]. Subjects were fully informed of the nature and possible risks of the experimental procedures, before written informed consent was obtained. Trials were conducted at the Newcastle Magnetic Resonance Centre (Newcastle-upon-Tyne, UK) in accordance with the Second Declaration of Helsinki and following approval from the Northumbria University Faculty of Health and Life Sciences Ethics Committee.

**Preliminary testing.** All subjects participated in a screening session, which was performed $\pm 1$ wk before the first experiment. Subjects performed an incremental cycling test on an electromagnetically braked cycle ergometer (Velotron; RacerMate, Seattle, WA) to determine maximal workload capacity ($W_{\text{max}}$) and peak oxygen uptake ($V_{\text{O}_2\text{peak}}$). Following a 5-min warm-up at 100 W, the workload began at 150 W and was increased by 50 W every 2.5 min to exhaustion (27). Expired gas was sampled continuously to determine oxygen uptake (Oxycon gas analyser; CareFusion, San Diego, CA).

**Diet and physical activity.** All subjects received the same standardized dinner ($2,797$ kJ; $666$ kcal; providing $23.9$ g fat, $83.7$ g carbohydrate, and $23.9$ g protein) the evening before each test day. All volunteers refrained from exhaustive physical activity $24$ h before each main trial and kept their diet as constant as possible $2$ days before each experimental day. In addition, subjects filled in food intake and physical activity diaries for $2$ days before the start of the first and second trials.

**Study design.** Participants performed two trials in a randomized, double-blind, crossover design separated by at least $7$ days. During each trial, they were first subjected to a glycogen depletion protocol on a cycle ergometer. Thereafter subjects were studied for $5$ h while ingesting only glucose in the control trial (GLU) or sucrose in the SUC trial. During the $5$h postexercise recovery period, subjects remained at rest in a supine position. Magnetic resonance spectroscopy (MRS) was performed immediately postexercise and after $2$ and $5$ h of postexercise recovery to determine liver and muscle glycogen concentrations. In addition, magnetic resonance imaging (MRI) was performed immediately postexercise and after $2$ and $5$ h of postexercise recovery to determine liver volume...

**Experimental protocol.** Participants arrived at Newcastle Magnetic Resonance Centre at 0700-0730 following a $12$-h fast. Liver and muscle glycogen depletion was established by performing an intense exercise protocol on an electromagnetically braked cycle ergometer (26). The exercise protocol started with a $10$-min warm-up at $50\% W_{\text{max}}$. Thereafter subjects cycled for $2$-min block periods at alternating workloads of $90\%$ and $50\% W_{\text{max}}$, respectively. This was continued until subjects were no longer able to complete a $2$-min, $90\% W_{\text{max}}$ exercise period at a cycling cadence of $60$ rpm. At this point, the high-intensity blocks were reduced to $80\% W_{\text{max}}$, after which the same regimen was continued. When subjects were no longer able to complete the $2$-min blocks at $80\% W_{\text{max}}$, the exercise intensity of the blocks was further reduced to $70\%$. Subjects were allowed to stop when pedaling speed could not be maintained at $70\% W_{\text{max}}$. Water was provided ad libitum during the exercise protocol. Two fans were placed $1$ m from the subjects to provide cooling and air circulation during the exercise protocol. After cessation of exercise, gastrointestinal (GI) comfort was assessed using a visual analog scale. Subsequently, the participants underwent a basal MRS and MRI measurement for $-45$ min (Fig. 1). After this, they were allowed to take a brief ($<15$ min) shower before the postexercise recovery period started. While supine, a catheter was inserted into an antecubital vein of the forearm to allow frequent blood sampling. Following a resting...
blood sample (10 ml), subjects filled out another visual analog scale for GI comfort before the first test drink was given (t = 0 min). Participants were observed for the following 5 h, during which they received a drink with a volume of 3.33 ml/kg every 30 min until t = 270 min. Blood samples were taken at 15-min intervals for the first 90 min of recovery and every 30 min thereafter until t = 300 min. Further visual analog scales for GI comfort were completed every 30 min until t = 300 min. Because of time constraints of the MR measurement it was not possible to acquire a blood sample and collect a visual analog scale at time point t = 150 min. At t = 120 and 300 min in the postexercise recovery period another MR measurement was performed to assess liver and muscle glycogen concentrations as well as liver volume.

GI (dis)comfort. Subjects were asked to fill out computerized visual analog scales to assess GI comfort. The visual analog scales consisted of 16 questions. Each question started with “To what extent are you experiencing . . . right now?” and was answered by ticking a 100-mm analog scales to assess GI comfort. The visual analog scales consisted of six questions related to upper GI symptoms (nausea, general stomach problems, belching, an urge to vomit, heartburn, stomach cramps), four questions related to lower GI symptoms (flatulence, an urge to defecate, intestinal cramps, diarrhea), and six questions related to central or other symptoms [dizziness, a headache, an urge to urinate, a bloated feeling, side aches (left), side aches (right)].

Drinks. Subjects received a drink volume of 3.33 ml/kg every 30 min during recovery to ensure a given dose of 1.5 g kg⁻¹ h⁻¹ glucose (GLU) or 1.5 g kg⁻¹ h⁻¹ sucrose (SUC). To minimize differences in carbon isotope ratio between GLU and SUC, similar plant sources with low natural ¹³C enrichments (i.e., wheat, potato, and beet sugar, all of which use C₃ metabolism) were selected for use in this study. The carbohydrates in the glucose drink (GLU) consisted of 60% dextrose monohydrate (Roquette, Lestrem, France) and 40% maltodextrin (MD14; AVEBE, Veendam, The Netherlands). The carbohydrate in the sucrose drink (SUC) consisted of 100% sucrose derived from sugar beet (AB Sugar, Peterborough, United Kingdom). Both drinks contained 20 mmol/l NaCl (Tesco, Cheshunt, United Kingdom).

Measurement of muscle and liver glycogen concentrations. Glycogen concentration was determined from the magnitude of the natural abundance signal from the C-1 carbon of glycogen at a frequency of 100.3 ppm. A Philips 3 Tesla Achieva scanner (Philips Healthcare, Best, The Netherlands) was used with a 6-cm-diameter ¹³C surface coil with integral ¹H decoupling surface coil (PulseTec, Worton under Edge, UK) to measure muscle glycogen concentration and an in-house-built 12-cm ¹³C/¹H surface coil used to measure liver glycogen concentration. The intraindividual coefficient of variation of hepatic glycogen content measured by ¹³C MRS has been shown to be 7% (36).

For muscle glycogen concentration measurements, the surface coil was placed over the widest part of the vastus lateralis muscle and was held in position with fabric straps to prevent movement. Pulse power was calibrated to a nominal value of 80° by observing the power-dependent variation in signal from a fiducial marker located in the coil housing, containing a sample exhibiting ¹³C signal with short T₁ (213 mM [2-¹³C]-acetone and 25 mM GdCl₃ in water). Automated shimming was carried out to ensure that the magnetic field within the scanner was uniform over the active volume of the ¹³C coil. The ¹³C spectra were acquired over 15 min using a nonlocalized ¹H decoupled ¹³C pulse-acquire sequence [repetition time (TR) 120 ms, spectral width 8 kHz, 7,000 averages, Wideband, Alternating-Phase, Low-Power Technique for Zero Residual Splitting (WALTZ) decoupling]. ¹H decoupling was applied for 60% of the ¹³C signal acquisition to allow a relatively fast TR of 120 ms to be used within specific absorption rate safety limitations.

For liver glycogen measurements the ¹³C/¹H surface coil was placed over the right lobe of the liver. Spectra were acquired over 15 min using non-localized ¹H decoupled ¹³C pulse acquisition sequences (TR 300 ms, spectral width 8 kHz, 2,504 averages, WALTZ decoupling, nominal ¹³C tip angle of 80°). Spectral images were obtained at the start of each study to confirm optimal coil position relative to the liver.

Tissue glycogen concentration was calculated from the amplitude of the C1-glycogen ¹³C signal using Java Based Magnetic Resonance User Interface (jMRUI) version 3.0 and the Advanced Method for Accurate, Robust and Efficient Spectral Fitting (AMARES) algorithm (7). For each subject the separation between radio frequency coil and muscle/liver tissue was measured from ¹H images, and ¹³C coil loading was assessed from ¹³C flip angle calibration data. Tissue glycogen concentration was determined by comparison of glycogen signal amplitude to spectra acquired from liver- and leg-shaped phantoms filled with aqueous solutions of glycogen (100 mM) and potassium chloride (70 mM). Phantom data were acquired at a range of flip angles and separation distances between coil and phantom. Quantification of each human ¹³C spectrum employed a phantom data set matched to body geometry and achieved flip angle so that account differences in coil sensitivity profile and loading were taken into account for each subject.

Measurement of liver volume. A turbo spin echo (TSE) sequence was used to obtain T₂-weighted axial images of the liver with a repetition time (TR) of 1.687 ms. The matrix size was 188 × 152 mm, with a field of view of (303 × 240 × 375) mm. The body coil was used for both transmission and reception. Slice thickness was 10 mm with a 0-mm gap. Scans were obtained on expiration. The total number of liver slices used for volume analysis differed between subjects because of anatomical differences but numbered on average 20 slices. Liver volumes were measured in the open-source Java image-processing program ImageJ (38).

Calculation of liver glycogen content. Total liver glycogen content was calculated by multiplying liver volume with liver glycogen concentration. Subsequent conversion from millimolar to grams was performed by using the molar mass of a glycosyl unit (i.e., 162 g/M). Plasma analysis. Blood samples (10 ml) were collected in EDTA-containing tubes and immediately centrifuged at 3,000 rpm for 10 min at 4°C. Plasma was then aliquoted and stored at −180°C for subsequent determination of glucose and lactate concentrations (Randox Daytoma spectrophotometer; Randox, Ireland), insulin (IBL International, Hamburg, Germany), and nonesterified fatty acid concentrations (WAKO Diagnostics, Richmond, VA).

Statistics. Sample size estimation was based on previous data on liver glycogen content (14). On the basis of this, the expected effect size was calculated from the difference in postexercise liver glycogen content after ingesting a mixture of maltodextrin with fructose vs. glucose (polymer) (52 ± 23 vs. 23 ± 9 g, respectively). A sample size of n = 10 in a crossover design would provide statistical power above 90% with an α-level of 0.05. We therefore recruited 15 participants to ensure adequate power and ample data sets. Unless otherwise stated, all data are expressed as means ± SD. Differences between primary outcomes in the text and the data in the figures are presented as means ± 95% confidence interval (CI). All data were analyzed by two-way repeated measures ANOVA with treatment (GLU vs. SUC) and time as within-subject factors. In case of a significant interaction, Bonferroni post hoc tests were applied to locate the differences. For non-time-dependent variables, a paired Student’s t-test was used to compare differences between treatments. A P value <0.05 was used to determine statistical significance. All calculations were performed by using the SPSS 21.0.0.0 software package.

RESULTS

Glycogen depletion protocol. Maximal workload capacity measured during preliminary testing averaged 350 ± 30 W (4.75 ± 0.6 W/kg). Consequently, average workload settings in
the depletion protocol were 315 ± 27, 280 ± 24, 245 ± 21, and 175 ± 15 W for the 90, 80, 70, and 50% Wmax workload intensities, respectively. On average, subjects cycled a total of 21 ± 7 and 19 ± 5 high-intensity blocks, which resulted in a total cycling time of 93 ± 27 and 89 ± 21 min in the SUC and GLU experiments, respectively. Total cycling time did not differ between trials (P = 0.434).

Drink ingestion and gastrointestinal complaints. The total amount of drink ingested in both treatments was 2.48 ± 0.25 liters. The first drinks were ingested 75 ± 7 min after cessation of exercise, because of timing of the MR measurements. Subjects reported upper GI issues following ingestion of the glucose drink only, and these issues included nausea, general stomach problems, belching, and urge to vomit. These symptoms all displayed significant differences over time and between treatments (time × treatment, P < 0.05; data not shown), and for every symptom the sucrose drink was better tolerated than the glucose drink.

Liver glycogen concentration. No significant differences in baseline liver glycogen concentrations were found between SUC and GLU (P = 0.210; Table 1). Liver glycogen concentrations increased significantly over time during postexercise recovery in both SUC and GLU (P < 0.001). Liver glycogen repletion rates during 5 h of postexercise recovery in SUC and GLU were 3.4 ± 0.24 and 1.7 ± 0.24 mmol·l⁻¹·h⁻¹, respectively (P = 0.002). Differences in liver glycogen repletion rates between SUC and GLU were 0.9 mmol·l⁻¹·h⁻¹ (95% CI: 0.4 to 11.2 mmol·l⁻¹·h⁻¹).

Liver volume. Liver volume data are shown in Table 1. Over the 5-h postexercise recovery period, liver volume increased significantly in SUC (P = 0.036), whereas no significant changes were observed in GLU (P = 0.151). A significant time × treatment interaction was found between SUC and GLU (P = 0.001).

Liver glycogen content. Liver glycogen content increased over time in both treatments (P < 0.01; Fig. 2). Over time, liver glycogen content increased significantly more in the SUC compared with the GLU treatment (time × treatment interaction, P < 0.001). Liver glycogen repletion rates during 5 h of postexercise recovery in SUC and GLU were 6.6 ± 3.3 vs. 3.3 ± 3.0 g/h, respectively (P = 0.002). Differences in liver glycogen repletion rates between SUC and GLU were 3.4 g/h (95% CI: 1.6–5.1 g/h), leading to a 17-g difference (95% CI: 8–26 g) over the 5-h recovery period.

Liver glycogen concentration and liver volume

<table>
<thead>
<tr>
<th>Time, min</th>
<th>0</th>
<th>120</th>
<th>300</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver glycogen concentration, mmol/l</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GLU</td>
<td>167 ± 65</td>
<td>191 ± 66#</td>
<td>234 ± 81#@</td>
</tr>
<tr>
<td>SUC</td>
<td>183 ± 47</td>
<td>219 ± 63#</td>
<td>280 ± 72##</td>
</tr>
<tr>
<td>Liver volume, liters</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GLU</td>
<td>1.79 ± 0.28</td>
<td>1.70 ± 0.24#</td>
<td>1.72 ± 0.24</td>
</tr>
<tr>
<td>SUC</td>
<td>1.80 ± 0.26</td>
<td>1.78 ± 0.24*</td>
<td>1.89 ± 0.28##</td>
</tr>
</tbody>
</table>

Values are means ± SD. Liver glycogen concentration (mmol/l) and liver volume (liters) at t = 0, 120, and 300 min postexercise, after ingesting 1.5 g·kg⁻¹·h⁻¹ glucose (n = 15; GLU) or sucrose (n = 15; SUC). Mean values were *significantly different from baseline values (P < 0.05), @significantly different from values at 120 min (P < 0.05), and #significantly different from GLU (P < 0.05). GLU; glucose; SUC, sucrose.

**Fig. 2.** Liver glycogen contents during 5 h of postexercise recovery while ingesting glucose or sucrose in well-trained cyclists (n = 15). #P < 0.05, significantly different compared with baseline values; @P < 0.05, significantly different compared with values at 120 min; *P < 0.05, significantly different from the glucose treatment.

**Muscle glycogen concentration.** No significant differences in baseline muscle glycogen concentrations were observed between SUC and GLU (P = 0.940; Fig. 3). Muscle glycogen concentrations increased significantly over the 5-h recovery period in both SUC and GLU (P < 0.001). No significant differences were observed between treatments (time × treatment, P = 0.673). Muscle glycogen repletion rates during 5 h of postexercise recovery in SUC and GLU were 11 ± 3 vs. 10 ± 5 mmol·l⁻¹·h⁻¹, respectively (P = 0.558). Differences in muscle glycogen repletion rates between SUC and GLU were 0.9 mmol·l⁻¹·h⁻¹ (95% CI: −1.9 to 3.6 mmol·l⁻¹·h⁻¹).

**Fig. 3.** Muscle glycogen concentrations during 5 h of postexercise recovery while ingesting glucose or sucrose in well-trained cyclists (n = 15). #P < 0.05, significantly different compared with baseline values; @P < 0.05, significantly different compared with values at 120 min; *P < 0.05, significantly different from the glucose treatment.
min ($P < 0.05$). Plasma lactate concentrations increased significantly after 15 min in the SUC trial compared with GLU and remained significantly higher over the entire postexercise recovery period ($P < 0.01$; Fig. 4B). Plasma insulin concentrations increased during the first 120 min of postexercise recovery. Thereafter plasma insulin concentrations decreased but remained elevated compared with baseline values during the entire postexercise recovery period (Fig. 4C). Plasma insulin concentrations were significantly higher in the GLU compared with the SUC treatment at $t = 45, 75,$ and 90 min ($P < 0.05$). Plasma nonesterified fatty acid (NEFA) concentrations decreased immediately after carbohydrate ingestion and remained low over the entire recovery period, with no differences between treatments (Fig. 4D).

**DISCUSSION**

In this experiment we observed that sucrose ingestion (1.5 g·kg$^{-1}$·h$^{-1}$) during recovery from exhaustive exercise results in more rapid liver glycogen repletion, despite lower plasma insulin levels, when compared with the ingestion of glucose. Ingestion of sucrose or glucose did not result in differences in postexercise muscle glycogen repletion rates.

Carbohydrate ingestion during 5 h of postexercise recovery allowed substantial increases in muscle glycogen concentrations (Fig. 3). This represents muscle glycogen repletion rates of $10 \pm 5$ mmol·l$^{-1}$·h$^{-1}$ after glucose ingestion and $11 \pm 3$ mmol·l$^{-1}$·h$^{-1}$ after sucrose ingestion. Assuming a skeletal muscle mass density of 1.112 g·cm$^{-3}$ (46) and a wet-to-dry mass fraction of 0.428 (22), our muscle glycogen repletion rates assessed using $^{13}$C MRS would translate to glycogen repletion rates of $39 \pm 20$ and $42 \pm 11$ mmol·kg$^{-1}$·h$^{-1}$, respectively. These values are in line with previously published data on postexercise muscle glycogen resynthesis rates when ingesting ample amounts of carbohydrate ($\sim 1.2$ g·kg$^{-1}$·h$^{-1}$), based upon muscle biopsy collection and concomitant muscle glycogen analyses, showing values ranging between 30 and 45 mmol·kg$^{-1}$·h$^{-1}$ (3, 23, 43, 44). We did not observe differences in muscle glycogen repletion rates following ingestion of either sucrose or glucose (polymers) during the 5-h postexercise recovery period ($P = 0.558$). Hence muscle glycogen resynthesis rates are not limited by exogenous carbohydrate availability when large amounts of glucose, glucose polymers, and/or sucrose ($\geq 1.2$ g·kg$^{-1}$·h$^{-1}$) are consumed. This supports the contention that ingestion of $\geq 1.2$ g carbohydrate·kg$^{-1}$·h$^{-1}$ maximizes postexercise muscle glycogen synthesis rates. This also implies that the limitation in exogenous carbohydrate oxidation rates residing in the rate of intestinal glucose absorption does not impose a restriction for postexercise muscle glycogen synthesis in a postexercise resting condition.

After exhaustive exercise, the ingestion of glucose and sucrose resulted in liver glycogen repletion rates of $14 \pm 12$ and $19 \pm 8$ mmol·l$^{-1}$·h$^{-1}$, respectively. These liver glycogen repletion rates together with our observed liver glycogen content values (Fig. 2) are comparable to previous observations made by Décombaz et al. (14). However, we extend on previous work by showing a doubling of liver glycogen repletion rates during recovery from exercise when sucrose as opposed to glucose (polymers) was ingested (6.6 $\pm$ 3.3 vs. 3.3 $\pm$ 3.0 g/h, respectively; $P = 0.002$). When looking at the present data together with the results of Décombaz et al. (14), it can be concluded that ingestion of both submaximal ($\sim 0.93$ g·kg$^{-1}$·h$^{-1}$) and maximal amounts (1.5 g·kg$^{-1}$·h$^{-1}$) of multiple transportable carbohydrates further accelerates postexercise liver glycogen repletion compared with the ingestion of glucose (polymers) only. These observations can be attributed to the differential effects that glucose and fructose exert on hepatic carbohydrate metabolism. Glucose is a relatively poor substrate for hepatic glycogen synthesis (14, 32, 33), and much of it seems to be released into the systemic circulation to be either oxidized or stored as muscle glycogen (7, 10, 11). In
DISCLOSURES
This study was co-funded by Sugar Nutrition UK and Kenniscentrum Suiker en Voeding Nederland. Sugar Nutrition UK and Kenniscentrum Suiker en Voeding Nederland had no input into the data analysis, interpretation, or conclusions.

AUTHOR CONTRIBUTIONS

REFERENCES

ACKNOWLEDGMENTS
The authors wish to thank the volunteers for their time and effort in participating in this study. We also thank J. Maessen for data acquisition, R. Veasy, J. Forster, and T. Esche for blinding and test drink preparation, and L. Ward, D. Wallace, and T. Hodgson for assistance with magnetic resonance examinations.

This trial was registered at clinicaltrials.gov as NCT02344381.