

## High rates of exogenous carbohydrate oxidation from a mixture of glucose and fructose ingested during prolonged cycling exercise

Roy L. P. G. Jentjens and Asker E. Jeukendrup\*

Human Performance Laboratory, School of Sport and Exercise Sciences, University of Birmingham, Edgbaston B15 2TT, UK

(Received 12 August 2004 – Revised 26 October 2004 – Accepted 4 November 2004)

A recent study from our laboratory has shown that a mixture of glucose and fructose ingested at a rate of 1.8 g/min leads to peak oxidation rates of approximately 1.3 g/min and results in approximately 55% higher exogenous carbohydrate (CHO) oxidation rates compared with the ingestion of an isocaloric amount of glucose. The aim of the present study was to investigate whether a mixture of glucose and fructose when ingested at a high rate (2.4 g/min) would lead to even higher exogenous CHO oxidation rates ( $>1.3$  g/min). Eight trained male cyclists ( $VO_{2max}$ :  $68 \pm 1$  ml/kg per min) cycled on three different occasions for 150 min at 50% of maximal power output ( $60 \pm 1\%$   $VO_{2max}$ ) and consumed either water (WAT) or a CHO solution providing 1.2 g/min glucose (GLU) or 1.2 g/min glucose+1.2 g/min fructose (GLU+FRUC). Peak exogenous CHO oxidation rates were higher ( $P<0.01$ ) in the GLU+FRUC trial compared with the GLU trial (1.75 (SE 0.11) and 1.06 (SE 0.05) g/min, respectively). Furthermore, exogenous CHO oxidation rates during the last 90 min of exercise were approximately 50% higher ( $P<0.05$ ) in GLU+FRUC compared with GLU (1.49 (SE 0.08) and 0.99 (SE 0.06) g/min, respectively). The results demonstrate that when a mixture of glucose and fructose is ingested at high rates (2.4 g/min) during 150 min of cycling exercise, exogenous CHO oxidation rates reach peak values of approximately 1.75 g/min.

### Substrate utilization: Stable isotopes: Intestinal carbohydrate transport

Numerous studies have shown that carbohydrate (CHO) supplementation during prolonged exercise at moderate to high intensities can increase exercise performance (Coyle *et al.* 1986; Coggan & Coyle, 1987). This effect is thought to be due to the prevention of hypoglycaemia and the maintenance of high rates of CHO oxidation late in exercise when endogenous CHO stores become depleted (Coyle *et al.* 1986; Bosch *et al.* 1994). Therefore, high exogenous CHO oxidation rates have the potential to improve prolonged exercise performance.

In a recent review we suggested that the maximal oxidation rate of (most) ingested carbohydrates is 1.0–1.1 g/min (Jeukendrup & Jentjens, 2000). When large amounts of glucose or glucose polymers are ingested ( $>1.2$  g/min; Jentjens *et al.* 2004b), intestinal glucose transporters (SGLT1) may become saturated and therefore intestinal CHO absorption may be a limiting factor for exogenous CHO oxidation (Hawley *et al.* 1992; Jeukendrup *et al.* 1999; Jeukendrup & Jentjens, 2000; Jentjens *et al.* 2004b). Of note, free fructose and most probably fructose released during sucrose hydrolysis use a different intestinal transporter (GLUT-5) from glucose (SGLT1; Davidson & Leese, 1977; Sandle *et al.* 1983; Burant *et al.* 1992; Ferraris & Diamond, 1997). Interestingly, Shi *et al.* (1995) demonstrated with an intestinal perfusion study that a beverage containing glucose and fructose resulted in approximately 65% higher CHO absorption rates compared with an isoenergetic glucose solution. This effect was attributed to the separate intestinal transport mechanisms for glucose and fructose.

We have recently shown that a mixture of glucose+fructose (Jentjens *et al.* 2004b), glucose+sucrose (Jentjens *et al.* 2004c) or glucose+fructose+sucrose (Jentjens *et al.* 2004a) results in approximately 20–55% higher exogenous CHO oxidation rates compared with the ingestion of an isocaloric amount of glucose and can lead to peak oxidation rates of approximately 1.7 g/min (Jentjens *et al.* 2004a). Although speculative, a faster intestinal CHO absorption might have increased the availability of exogenous CHO for oxidation and hence this could explain the high exogenous CHO oxidation rates ( $>1.1$  g/min) observed when a mixture of glucose+fructose or glucose+sucrose (+sucrose) was ingested (Jentjens *et al.* 2004a,b,c). Interestingly, ingestion of equal amounts of glucose and sucrose at a rate of 2.4 g/min resulted in relatively low oxidation rates (approximately 1.2 g/min; Jentjens *et al.* 2005). It has been suggested that when SGLT1 transporters become saturated (either from free glucose and/or glucose released from sucrose hydrolysis), hydrolysis of sucrose is inhibited (Gray & Ingelfinger, 1966; Sandle *et al.* 1983) and this might limit the amount of sucrose available for absorption and subsequent oxidation. This could explain why exogenous CHO oxidation rates were relatively low (1.2 g/min), despite a fairly high CHO intake rate (2.4 g/min; Jentjens *et al.* 2005).

The results of the studies described above suggest that the 'maximum' rate of exogenous CHO oxidation may depend on the amount of glucose and fructose available for absorption and the 'maximum' intestinal transport capacity for glucose and fructose. However, in our previous study (Jentjens *et al.* 2004b)

**Abbreviations:** CHO, carbohydrate; FRUC, fructose; GI, gastrointestinal; GLU, glucose; HR, heart rate; PDB, Pee Dee Bellemnitella (international standard); RPE, rate of perceived exertion; WAT, water;  $W_{max}$ , maximum power output.

\* **Corresponding author:** Dr Asker E. Jeukendrup, fax +44 (0)121 414 4121, email A.E.Jeukendrup@bham.ac.uk

the fructose ingestion rate (0.6 g/min) was relatively low and hence it is possible that not all fructose transporters were saturated. In an attempt to saturate the SGLT1 and GLUT-5 transporters, in the present study, fructose was ingested at a higher intake rate (1.2 g/min) and glucose was ingested at a rate of 1.2 g/min. Saturation of both CHO transport systems might further increase the rate of intestinal CHO absorption and this could potentially lead to even higher (and maximal) exogenous CHO oxidation rates. Therefore, we hypothesized that a mixture of glucose and fructose when ingested at a high rate (2.4 g/min) would further increase the rate of exogenous CHO oxidation (> 1.3 g/min).

## Methods

### Subjects

Eight trained male cyclists or triathletes took part in the present study. Their characteristics are presented in Table 1. Prior to participation, each of the subjects was fully informed of the purpose and the risks associated with the procedures, and a written informed consent was obtained. All subjects were healthy as assessed by a general health questionnaire. The study was approved by the Ethics Committee of the School of Sport and Exercise Sciences of the University of Birmingham, UK.

### Preliminary testing

At least 1 week before the start of the experimental trials an incremental cycle exercise test to volitional exhaustion was performed in order to determine the individual maximum power output ( $W_{\max}$ ) and maximal oxygen consumption ( $VO_{2\max}$ ). This test was performed on an electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands), modified to the configuration of a racing bicycle with adjustable saddle height and handlebar position. After reporting to the laboratory, body mass and height were recorded. Subjects then started cycling at 95 W for 3 min, followed by incremental steps of 35 W every 3 min until exhaustion. Heart rate (HR) was recorded continuously by a radiotelemetry heart rate monitor (Polar Vantage NV, Kempele, Finland).  $W_{\max}$  was calculated from the last completed work rate, plus the fraction of time spent in the final non-completed work rate multiplied by the work rate increment (Kuipers *et al.* 1985). The results were used to determine the work rate corresponding to 50%  $W_{\max}$ , which was later employed in the experimental exercise trials. Breath-by-breath measurements were performed throughout exercise using an online automated gas analysis system (Oxycon Pro, Jaeger, Hoechberg, Germany). The volume sensor was calibrated using a 3 litre calibration syringe and the gas analysers were calibrated using a  $CO_2$ - $N_2$  gas mixture (5.03%:94.97%). Oxygen uptake ( $VO_2$ ) was considered

to be maximal ( $VO_{2\max}$ ) when at least two of the three following criteria were met: (1) a levelling off of  $VO_2$  with increasing workload (increase of no more than 2 ml/kg per min); (2) HR within 10 beats/min of predicted maximum (HR 220 minus age); (3) RER > 1.05.  $VO_{2\max}$  was calculated as the average oxygen uptake over the last 60 s of the test. The  $VO_{2\max}$  and  $W_{\max}$  achieved during the incremental exercise test were 68 (SE 1) ml/kg per min and 376 (SE 12) W, respectively (Table 1).

### Experimental design

Each subject performed three exercise trials which consisted of 150 min of cycling at 50%  $W_{\max}$  while ingesting a glucose drink (GLU), a glucose+fructose drink (GLU+FRUC; ingested glucose-fructose ratio of 1:1) or plain water (WAT). In order to quantify exogenous glucose oxidation, corn-derived glucose monohydrate (Cerestar, Manchester, UK) and crystalline fructose (Krystar 300; A.E. Staley Manufacturing Company, Decatur, IL, USA) were used which have a high natural abundance of  $^{13}C$  (-10.70 and -10.69 ‰ v. Pee Dee Bellemnitella (PDB), respectively). The  $^{13}C$ -enrichment of the ingested glucose and fructose was determined by elemental analyser-isotope ratio mass spectrometry (IRMS; Europa Scientific GEO 20-20, Crewe, UK). To all CHO drinks 20 mmol/l NaCl (Sigma-Aldrich, Poole, UK) was added. The order of the experimental drinks was randomly assigned in a crossover design. Experimental trials were separated by at least 5 d. The composition of the experimental drinks is shown in Table 2.

### Diet and activity prior to testing

Subjects were asked to record their food intake and activity pattern 2 d prior to the first exercise trial and were then instructed to follow the same diet and exercise activities before the other three trials. In addition, 5–7 d prior to each experimental testing day, they were asked to perform an intense training session ('Glycogen depleting' exercise bout) in an attempt to empty any  $^{13}C$ -enriched glycogen stores. Subjects were further instructed not to consume any food products with a high natural abundance of  $^{13}C$  (CHO derived from  $C_4$  plants: corn, sugar cane) at least 1 week before and during the entire experimental period in order to reduce the background shift (change in  $^{13}CO_2$ ) from endogenous substrate stores.

### Protocol

Subjects reported to the Human Performance Laboratory in the morning (between 07.00 and 09.00 hours) after an overnight fast (10–12 h) and having refrained from any strenuous activity or drinking any alcohol in the previous 24 h. For a given subject, all trials were conducted at the same time of the day to avoid any

**Table 1.** Subject characteristics

(Mean values with their standard errors of the mean for eight subjects)

	Mean	SEM
Age (years)	26.3	2.6
Height (cm)	181.4	1.4
Body mass (kg)	74.3	1.8
$VO_{2\max}$ (ml/kg per min)	68.1	0.6
$W_{\max}$ (W)	376	12
$HR_{\max}$ (beats/min)	185	4

$HR_{\max}$ , maximal heart rate;  $W_{\max}$ , maximum power output.

**Table 2.** Composition of the three experimental beverages

	WAT	GLU	GLU+FRUC
Glucose (g/l)	–	92.3	92.3
Fructose (g/l)	–	–	92.3
NaCl (mmol/l)	–	20	20

GLU, ingestion of glucose; GLU + FRUC, ingestion of glucose and fructose; WAT, ingestion of water only.

influence of circadian variance. On arrival in the laboratory, a flexible 21-gauge Teflon catheter (Quickcath, Baxter) was inserted in an antecubital vein of an arm and attached to a three-way stopcock (Sims Portex, Kingsmead, UK) to allow for repeated blood sampling during exercise. The catheter was kept patent by flushing with 1.0–1.5 ml of isotonic saline (0.9%; Baxter) after each blood sample collection.

The subjects then mounted a cycle ergometer and a resting breath sample was collected in 10 ml Exetainer tubes (Labco Ltd, Brow Works, High Wycombe, UK), which were filled directly from a mixing chamber in duplicate in order to determine the  $^{13}\text{C}:^{12}\text{C}$  ratio in the expired air.

Next, a resting blood sample (8 ml) was taken and stored on ice and later centrifuged. Subjects then started a 150 min exercise bout at a work rate equivalent to 50%  $W_{\max}$  ( $60 \pm 1\%$   $\text{VO}_{2\max}$ ). Additionally, blood samples were drawn at 15 min intervals during exercise. Expiratory breath samples were collected every 15 min until the end of exercise.  $\text{VO}_2$ ,  $\text{VCO}_2$  ( $\text{CO}_2$  production) and RER were measured every 15 min for periods of 4 min using an online automated gas analysis system as previously described.

During the first 3 min of exercise subjects drank an initial bolus (600 ml) of one of the three experimental drinks: GLU, GLU+FRUC or WAT. Thereafter, every 15 min a beverage volume of 150 ml was provided. The total fluid provided during the 150 min exercise bout was 1.95 litres. The average rate of glucose intake in the GLU and GLU+FRUC trial was 1.2 g/min. Furthermore, in the GLU+FRUC trial subjects ingested on average 1.2 g/min fructose which brought the total CHO intake rate in the GLU+FRUC to 2.4 g/min.

Subjects were asked to rate their perceived exertion (RPE) for whole body and legs every 30 min on a scale from 6 to 20 using the Borg category scale (Borg, 1982). In addition, subjects were asked every 30 min to fill in a questionnaire in order to rate (possible) gastrointestinal problems (Jeukendrup *et al.* 2000). All exercise tests were performed under normal and standard environmental conditions (17–21°C dry bulb temperature and 55–65% relative humidity). During the exercise trials subjects were cooled with standing floor fans in order minimize thermal stress.

### Questionnaires

Subjects were asked to fill out a questionnaire every 30 min during the exercise trials. The questionnaire contained questions regarding the presence of gastrointestinal (GI) problems at that moment and addressed the following complaints; stomach problems, gastrointestinal cramping, bloated feeling, diarrhoea, nausea, dizziness, headache, belching, vomiting, and urge to urinate/defecate. While subjects were on the bike and continued their exercise each question was answered by simply ticking a box on the questionnaire that corresponded to the severity of the GI problem addressed. The items were scored on a 10-point scale (1 = not at all, 10 = very, very much). The severity of the GI symptoms was divided into two categories: severe and non-severe symptoms, as was previously described by Jeukendrup *et al.* (2000). Severe complaints included nausea, stomach problems, bloated feeling, diarrhoea, urge to vomit, stomach and intestinal cramps because these are symptoms that commonly impair performance and may bring with them health risks. The above symptoms were only registered as severe symptoms when a score of 5 or higher out of 10 was reported. When a score below 5 was given, they were registered

as non-severe. All other symptoms were registered as non-severe regardless of the score reported.

### Analyses

Blood samples were collected into pre-chilled EDTA-containing tubes (Beckton Dickinson, Plymouth, UK) and centrifuged at 2300 g and 4°C for 10 min. Aliquots of plasma were immediately frozen in liquid nitrogen and stored at  $-25^\circ\text{C}$  until analyses for glucose and lactate. Glucose (Glucose HK 125, ABX Diagnostics, Shefford, UK) and lactate (Lactic Acid 10, ABX Diagnostics, Shefford) were analysed on a COBAS MIRA semi-automatic analyser (ABX Diagnostics, Montpellier, France).

Breath samples were analysed for  $^{13}\text{C}:^{12}\text{C}$  ratio by gas chromatography continuous flow isotope ratio mass spectrometry (GC-IRMS; Europa Scientific). From indirect calorimetry ( $\text{VO}_2$  and  $\text{VCO}_2$ ) and stable isotope measurements (breath  $^{13}\text{CO}_2:^{12}\text{CO}_2$  ratio), oxidation rates of total fat, total CHO, endogenous CHO and exogenous glucose were calculated.

### Calculations

From  $\text{VCO}_2$  and  $\text{VO}_2$  (l/min), total CHO and fat oxidation rates (g/min) were calculated using stoichiometric equations of Frayn (1983) with the assumption that protein oxidation during exercise was negligible:

$$\text{CHO oxidation} = 4.55 \text{ VCO}_2 - 3.21 \text{ VO}_2 \quad (1)$$

$$\text{Fat oxidation} = 1.67 \text{ VO}_2 - 1.67 \text{ VCO}_2 \quad (2)$$

The isotopic enrichment was expressed as ‰ difference between the  $^{13}\text{C}:^{12}\text{C}$  ratio of the sample and a known laboratory reference standard according to the formula of Craig (1957):

$$\delta^{13}\text{C} = \left( \left( \frac{^{13}\text{C}:^{12}\text{C} \text{ sample}}{^{13}\text{C}:^{12}\text{C} \text{ standard}} \right) - 1 \right) \times 10^3 \text{ per mil} \quad (3)$$

The  $\delta^{13}\text{C}$  was then related to an international standard (PDB).

In the GLU and GLU+FRUC trials, the rate of exogenous glucose oxidation was calculated using the following formula (Mosora *et al.* 1976):

$$\begin{aligned} &\text{Exogenous glucose oxidation} \\ &= \text{VCO}_2 \times \left( \frac{\delta\text{Exp} - \delta\text{Exp}_{\text{bkg}}}{\delta\text{Ing} - \delta\text{Exp}_{\text{bkg}}} \right) \left( \frac{1}{k} \right) \quad (4) \end{aligned}$$

In which  $\delta\text{Exp}$  is the  $^{13}\text{C}$  enrichment of expired air during exercise at different time points,  $\delta\text{Ing}$  is the  $^{13}\text{C}$  enrichment of the ingested CHO solution,  $\delta\text{Exp}_{\text{bkg}}$  is the  $^{13}\text{C}$  enrichment of expired air in the WAT trial (background) at different time points and  $k$  is the amount of  $\text{CO}_2$  (in litres) produced by the oxidation of 1 g of glucose ( $k = 0.7467$  litres  $\text{CO}_2$  per g glucose).

Endogenous CHO oxidation was calculated by subtracting exogenous CHO oxidation from total CHO oxidation.

A methodological consideration when using  $^{13}\text{CO}_2$  in expired air to calculate exogenous substrate oxidation is the trapping of  $^{13}\text{CO}_2$  in the bicarbonate pool, in which an amount of  $\text{CO}_2$  arising from decarboxylation of energy substrates is temporarily trapped (Robert *et al.* 1987). However, during exercise the  $\text{CO}_2$  production increases severalfold so that a physiological steady-state condition will occur relatively rapidly, and  $^{13}\text{CO}_2$  in the expired air will be equilibrated with the  $^{13}\text{CO}_2/\text{H}^{13}\text{CO}_3^-$  pool,

respectively. Recovery of  $^{13}\text{CO}_2$  from oxidation will approach 100% after 60 min of exercise when dilution in the bicarbonate pool becomes negligible (Robert *et al.* 1987; Pallikarakis *et al.* 1991). As a consequence of this, all calculations on substrate oxidation were performed over the last 90 min of exercise (60–150 min).

#### Statistical analyses

Two-way ANOVA for repeated measures was used to compare differences in substrate utilization and in blood-related parameters over time between the trials. A Tukey *post hoc* was applied in the event of a significant *F* ratio. Where appropriate, comparison of variables between two conditions was conducted by using a Student's *t* test for paired samples. Data evaluation was performed using SPSS for Windows version 10.0 software package (SPSS, Chicago, IL, USA). All data are reported as means with their standard errors. Statistical significance was set at  $P < 0.05$ .

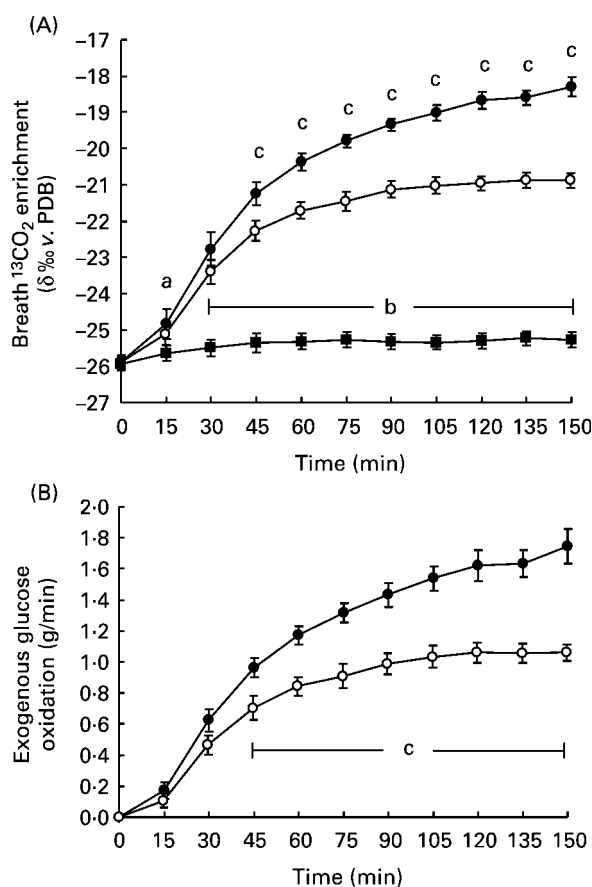
## Results

#### Stable-isotope measurements

Changes in isotopic composition of expired  $\text{CO}_2$  in response to exercise with ingestion of water (WAT), glucose (GLU) or a mixture of glucose and fructose (GLU+FRUC) are shown in Fig. 1(A). In the GLU and GLU+FRUC trials,  $^{13}\text{CO}_2$  enrichment of expired breath increased ( $P < 0.01$ ) from  $-25.87$  (SE 0.16) and  $-25.89$  (SE 0.20) ‰ v. PDB at rest to  $-20.88$  (SE 0.20) and  $-18.31$  (SE 0.26) ‰ v. PDB by the end of the 150 min exercise, respectively. From the 45 min point onwards, breath  $^{13}\text{CO}_2$  enrichment in the GLU+FRUC trial was significantly ( $P < 0.01$ ) higher compared with the GLU trial. During the WAT trial, there was also a significant increase in  $^{13}\text{CO}_2$  enrichment of the expired air ( $P < 0.01$ ). The rise in background  $^{13}\text{CO}_2$  enrichment during the WAT trial was relatively small (approximately 10–14%) compared with the rise in breath  $^{13}\text{CO}_2$  enrichment observed during the two CHO trials. Although the background shift was small in the present study, a background correction was made for the calculation of exogenous CHO oxidation in the two CHO trials by using the data from the WAT trial.

#### Oxygen uptake, rate of perceived exertion, total carbohydrate and fat oxidation

Data for  $\text{VO}_2$ , RER, total CHO and fat oxidation over the 60–150 min exercise period are shown in Table 3. There was no significant difference in  $\text{VO}_2$  between the three experimental trials. RER in the WAT trial was significantly lower ( $P < 0.01$ ) compared with the GLU and GLU+FRUC trial. During the 90–120 min and the 120–150 min exercise periods, RER was significantly higher in GLU+FRUC compared with GLU. The average CHO oxidation rates during the last 90 min of exercise were 1.56 (SE 0.13), 2.26 (SE 0.12) and 2.57 (SE 0.15) g/min for WAT, GLU and GLU+FRUC, respectively. The rate of CHO oxidation was significantly higher ( $P < 0.01$ ) after CHO ingestion compared with WAT ingestion (Table 3). Furthermore, CHO oxidation between 60 and 150 min was significantly higher ( $P < 0.05$ ) in GLU+FRUC compared with GLU. The ingestion of CHO (GLU and GLU+FRUC) resulted in significantly lower ( $P < 0.01$ ) fat oxidation rates compared with the WAT trial. Furthermore, fat oxidation rates were significantly lower ( $P < 0.01$ )



**Fig. 1.** Breath  $^{13}\text{CO}_2$  enrichment (A) and exogenous carbohydrate oxidation (B) during exercise without ingestion of carbohydrate (WAT, ■), with ingestion of glucose (GLU, ○) or with ingestion of glucose+fructose (GLU+FRUC, ●). Values are means with their standard errors represented by vertical bars ( $n$  8). a, Significant difference between WAT and GLU+FRUC ( $P < 0.05$ ); b, significant difference between WAT and CHO trials ( $P < 0.01$ ); c, significant difference between GLU and GLU+FRUC ( $P < 0.01$ ). PDB, Pee Dee Belemnite (international standard).

in GLU+FRUC compared with GLU. The average fat oxidation rates over the 60–150 min exercise period were 0.97 (SE 0.05), 0.68 (SE 0.05) and 0.51 (SE 0.04) g/min for WAT, GLU and GLU+FRUC, respectively. The relative contribution of substrates to total energy expenditure during the 60–150 min period of exercise is depicted in Fig. 2. Fat oxidation represented 61 (SE 3), 43 (SE 3) and 34 (SE 3) % of total energy expenditure in WAT, GLU and GLU+FRUC, respectively (WAT > GLU > GLU+FRUC;  $P < 0.05$ ) (Fig. 2).

#### Exogenous and endogenous carbohydrate oxidation

In the GLU and GLU+FRUC trials, the rate of exogenous CHO oxidation increased significantly ( $P < 0.01$ ) during exercise. However, in GLU, exogenous CHO oxidation rates levelled off after approximately 105–120 min of exercise (Fig. 1(B)), while the rate of exogenous CHO oxidation in GLU+FRUC increased until the end of exercise (no levelling off). Peak exogenous CHO oxidation rates were reached at the end of exercise (150 min) and were significantly higher ( $P < 0.01$ ) in the GLU+FRUC trial (1.75 (SE 0.11) g/min) compared with the GLU trial (1.06 (SE 0.04) g/min) (Fig. 1(B)). During the 60–150 min exercise period, exogenous CHO oxidation rates

**Table 3.** Mean oxygen uptake (VO<sub>2</sub>), rate of perceived exertion (RER), total carbohydrate (CHO) oxidation (CHO<sub>tot</sub>), total fat oxidation (FAT<sub>tot</sub>), endogenous CHO oxidation and exogenous glucose oxidation during cycling exercise with ingestion of water, glucose and glucose+fructose.

(Mean values with their standard errors for eight subjects)

	Time (min)	VO <sub>2</sub> (l/min)		RER		CHO <sub>tot</sub> (g/min)		FAT <sub>tot</sub> (g/min)		Endogenous CHO oxidation (g/min)		Exogenous glucose oxidation (g/min)	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
WAT	60–90	3.10	0.12	0.83	0.01 <sup>b</sup>	1.68	0.14 <sup>b</sup>	0.90	0.05 <sup>b</sup>	1.68	0.14 <sup>ae</sup>		
	90–120	3.14	0.12	0.81	0.01 <sup>b</sup>	1.54	0.13 <sup>b</sup>	0.98	0.06 <sup>b</sup>	1.54	0.13 <sup>ae</sup>		
	120–150	3.16	0.16	0.81	0.01 <sup>b</sup>	1.44	0.10 <sup>b</sup>	1.03	0.05 <sup>b</sup>	1.44	0.10 <sup>ae</sup>		
GLU	60–90	3.03	0.09	0.87	0.01	2.28	0.13	0.65	0.05 <sup>d</sup>	1.36	0.13	0.91	0.07 <sup>c</sup>
	90–120	3.06	0.09	0.87	0.01 <sup>d</sup>	2.27	0.11	0.67	0.05 <sup>c</sup>	1.25	0.12	1.03	0.07 <sup>c</sup>
	120–150	3.10	0.09	0.86	0.07 <sup>c</sup>	2.23	0.11 <sup>d</sup>	0.70	0.04 <sup>c</sup>	1.18	0.10	1.06	0.06 <sup>c</sup>
GLU+FRUC	60–90	2.94	0.10	0.89	0.01	2.49	0.15	0.53	0.05	1.18	0.15	1.31	0.07
	90–120	2.96	0.10	0.90	0.01	2.57	0.15	0.51	0.04	1.04	0.15	1.53	0.08
	120–150	2.98	0.10	0.90	0.01	2.64	0.18	0.50	0.04	0.97	0.16	1.67	0.10

GLU, ingestion of glucose; GLU+FRUC, ingestion of glucose and fructose; WAT, ingestion of water only.

<sup>abcde</sup>Significant difference between <sup>a</sup>WAT and GLU+FRUC ( $P < 0.01$ ); <sup>b</sup>WAT and CHO trials ( $P < 0.01$ ); <sup>c</sup>GLU and GLU+FRUC ( $P < 0.05$ ); <sup>d</sup>GLU and GLU+FRUC ( $P < 0.05$ ); <sup>e</sup>WAT and GLU ( $P < 0.05$ ).

were approximately 50% higher ( $P < 0.01$ ) in GLU+FRUC compared with GLU (Table 2; Fig. 1(B) and Fig. 2).

The rate of endogenous CHO oxidation was significantly lower ( $P < 0.05$ ) in the two CHO trials compared with the WAT trial (Table 3; Fig. 2). No significant difference was found in endogenous CHO oxidation rates between the GLU and GLU+FRUC trial. The average endogenous CHO oxidation rates over the 60–150 min exercise period were 1.56 (SE 0.13), 1.26 (SE 0.12) and 1.07 (SE 0.15) g/min for WAT, GLU and GLU+FRUC, respectively. During the last 90 min of exercise, endogenous CHO oxidation represented 39 (SE 3), 32 (SE 3) and 28 (SE 4) % of total energy expenditure in WAT, GLU and GLU+FRUC, respectively (WAT > GLU and GLU+FRUC;  $P < 0.05$ ) (Fig. 2).

#### Plasma glucose and lactate

No differences were observed in fasting plasma glucose concentrations between trials (Fig. 3(A)). In the WAT trial, plasma glucose concentrations decreased gradually during exercise,

reaching a nadir of 3.7 (SE 0.2) mmol/l at the end of exercise ( $t$  150 min). In contrast, a large increase in plasma glucose was observed following ingestion of GLU and GLU+FRUC, with peak values of 5.8 (SE 0.3) and 6.0 (SE 0.6) mmol/l respectively, reached 15 min into the exercise period. Thereafter, plasma glucose concentrations decreased to fasting levels and remained at values varying between 4.6 and 5.0 mmol/l for the duration of exercise. Plasma glucose concentrations were higher ( $P < 0.05$ ) throughout exercise in the two CHO trials compared with the WAT trial. There were no significant differences in plasma glucose concentrations between the GLU and GLU+FRUC trials.

Plasma lactate concentrations at rest in the WAT, GLU and GLU+FRUC trials were 1.1 (SE 0.2), 1.0 (SE 0.1) and 1.1 (SE 0.1) mmol/l, respectively ( $P > 0.05$ ; Fig. 3(B)). At all time points during exercise, plasma lactate concentrations were higher ( $P < 0.01$ ) in the GLU+FRUC trial compared with the WAT and GLU trials. No differences in plasma lactate concentrations were observed between the GLU and WAT trials.

#### Gastrointestinal discomfort and ratings of perceived exertion

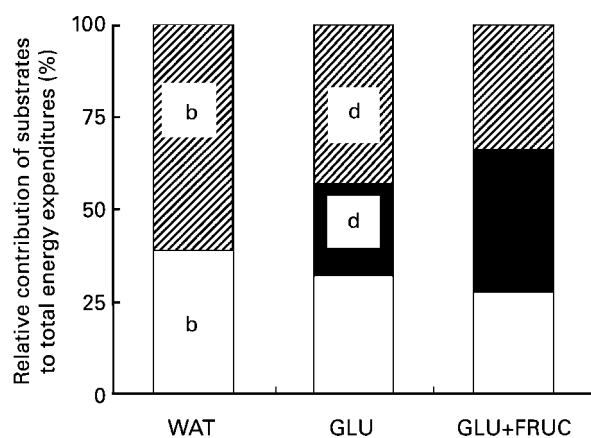
The most frequently reported complaints were urge to urinate, belching and bloated feeling. There were no differences (i.e. number of complaints or number of subjects that reported complaints) in GI discomfort between the three experimental trials, apart from one subject who reported severe stomach problems in the GLU+FRUC trial (stomach burn and bloated feeling).

No significant differences in RPE overall or RPE legs were observed between the three conditions. The mean values for RPE overall and RPE legs during 150 min of exercise were 11.7 (SE 0.4) and 11.8 (SE 0.4) for WAT, 11.6 (SE 0.3) and 11.9 (SE 0.4) for GLU, and 11.8 (SE 0.4) and 11.8 (SE 0.4) for GLU+FRUC.

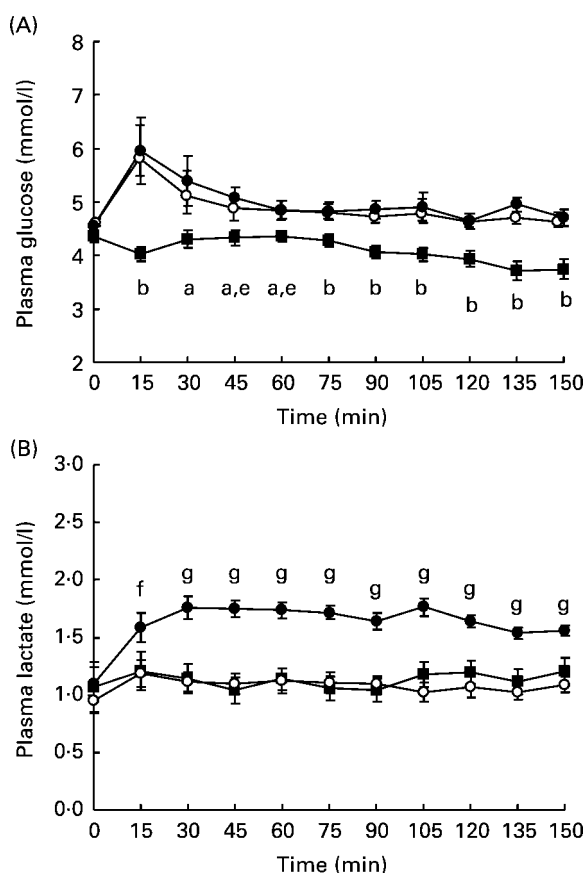
#### Discussion

The main finding of the present study was that combined ingestion of glucose and fructose at a rate of 1.2 and 1.2 g/min, respectively, resulted in peak exogenous CHO oxidation rates of 1.75 g/min (SE 0.11).

In a recent study from our laboratory, we have shown that ingestion of glucose at a rate of 1.2 g/min in combination with fructose at a rate of 0.6 g/min leads to peak exogenous oxidation



**Fig. 2.** Relative contributions of substrates to total energy expenditure calculated for the 60–150 min period of exercise without ingestion of carbohydrate (WAT), with ingestion of glucose (GLU) or with ingestion of glucose+fructose (GLU+FRUC). Values are means with their standard errors ( $n$  8). b, Significant difference between WAT and CHO trials ( $P < 0.05$ ); d, significant difference between GLU and GLU+FRUC ( $P < 0.05$ ). ▨, Fat; ■, exogenous carbohydrate; □, endogenous carbohydrate.



**Fig. 3.** Plasma glucose (A) and lactate (B) during exercise without ingestion of carbohydrate (WAT, ■), with ingestion of glucose (GLU, ○) or with ingestion of glucose+fructose (GLU+FRUC, ●). Values are means with their standard errors represented by vertical bars ( $n$  8). a, Significant difference between WAT and GLU+FRUC ( $P < 0.05$ ); b, significant difference between WAT and CHO trials ( $P < 0.01$ ); e, significant difference between WAT and GLU ( $P < 0.05$ ); f, GLU+FRUC significantly different from GLU and WAT ( $P < 0.05$ ); g, GLU+FRUC significantly different from GLU and WAT ( $P < 0.01$ ).

rates of 1.26 (SE 0.07) g/min (Jentjens *et al.* 2004b). When a mixture of glucose and sucrose was ingested at a rate of 1.2 and 0.6 g/min, respectively, almost similar rates of exogenous CHO oxidation were found (1.25 (SE 0.07) g/min; Jentjens *et al.* 2004c). More importantly, combined ingestion of glucose+sucrose and glucose+fructose resulted in approximately 20–55% higher exogenous CHO oxidation rates compared with the ingestion of an isocaloric amount of glucose (Jentjens *et al.* 2004b,c). These findings support the data of Shi *et al.* (1995) who demonstrated that a beverage containing two or three transportable CHO (glucose, fructose and sucrose) resulted in higher CHO and/or water absorption rates compared with an isoenergetic glucose solution (Shi *et al.* 1995). Our previous findings and those of Shi *et al.* (1995) might be explained by the fact that free fructose and most probably fructose released during sucrose hydrolysis uses a different intestinal transporter (GLUT-5) than glucose (SGLT1; Davidson & Leese, 1977; Sandle *et al.* 1983; Burant *et al.* 1992; Ferraris & Diamond, 1997) and hence intestinal CHO absorption might be increased when a mixture of multiple transportable CHO is consumed. Furthermore, studies in humans and rats have indicated that fructose absorption is enhanced by the presence of glucose (Holdsworth & Dawson, 1964; Fujisawa *et al.* 1991; Hoekstra & van den Aker, 1996;

Shi *et al.* 1997; Corpe *et al.* 1999). The facilitated fructose absorption probably occurs as a result of glucose-induced water streaming through the mucosal layer, also known as solvent drag (Holdsworth & Dawson, 1964; Fine *et al.* 1994; Hoekstra & van den Aker, 1996; Shi *et al.* 1997). This process requires the opening of tight junctions by glucose absorption and the subsequent movement of water through the paracellular pathway. Small solutes, including fructose, will move passively with water through the same pathway. In addition, glucose-induced water absorption increases intraluminal fructose concentrations and this might also lead to increased fructose transport (Holdsworth & Dawson, 1964; Fine *et al.* 1994). Thus, in addition to the different intestinal transport mechanisms for glucose and fructose, the stimulating effect of glucose on fructose absorption might further contribute to an increased intestinal CHO absorption rate when mixtures of multiple transportable CHO are ingested. It has been postulated that a faster intestinal CHO absorption might increase the availability of exogenous CHO for oxidation (Jentjens *et al.* 2004a,b,c, 2005) and this might explain the high exogenous CHO oxidation rates (approximately 1.25–1.75 g/min) observed in the present study and in our previous studies in which mixtures of glucose, sucrose and/or fructose were ingested (Jentjens *et al.* 2004a,b,c, 2005).

Ingestion of fructose in combination with other carbohydrates (present study; Jentjens *et al.* 2004a,b; L Moseley, GI Mainwaring, S Samuels, S Perry, CH Mann and AE Jeukendrup, unpublished results) or when ingested alone (Macdonald *et al.* 1978; Koivisto *et al.* 1981) has been shown to result in elevated lactate concentrations. The increased lactate concentrations following fructose ingestion might be due to the high activity of fructokinase, stimulation of pyruvate kinase and the fact that fructolysis bypasses phosphofructokinase (the main rate-controlling step in glycolysis). Fructose is therefore rapidly phosphorylated, resulting in increased concentrations of glycolytic intermediates which will lead to an increased glycolytic flux, evidenced by elevated plasma lactate concentrations. Furthermore, studies in animals and humans have shown that during absorption a considerable amount of absorbed glucose (Hanson & Parsons, 1976; Porteous, 1978; Nicholls *et al.* 1983; Bjorkman *et al.* 1990) or absorbed fructose (Bjorkman *et al.* 1984; Holloway & Parsons, 1984) is converted in the intestine into lactate, most of which is secreted into the portal vein. Most of the lactate will be converted into glucose by the liver. However, some lactate might escape from the liver into the systemic circulation and this could lead to increased plasma lactate concentrations. Although speculative, the higher plasma lactate concentrations following ingestion of GLU+FRUC may indicate faster intestinal CHO absorption and this may have caused the higher exogenous CHO oxidation rates.

It has been shown that when equal amounts of glucose and sucrose are ingested at a rate of 2.4 g/min (Jentjens *et al.* 2005), exogenous CHO oxidation rates are not much different from the oxidation rates found when glucose and sucrose are ingested at a rate of 1.2 and 0.6 g/min, respectively (Jentjens *et al.* 2004c). These findings indicate that the rate of exogenous CHO oxidation does not increase when the rate of sucrose intake is increased from 0.6 to 1.2 g/min and glucose is ingested simultaneously at a rate of 1.2 g/min (Jentjens *et al.* 2004c, 2005). On the contrary, the combined results from the present and a previous study (Jentjens *et al.* 2004b) suggest that an increase in the rate of fructose ingestion from 0.6 to 1.2 g/min when coingested with glucose at a rate of 1.2 g/min leads to higher peak oxidation rates

(1.75 (SE 0.11) v. 1.26 (SE 0.07) g/min, respectively). These data support the results of an earlier study from our laboratory, in which a mixture of glucose, fructose and sucrose when ingested at a rate of 1.2, 0.6 and 0.6 g/min, respectively, resulted in peak exogenous CHO oxidation rates of 1.70 (SE 0.07) g/min (Jentjens *et al.* 2004a). The present data and previous findings from our laboratory (Jentjens *et al.* 2004a) indicate that in order to achieve high exogenous CHO oxidation rates (approximately 1.7 g/min) a mixture of glucose+fructose (+sucrose) should be consumed at high intake rates (i.e. 1.2 g/min glucose+1.2 g/min fructose).

One potential limitation of our study is that we did not include an isoenergetic glucose (only) trial and hence it may be difficult to compare the results of the two CHO trials. However, it should be noted that we and others have previously demonstrated that the rate of exogenous CHO oxidation does not increase when the rate of glucose or maltodextrin ingestion is increased from 1.2 to 1.8 g/min (Wagenmakers *et al.* 1993; Jentjens *et al.* 2004b). Furthermore, we have recently shown that when glucose is ingested at a rate of 2.4 g/min, average exogenous glucose oxidation rates during the last 90 min of exercise are 1.01 (SE 0.04) g/min (Jentjens *et al.* 2004a). The rate of exogenous glucose oxidation in the present study (GLU trial) is almost similar to the oxidation rate observed in our previous study (Jentjens *et al.* 2004a), despite a 50% lower glucose ingestion rate (1.2 v. 2.4 g/min, respectively). The above findings indicate that maximal glucose oxidation rates are reached when glucose is ingested at a rate of approximately 1.2 g/min. In the present study, glucose and fructose were both ingested at rates of 1.2 g/min and therefore the higher exogenous CHO oxidation rates in the GLU+FRUC trial could be fully attributed to the oxidation of ingested fructose, which supports our previous findings (Jentjens *et al.* 2004b). Of note, previous studies have shown that when fructose is ingested alone, average oxidation rates vary between 0.32 and 0.44 g/min for exercise durations up to 180 min (Massicotte *et al.* 1986, 1989, 1990; Jandrain *et al.* 1993; Adopo *et al.* 1994). Unfortunately most studies did not report 'peak' exogenous CHO oxidation rates. In some studies it was found that the oxidation rate of ingested fructose reached peak values of approximately 0.50–0.64 g/min at the end of 120–180 min of cycling exercise (Massicotte *et al.* 1986, 1990; Jandrain *et al.* 1993). During the 120–150 min exercise period in the present study, the difference in the rate of exogenous CHO oxidation between GLU and GLU+FRUC was approximately 0.6 g/min. If it is assumed that this oxidation rate represents the oxidation rate of ingested fructose then these results suggest that the ingested fructose and glucose in the GLU+FRUC trial were both oxidized at peak rates (approximately 0.6 and approximately 1.1 g/min, respectively). Therefore, the exogenous CHO oxidation rate observed in the GLU+FRUC trial (approximately 1.75 g/min) could be the highest oxidation rate that is physiologically possible when multiple transportable CHO (i.e. glucose+fructose) are ingested orally.

It should be noted that in order to reach very high rates of exogenous CHO oxidation, it may be important that both glucose and fructose transporters (SGLT1 and GLUT-5, respectively) are saturated. As mentioned earlier, intestinal glucose transporters (SGLT1) may become saturated when glucose or glucose polymers are ingested at a rate of >1.2 g/min (Jentjens *et al.* 2004b). Therefore, in the present study and in our previous studies (Jentjens *et al.* 2004a,b,c, 2005), glucose was ingested at a rate of at least 1.2 g/min. The absorption rate of fructose is, however, much lower than that of glucose or sucrose (Ravich *et al.* 1983;

Riby *et al.* 1993; Rumessen & Gudmand-Hoyer, 1986) and thus ingestion of large amounts of fructose should be avoided as the unabsorbed fructose might accumulate in the GI tract which might increase the risk of GI discomfort (Murray *et al.* 1989; Fujisawa *et al.* 1993). In our previous study, the maximum amount of fructose theoretically available for absorption was 0.9 g/min (0.6 g/min fructose and 0.3 g/min fructose released from sucrose hydrolysis) and glucose was ingested at a rate of 1.2 g/min (Jentjens *et al.* 2004a). In the present study, a higher fructose intake rate (1.2 v. 0.9 g/min) did not lead to higher exogenous CHO oxidation rates (1.75 (SE 0.11) v. 1.70 (SE 0.07)). Furthermore, previous studies have shown that oxidation rates of ingested fructose (alone) are highest when fructose is ingested at a rate of approximately 0.8 g/min (Massicotte *et al.* 1986, 1990). Although speculative, GLUT-5 transporters may become 'saturated' at a fructose ingestion rate of 0.8–0.9 g/min and therefore no increase in exogenous fructose oxidation is observed when fructose is ingested at higher rates.

In conclusion, combined ingestion of large amounts of glucose and fructose during 150 min of cycling exercise resulted in peak exogenous CHO oxidation rates of approximately 1.75 g/min and resulted in approximately 50% higher exogenous CHO oxidation rates compared with the ingestion of glucose alone. The present findings are in agreement with our earlier findings and suggest that intestinal CHO absorption could be a rate-limiting factor for exogenous CHO oxidation.

### Acknowledgements

The authors would like to thank Cerestar (Manchester, UK) for donating glucose monohydrate and A.E. Staley Manufacturing Company (USA) for donating fructose. This study was supported by a grant of GlaxoSmithKline Consumer Healthcare, Brentford, UK.

### References

- Adopo E, Peronnet F, Massicotte D, Brisson GR & Hillaire-Marcel C (1994) Respective oxidation of exogenous glucose and fructose given in the same drink during exercise. *J Appl Physiol* **76**, 1014–1019.
- Bjorkman O, Crump M & Phillips RW (1984) Intestinal metabolism of orally administered glucose and fructose in Yucatan miniature swine. *J Nutr* **114**, 1413–1420.
- Bjorkman O, Eriksson LS, Nyberg B & Wahren J (1990) Gut exchange of glucose and lactate in basal state and after oral glucose ingestion in postoperative patients. *Diabetes* **39**, 747–751.
- Borg G (1982) Ratings of perceived exertion and heart rates during short-term cycle exercise and their use in a new cycling strength test. *Int J Sports Med* **3**, 153–158.
- Bosch AN, Dennis SC & Noakes TD (1994) Influence of carbohydrate ingestion on fuel substrate turnover and oxidation during prolonged exercise. *J Appl Physiol* **76**, 2364–2372.
- Burant CF, Takeda J, Brot-Laroche E, Bell GL & Davidson NO (1992) Fructose transporter in human spermatozoa and small intestine is GLUT5. *J Biol Chem* **267**, 14523–14526.
- Coggan AR & Coyle EF (1987) Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. *J Appl Physiol* **63**, 2388–2395.
- Corpe CP, Burant CF & Hoekstra JH (1999) Intestinal fructose absorption: clinical and molecular aspects. *J Pediatr Gastroenterol Nutr* **28**, 364–374.

- Coyle EF, Coggan AR, Hemmert MK & Ivy JL (1986) Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. *J Appl Physiol* **61**, 165–172.
- Craig H (1957) Isotopic standards for carbon and oxygen and correction factors. *Geochim Cosmochim Acta* **12**, 133–149.
- Davidson RE & Leese HJ (1977) Sucrose absorption by the rat small intestine in vivo and in vitro. *J Physiol* **267**, 237–248.
- Ferraris RP & Diamond J (1997) Regulation of intestinal sugar transport. *Physiol Rev* **77**, 257–302.
- Fine KD, Santa Ana CA, Porter JL & Fordtran JS (1994) Mechanism by which glucose stimulates the passive absorption of small solutes by the human jejunum in vivo. *Gastroenterology* **107**, 389–395.
- Frayn KN (1983) Calculation of substrate oxidation rates in vivo from gaseous exchange. *J Appl Physiol* **55**, 628–634.
- Fujisawa T, Mulligan K, Wada L, Schumacher L, Riby J & Kretchmer N (1993) The effect of exercise on fructose absorption. *Am J Clin Nutr* **58**, 75–79.
- Fujisawa T, Riby J & Kretchmer N (1991) Intestinal absorption of fructose in the rat. *Gastroenterology* **101**, 360–367.
- Gray GM & Ingelfinger FJ (1966) Intestinal absorption of sucrose in man: interrelation of hydrolysis and monosaccharide product absorption. *J Clin Invest* **45**, 388–398.
- Hanson PJ & Parsons DS (1976) The utilization of glucose and production of lactate by in vitro preparations of rat small intestine: effects of vascular perfusion. *J Physiol* **255**, 775–795.
- Hawley JA, Dennis SC & Noakes TD (1992) Oxidation of carbohydrate ingested during prolonged endurance exercise. *Sports Med* **14**, 27–42.
- Hoekstra JH & van den Aker JH (1996) Facilitating effect of amino acids on fructose and sorbitol absorption in children. *J Pediatr Gastroenterol Nutr* **23**, 118–124.
- Holdsworth CD & Dawson AM (1964) The absorption of monosaccharides in man. *Clin Sci* **27**, 371–379.
- Holloway PA & Parsons DS (1984) Absorption and metabolism of fructose by rat jejunum. *Biochem J* **222**, 57–64.
- Jandrain BJ, Pallikaris N, Normand S, Pirnay F, Lacroix M, Mosora F, Pachiandi C, Gautier JF, Scheen AJ, Riou JP & Lefebvre PJ (1993) Fructose utilization during exercise in men: rapid conversion of ingested fructose to circulating glucose. *J Appl Physiol* **74**, 2146–2154.
- Jentjens RL, Achten J & Jeukendrup AE (2004a) High oxidation rates from combined carbohydrates ingested during exercise. *Med Sci Sports Exerc* **36**, 1551–1558.
- Jentjens RL, Moseley L, Waring RH, Harding LK & Jeukendrup AE (2004b) Oxidation of combined ingestion of glucose and fructose during exercise. *J Appl Physiol* **96**, 1277–1284.
- Jentjens RL, Venables MC & Jeukendrup AE (2004c) Oxidation of exogenous glucose, sucrose, and maltose during prolonged cycling exercise. *J Appl Physiol* **96**, 1285–1291.
- Jentjens RLPG, Shaw C, Birtles T, Waring RH, Harding LE & Jeukendrup AE (in press) Oxidation of combined ingestion of glucose and sucrose during exercise. *Metabolism*.
- Jeukendrup AE & Jentjens R (2000) Oxidation of carbohydrate feedings during prolonged exercise: current thoughts, guidelines and directions for future research. *Sports Med* **29**, 407–424.
- Jeukendrup AE, Vet-Joop K, Sturk A, Stegen JH, Senden J, Saris WHM & Wagenmakers AJM (2000) Relationship between gastro-intestinal complaints and endotoxaemia, cytokine release and the acute-phase reaction during and after a long-distance triathlon in highly trained men. *Clin Sci* **98**, 47–55.
- Jeukendrup AE, Wagenmakers AJM, Stegen JHCH, Gijsen AP, Brouns F & Saris WHM (1999) Carbohydrate ingestion can completely suppress endogenous glucose production during exercise. *Am J Physiol* **276**, E672–E683.
- Koivisto VA, Karonen SL & Nikkila EA (1981) Carbohydrate ingestion before exercise: comparison of glucose, fructose, and sweet placebo. *J Appl Physiol* **51**, 783–787.
- Kuipers H, Verstappen FTJ, Keizer HA, Geurten P & van Kranenburg G (1985) Variability of aerobic performance in the laboratory and its physiologic correlates. *Int J Sports Med* **6**, 197–201.
- Macdonald I, Keyser A & Pacy D (1978) Some effects, in man, of varying the load of glucose, sucrose, fructose, or sorbitol on various metabolites in blood. *Am J Clin Nutr* **31**, 1305–1311.
- Massicotte D, Peronnet F, Allah C, Hillaire-Marcel C, Ledoux M & Brisson G (1986) Metabolic response to [<sup>13</sup>C]glucose and [<sup>13</sup>C]fructose ingestion during exercise. *J Appl Physiol* **61**, 1180–1184.
- Massicotte D, Peronnet F, Brisson G, Bakkouch K & Hillaire-Marcel C (1989) Oxidation of a glucose polymer during exercise: comparison with glucose and fructose. *J Appl Physiol* **66**, 179–183.
- Massicotte D, Peronnet F, Brisson G, Boivin L & Hillaire-Marcel C (1990) Oxidation of exogenous carbohydrate during prolonged exercise in fed and fasted conditions. *Int J Sports Med* **11**, 253–258.
- Mosora F, Lefebvre P, Pirnay F, Lacroix M, Luyckx A & Duchesne J (1976) Quantitative evaluation of the oxidation of an exogenous glucose load using naturally labeled <sup>13</sup>C-glucose. *Metabolism* **25**, 1575–1582.
- Murray R, Paul GL, Seifert JG, Eddy DE & Halaby GA (1989) The effects of glucose, fructose, and sucrose ingestion during exercise. *Med Sci Sports Exerc* **21**, 275–282.
- Nicholls TJ, Leese HJ & Bronk JR (1983) Transport and metabolism of glucose by rat small intestine. *Biochem J* **212**, 183–187.
- Pallikarakis N, Sphiris N & Lefebvre P (1991) Influence of the bicarbonate pool and on the occurrence of <sup>13</sup>CO<sub>2</sub> in exhaled air. *Eur J Appl Physiol* **63**, 179–183.
- Porteous JW (1978) Glucose as a fuel for small intestine. *Biochem Soc Trans* **6**, 534–539.
- Ravich WJ, Bayless TM & Thomas M (1983) Fructose: incomplete intestinal absorption in humans. *Gastroenterology* **84**, 26–29.
- Riby JE, Fujisawa T & Kretchmer N (1993) Fructose absorption. *Am J Clin Nutr* **58**, 748S–753S.
- Robert JJ, Koziat J, Chauvet D, Darmaun D, Desjeux JF & Young VR (1987) Use of <sup>13</sup>C-labeled glucose for estimating glucose oxidation: some design considerations. *J Appl Physiol* **63**, 1725–1732.
- Rumessen JJ & Gudmand-Hoyer E (1986) Absorption capacity of fructose in healthy adults. Comparison with sucrose and its constituent monosaccharides. *Gut* **27**, 1161–1168.
- Sandle GI, Lobley RW, Warwick R & Holmes R (1983) Monosaccharide absorption and water secretion during disaccharide perfusion of the human jejunum. *Digestion* **26**, 53–60.
- Shi X, Schedl HP, Summers RM, Lambert GP, Chang RT, Xia T & Gisolfi CV (1997) Fructose transport mechanisms in humans. *Gastroenterology* **113**, 1171–1179.
- Shi X, Summers RW, Schedl HP, Flanagan SW, Chang R & Gisolfi CV (1995) Effects of carbohydrate type and concentration and solution osmolality on water absorption. *Med Sci Sports* **27**, 1607–1615.
- Wagenmakers AJM, Brouns F, Saris WHM & Halliday D (1993) Oxidation rates of orally ingested carbohydrates during prolonged exercise in man. *J Appl Physiol* **75**, 2774–2780.