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ORIGINAL ARTICLE

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Effects of pre-exercise ingestion of trehalose, galactose and glucose on subsequent metabolism and cycling performance

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Abstract The glycaemic and insulinaemic responses to different carbohydrates vary and these have been suggested to affect performance. The purpose of the present study was to determine the effects of pre-exercise ingestion of glucose (GLU), galactose (GAL) and trehalose (TRE) on metabolic responses at rest and during exercise and on subsequent time-trial (TT) performance. Eight well-trained male cyclists completed three exercise trials separated by at least 3 days. At 45 min before the start of exercise subjects consumed 500 ml of a beverage containing 75 g of either glucose, galactose or trehalose. The exercise trials consisted of 20 min of submaximal steady-state exercise (SS) at 65% of maximal power output immediately followed by a [mean (SEM)] 702 (25) kJ TT. Plasma glucose concentration 15 min postprandial was significantly higher in GLU compared to GAL and TRE ($P < 0.05$). This was accompanied by a more than twofold greater rise in plasma insulin concentration in GLU compared to GAL and TRE (118% and 145%, respectively). During SS exercise four subjects in GLU and one subject in TRE developed a rebound hypoglycaemia (plasma glucose concentration less than 3.5 mmol·l^{-1}). No differences were observed in TT performance between the three trials. Pre-exercise ingestion of trehalose and galactose resulted in lower plasma glucose and insulin responses prior to exercise and reduced the prevalence of rebound hypoglycaemia. Despite the attenuated insulin and glucose responses at rest and during exercise following pre-exercise ingestion of galactose and trehalose, there was no difference in TT performance compared with pre-exercise ingestion of glucose.

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Introduction

This study was one of a series designed to investigate systematically factors involved in pre-exercise carbohydrate (CHO) metabolism and subsequent time trial (TT) performance. When glucose, a high glycaemic index (GI) CHO, is consumed in the hour before exercise it results in hyperglycaemia followed by a large increase in plasma insulin concentration (Costill et al. 1977; Koivisto et al. 1981). At the onset of exercise, this hyperinsulinaemia may subsequently be followed by hypoglycemia (Costill et al. 1977; Foster et al. 1979; Koivisto et al. 1981), decreased lipolysis and free fatty acid availability (Costill et al. 1977; Foster et al. 1979), and increased muscle glycogen utilization (Costill et al. 1977; Hargreaves et al. 1985) and CHO oxidation (Costill et al. 1977; Febbraio and Stewart 1996). These metabolic disturbances may be attenuated by choosing pre-exercise sources of CHO intake that produce minimal glycaemic and insulinaemic responses (Guezennec et al. 1989; Hargreaves et al. 1985, 1987).

Ingestion of fructose, which has a low GI, has been shown to result in modest hyperinsulinaemia and little or no decline of blood glucose concentrations at the onset of exercise (Hargreaves et al. 1985, 1987; Koivisto et al. 1981). Therefore, fructose and possibly other CHO of low or moderate GI may be the preferred type of CHO for consumption prior to exercise. However, several studies that have compared fructose ingestion with that of glucose have found no difference in exercise performance (Hargreaves et al. 1987; McMurray et al. 1983; Scott van Zant and Lemon 1997). Fructose is absorbed more slowly from the gut than glucose (Holdsworth and Dawson 1964) and is primarily metabolized by the liver. Because of its slower absorption rate from the intestine, complaints of abdominal

discomfort and other symptoms generally associated with malabsorption are often observed when exercise follows fructose ingestion (Fujisawa et al. 1993; Murray et al. 1989).

Two alternative CHO that may not have the negative side-effects sometimes observed with pre-exercise glucose and fructose ingestion are galactose (GI of 20) and trehalose (GI of 67). Like glucose, galactose is actively absorbed by the small intestine. In the liver, galactose is converted to glucose and subsequently stored as glycogen or immediately released into the circulation. However, trehalose is a disaccharide consisting of two glucose molecules linked by an α-1,1 glycosidic bond. It is hydrolyzed by a specific intestinal brush-border disaccharidase (trehalase) to two glucose molecules, which are subsequently rapidly absorbed into the blood circulation. An important feature of trehalose is that it is not broken down to glucose in the oral cavity and hence the potential problem of tooth decay, associated with regular consumption of glucose or glucose polymer, would be avoided. Both galactose and trehalose have been well tolerated during prolonged exercise trials in previous studies (Leijssen et al. 1995; M. Gleeson and N. Bishop 2000, unpublished observations) and may therefore be more suitable as CHO sources compared to fructose when ingested prior to exercise. Furthermore, because both galactose and trehalose have a lower GI than glucose, pre-exercise ingestion of these CHO may not cause a rebound hypoglycaemia (plasma glucose concentration less than 3.5 mmol·l^{-1}) which is often associated with decreased performance (Foster et al. 1979; Keller and Schwarzkopf 1984).

To our knowledge, no studies have investigated the effect of trehalose and galactose on TT performance, when ingested before exercise. The purpose of the present study, therefore, was to determine the effects of pre-exercise ingestion of glucose, galactose and trehalose on glucose and insulin responses at rest and during exercise and on subsequent TT performance.

Methods

Subjects

Eight endurance-trained cyclists or triathletes were recruited to take part in this study. Their characteristics are presented in Table 1. Prior to participation, each of the subjects was fully informed of the purpose and risks associated with the procedures and written informed consent was obtained. All subjects were healthy as assessed from the responses to 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.

Table 1 Mean (SEM) subject characteristics ($n = 8$). $\dot{V}O_{2max}$ Maximal oxygen uptake expressed per kilogram body mass, $\dot{W}_{max}$ maximal exercise intensity, HR_{max} maximal heart rate

Age (years)	28.1 (3.0)
Height (cm)	180.9 (3.2)
Body mass (kg)	72.7 (1.9)
Body fat (%)	13.5 (1.3)
$\dot{V}O_{2max}$ (ml·kg^{-1}·min^{-1})	61.5 (1.6)
$\dot{W}_{max}$ (W)	366 (13)
HR_{max} (beats·min^{-1})	192 (2)

General design

All the studies in this series were based on the same protocol. A brief outline is given below with attention being given to deviations from the basic protocol. For a detailed description of the basic protocol the reader is referred to Jentjens et al. (2002). Each subject reported to the laboratory on two different occasions before the start of the actual trials. On the first visit, the individual maximal exercise intensity ($\dot{W}_{max}$), maximal oxygen uptake ($\dot{V}O_{2max}$), body mass, height and body fat were determined as previously described (Jentjens et al. 2002). On a separate day, subjects performed a TT, based on Jeukendrup et al. (1996) to become familiar with the procedure and to ensure that they could complete the required exercise. On three subsequent occasions separated by periods of 7 days, each subject completed an exercise performance trial consisting of 20 min of steady-state (SS) cycling followed by a simulated TT. At 45 min prior to the start of exercise, subjects consumed one of three experimental drinks, GLU, GAL, or TRE, containing either glucose, galactose or trehalose. The order in which the drinks were allotted was counterbalanced in six of the eight subjects; the remaining two subjects were randomly assigned a drink sequence that was different one from the other. During exercise, blood samples were collected for determination of plasma glucose, insulin and lactate concentrations and gas exchange measurements were performed.

The trials

The subjects were asked to record their food intakes and activity patterns during the day prior to the first trial and were then instructed to repeat the diet and activitypattern during the day before all subsequent trials. In addition, the subjects were asked to avoid vigorous exercise and to abstain from drinking alcohol and using tobacco during the 24 h prior to each test. For a given subject, all trials were conducted at the same time of the day to avoid any influence of variations in his circadian rhythm.

The subjects reported to the Human Performance Laboratory in the morning (between 7–9 a.m.) after an overnight fast (10–12 h). After the collection of a 5 ml fasting blood sample (referred to as $t = -45$ min) the subjects ingested a solution containing either 75 g of glucose (D-Glucose monohydrate, Meritose 200, Amylum UK, UK), galactose (D-Galactose, G-Push Sport Limited, Pontefract, UK) or trehalose (D-Trehalose dihydrate, British Sugar, Norwich, UK) made up with distilled water to a volume of 400 ml. In the present study, we aimed to give the same amount of CHO in each trial. Because glucose monohydrate contains one extra H_2O molecule, 82.5 g of glucose monohydrate was given in GLU. However, because of an error in the correction 78.8 g of trehalose dihydrate was given in TRE instead of 82.9 g. In an attempt to make the drinks similar in taste, 100 ml of an energy-free liquid orange flavour (Lucozade placebo drink, SmithKline Beecham, UK) was added to all drinks, which brought the total volume to be consumed to 500 ml. The osmolality of the drinks was measured using a freezing point osmometer (Micro Osmometer, 3300, Vitech Scientific Ltd, West Sussex, UK) and was 457, 811 and 852 mosmol·kg^{-1} for TRE, GAL and GLU, respectively. It should be noted that 12 mosmol·kg^{-1} of each drink was attributable to the added orange flavour. On a separate day, prior to commencement of the first experimental trial, the subjects were given a test drink containing 5 g of trehalose. Ingestion of a small amount of this sugar would be enough to detect any trehalose intolerance (i.e. abdominal cramps, bloating, diarrhoea), which would immediately have excluded the subject from further participation in the study. None of the subjects reported gastrointestinal discomfort following the test drink.

After consumption of the CHO drinks subjects rested quietly in the laboratory for 45 min. During this period a 5 ml blood sample was obtained 15 and 30 min postprandial ($t=-30$ and $t=-15$). Exactly 45 min after consumption of the CHO drink subjects mounted an electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) and began the exercise protocol as described by Jentjens et al. (2002). Briefly, this consisted of 20 min of submaximal SS exercise at 65% $\dot{W}_{max}$ [76 (1)% $\dot{V}O_{2max}$], immediately followed by a TT equal to approximately 40 min of exercise at 80% $\dot{W}_{max}$. In the present study the total amount of work that had to be completed during TT was [mean SEM] 702 (25) kJ. Blood samples were collected as previously described (Jentjens et al. 2002).

Oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$) and respiratory exchange ratio (R) were measured throughout the SS exercise using an online automated gas analysis system (Oxycon Alpha, Jaeger, Wuerzberg, Germany) and averages were taken of each 5 min period. The heart rate (HR) was recorded in 30 s intervals throughout SS using a radiotelemetry HR monitor (Polar Vantage NV, Kempele, Finland) and later averaged for 5 min periods.

All exercise tests were performed under normal and standard environmental conditions [18 (2)°C dry bulb temperature and 60 (2)% relative humidity]. During the exercise trials (SS and TT) subjects were cooled with floor-standing fans to minimize thermal stress, and during the TT water was available ad libitum.

Questionnaires

Subjects were asked to fill out a questionnaire prior to ingestion of the experimental drink, before the start of SS and TT and immediately after TT. The questionnaire contained questions regarding the presence of gastrointestinal problems at that moment as follows – stomach problems, gastrointestinal cramping, bloated feeling, diarrhoea, nausea, dizziness, headache, belching, vomiting, urge to urinate/defaecate. The items were scored on a 10-point scale (1 = not at all, 10 = very, very much).

Analysis

Plasma glucose, insulin and lactate concentrations were determined as previously described (Jentjens et al. 2002). From the $\dot{V}CO_2$, (litres per minute) and $\dot{V}O_2$, rates of total carbohydrate (CHO_{tot}) and fat (FAT_{tot}) oxidation (grams per minute) were calculated using the stoichiometric equations of Frayn (1983), making the assumption that the rate of nitrogen excretion during exercise was negligible.

Statistics

The data from the three trials were compared by using a two-factor (time and treatment) ANOVA for repeated measurements. Greenhouse-Geisser ϵ-correction was used to adjust the significance level of the test statistics for violation of the assumed sphericity. A Tukey post-hoc test was applied to locate differences when ANOVA revealed a significant interaction. Data was evaluated using an SPSS for Windows version 10.0 software package (Chicago, USA). Questionnaire data are reported as medians (ranges), all other data are reported as means (SEM). Statistical significance was set at $P<0.05$.

Results

There was no order effect on any of the measured parameters ($P>0.05$). Body masses before the start of exercise, body mass losses and fluid intakes during exercise were no different between the three experimental trials [on average 73.1 (1.1) kg, 1.0 (0.1) kg and 0.4 (0.1) l, respectively].

Plasma glucose and insulin responses

Plasma glucose concentrations at rest and during exercise are shown in Fig. 1. Fasting plasma glucose concentrations were similar in the three trials [on average; 4.7 (0.1) mmol·l^{-1}]. After ingestion of GLU and TRE there was a significant rise ($P<0.05$) in plasma glucose concentration, reaching peak values of 6.6 (0.1) and 5.7 (0.3) mmol·l^{-1} at $t=-30$ and at $t=-15$ min, respectively. The pre-exercise ingestion of GAL resulted in only a modest increase in plasma glucose at rest [5.3 (0.1) mmol·l^{-1} at $t=-30$]. The plasma glucose concentration in GLU was significantly higher ($P<0.05$) compared with GAL and TRE at $t=-30$ min. Furthermore, the plasma glucose concentration at $t=-15$ min was significantly higher in GLU compared with GAL ($P<0.05$) and in TRE compared with GAL ($P<0.05$). During the first 10 min of SS exercise plasma glucose concentrations in all trials dropped to concentrations lower than fasting plasma glucose values (3.9–4.1 mmol·l^{-1}) ($P<0.05$). After the initial fall in plasma glucose concentration, plasma glucose gradually rose until the end of the TT. Plasma glucose concentrations following GAL were significantly higher ($P<0.05$) during the final 10 min of SS exercise compared with GLU and TRE. No differences in plasma glucose concentrations were observed between the three CHO trials at any time during TT.

Plasma insulin concentrations at rest and during exercise are shown in Fig. 2. Fasting plasma insulin

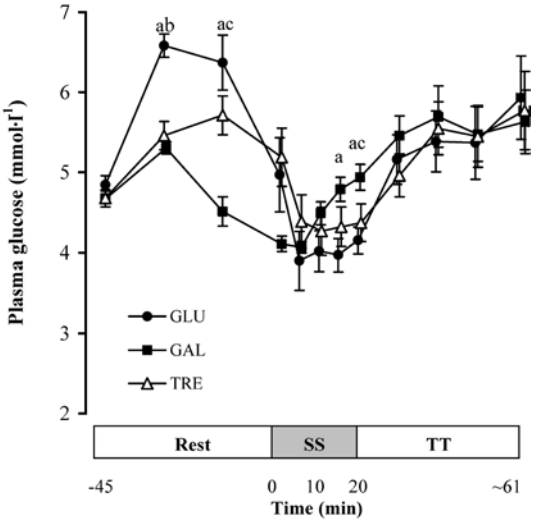

Fig. 1 Mean (SEM) plasma glucose concentrations at rest, during submaximal steady-state exercise (*SS*) and time trial (*TT*) performance after ingestion of 75 g of glucose (*GLU*), galactose (*GAL*) or trehalose (*TRE*) 45 min prior to SS. [a]Significant difference between GLU and GAL ($P<0.05$), [b]significant difference between GLU and TRE ($P<0.05$), [c]significant difference between GAL and TRE ($P<0.05$). $n=8$

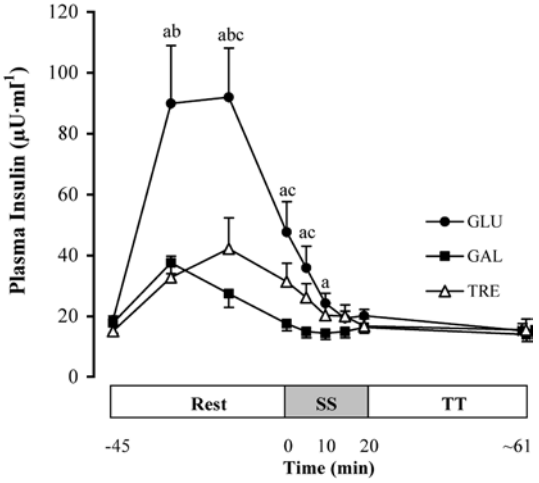

Fig. 2 Mean (SEM) plasma insulin concentrations at rest, during submaximal steady state exercise (*SS*) and time trial (*TT*) performance after ingestion of 75 g of glucose (GLU), galactose (GAL) or trehalose (TRE) 45 min prior to SS. [a]Significant difference between GLU and GAL ($P < 0.05$), [b]significant difference between GLU and TRE ($P < 0.05$), [c]significant difference between GAL and TRE ($P < 0.05$). $n = 8$

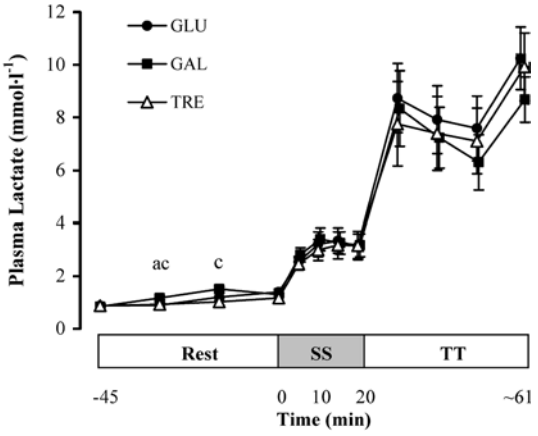

Fig. 3 Mean (SEM) plasma lactate concentrations at rest, during submaximal steady state exercise (*SS*) and time trial (*TT*) performance after ingestion of 75 g of glucose (GLU), galactose (GAL) or trehalose (TRE) 45 min prior to SS. [a]Significant difference between GLU and GAL ($P < 0.05$), [c]significant difference between GAL and TRE ($P < 0.05$). $n = 8$

concentrations were similar in the three trials. In GLU, plasma insulin concentrations rose rapidly during the first 15 min of rest ($P < 0.01$) and remained high for the next 15 min. Thereafter, plasma insulin concentration in GLU declined to fasting levels at 10 min into SS exercise. The rise in pre-exercise plasma insulin concentration in GLU was more than twofold higher compared with GAL and TRE (118% and 145%, respectively). After the peaks in plasma insulin concentrations in GAL and TRE they gradually declined to fasting levels at 0 and 5 min into SS exercise, respectively. Plasma insulin concentrations were significantly higher in GLU compared to GAL from $t = -30$ to $t = 10$ ($P < 0.01$) and compared to TRE from $t = -30$ to $t = -15$ ($P < 0.01$). In addition, plasma insulin concentrations were significantly higher in TRE compared to GAL from $t = -15$ to $t = 5$ ($P < 0.05$). From 15 min of SS exercise onwards there were no differences in insulin between the three trials.

Plasma lactate concentration

The fasting plasma lactate concentrations for the three different CHO drinks averaged between 0.8 and 0.9 mmol·l^{-1} (Fig. 3). During the 45 min rest period plasma lactate concentrations remained steady in GLU and TRE, while plasma lactate in GAL rose slightly [from 0.9 (0.1) mmol·l^{-1} at rest to 1.5 (0.1) mmol·l^{-1} at $t = -15$; $P > 0.05$]. Pre-exercise plasma lactate concentrations in GAL were significantly higher compared to GLU at $t = -30$ min and compared with TRE at $t = -30$ and $t = -15$ min ($P < 0.05$). There were no differences in plasma lactate concentrations during exercise between the three trials. Plasma lactate concentrations increased ($P < 0.05$) from 1.3 (0.1) mmol·l^{-1} before the start of exercise ($t = 0$) to an average value of 3.1 (0.1) mmol·l^{-1} during SS exercise. Plasma lactate concentrations during TT were significantly higher ($P < 0.01$) compared to SS exercise and rest [8.1 (0.3) > 3.1 (0.1) > 1.3 (0.1) mmol·l^{-1}].

TT performance

No significant differences in time to complete the TT and in the accompanying power outputs were observed between the three trials (Table 2). In addition, no difference was found in mean HR and percentage maximal HR during the TT. Two subjects reported gastrointestinal problems in TRE and GAL before and after the TT (urge to vomit, stomach problems, nausea, bloated feeling).

$\dot{V}O_2$, *R*, CHO_{tot}, FAT_{tot}, HR and rating of perceived exertion

Respiratory data, CHO_{tot} and FAT_{tot} are shown in Table 3. No significant differences were observed between the three trials for $\dot{V}O_2$, CHO_{tot} and FAT_{tot}.

There was no significant interaction between drink and time for *R*. However, there was a main effect of time ($P < 0.05$). The *R* gradually declined throughout the 20 min of SS exercise in all trials ($P < 0.05$).

There was no significant interaction between drink and time for HR. However, there was a main effect of time ($P < 0.001$). The HR significantly increased during SS exercise in all three CHO trials. The mean HR values during SS for GLU, GAL and TRE were 155 (2), 154 (3), and 152 (3) beats·min^{-1}, respectively.

No significant differences were observed between trials for the rating of perceived exertion (RPE, Borg

Table 2 Mean (SEM) results of the time trial (*TT*) after 20 min of submaximal exercise and prior ingestion of a 75 g glucose (*GLU*), galactose (*GAL*) or trehalose (*TRE*) drink. Values shown are the time to complete a preset amount of work (Time TT), average exercise intensity, average exercise intensity expressed as a percentage of maximal exercise intensity (% $\dot{W}_{max}$), average heart rate (*HR*) and average HR expressed as a percentage of maximal HR (%HR_{max}). $n = 8$

Drink	Time TT (min:s)	Average exercise intensity (W)	% $\dot{W}_{max}$ (%)	Average HR (beats·min^{-1})	%HR_{max} (%)
GLU	41:05 (1:03)	287 (15)	78 (2)	178 (2)	93 (1)
GAL	42:04 (0:51)	280 (14)	76 (2)	175 (2)	91 (1)
TRE	41:57 (1:19)	282 (16)	77 (2)	176 (2)	92 (1)

Table 3 Mean (SEM) oxygen uptake ($\dot{V}O_2$), total carbohydrate oxidation (CHO_{tot}), total fat oxidation (FAT_{tot}) and respiratory exchange ratio (*R*) during cycling exercise at 65% maximal exercise intensity (65% $\dot{W}_{max}$) after ingestion of a beverage containing either 75 g of glucose (*GLU*), galactose (*GAL*) or trehalose (*TRE*) 45 min before the start of exercise. $n = 8$. However, the CHO_{tot} and FAT_{tot} results are reported for six subjects only

	Time (min)			
	0–5	5–10	10–15	15–20
$\dot{V}O_2$ (l·min^{-1})				
GLU	3.42 (0.14)	3.43 (0.15)	3.45 (0.15)	3.44 (0.16)
GAL	3.42 (0.13)	3.44 (0.14)	3.48 (0.16)	3.46 (0.14)
TRE	3.36 (0.12)	3.38 (0.13)	3.38 (0.15)	3.41 (0.16)
R				
GLU*	1.01 (0.01)	0.99 (0.01)	0.98 (0.01)	0.98 (0.01)
GAL*	0.99 (0.02)	0.97 (0.01)	0.97 (0.01)	0.96 (0.01)
TRE*	1.01 (0.01)	0.99 (0.01)	0.98 (0.01)	0.97 (0.01)
CHO_{tot} (g·min^{-1})				
GLU	–	–	4.38 (0.30)	4.32 (0.31)
GAL	–	–	4.09 (0.19)	3.99 (0.15)
TRE	–	–	4.09 (0.29)	4.07 (0.35)
FAT_{tot} (g·min^{-1})				
GLU	–	–	0.12 (0.03)	0.15 (0.04)
GAL	–	–	0.26 (0.06)	0.29 (0.06)
TRE	–	–	0.20 (0.04)	0.22 (0.04)

*Significant main effect of time ($P < 0.05$)

1982) for the body overall or RPE for the legs alone. Furthermore, RPE overall and RPE legs did not change with time during SS exercise. The medians (ranges) for RPE overall and RPE legs during SS were 11.0 (9.0–13.0) and 12.0 (9.0–17.0) for GLU, 11.0 (7.0–13.0) and 11.0 (7.0–13.0) for GAL, 11.0 (7.0–13.0) and 12.0 (7.0–15.0) for TRE, respectively.

Discussion

An intake of glucose during the hour prior to exercise can cause rebound hypoglycaemia 15–30 min after the onset of exercise (Costill et al. 1977; Foster et al. 1979; Koivisto et al. 1981). The extent to which hypoglycaemia can affect performance is largely unknown. Both galactose and trehalose have a lower GI than glucose and may not therefore cause a rebound hypoglycaemia when ingested before exercise. The present study, therefore, examined the effects of pre-exercise ingestion of glucose, trehalose and galactose on metabolic responses in rest and exercise and on TT performance.

The most important finding of the present study was that pre-exercise ingestion of different types of CHO, varying in GI between 20 and 100, did not affect TT performance following SS exercise. Although it was hypothesized that a pre-exercise intake of CHO with a lower GI than glucose would benefit exercise performance, the present data show that there was no performance advantage following ingestion of galactose (GI of 20) and trehalose (GI of 67) compared with glucose. In fact, the power output in the GLU trial was 1.8% and 2.5% higher compared to the GAL and TRE trials respectively, although this failed to reach significance ($P = 0.14$ and 0.09, respectively). This may reject the hypothesis that CHO with a low GI would be the preferred type of CHO in the hour before exercise in order to optimize performance. In addition, two subjects reported severe gastrointestinal problems in TRE (urge to vomit, stomach problems, nausea, bloated feeling) and one of these subjects made almost similar complaints in GAL. Although this may have contributed to a decreased performance in TRE and GAL, the observed gastrointestinal complaints could not be completely attributed to the type of CHO ingested since some of the gastrointestinal problems reported during exercise were already present in a mild form before ingestion of the CHO drink.

The present results are in close agreement with studies that examined the effect of pre-exercise intakes of glucose and fructose on exercise performance. Previous studies could not find statistical differences in performance when comparing the ingestion of fructose and glucose (Décombaz et al. 1985; Hargreaves et al. 1987;

McMurray et al. 1983; Scott van Zant and Lemon 1997; Ventura et al. 1994) and if there was a trend for a better performance in one trial above the other than this would be for glucose (Hargreaves et al. 1987; Scott van Zant and Lemon 1997; Ventura et al. 1994). In addition, recently, Mitchell et al. (2000) have investigated the effects of pre-exercise CHO ingestion on metabolic responses and 10 km performance in the heat, when using CHO beverages containing either fructose, glucose, or a sucrose/glucose mixture. Although the ingestion of the various CHO compositions produced different pre-exercise glycaemic and insulinaemic responses, there were no differences in exercise performance. The findings of Mitchell et al. (2000) support the results of the present study, in which no significant difference in performance was found between the three CHO drinks.

After ingestion of the drink in GAL, plasma glucose concentration showed a small but significant rise which was accompanied by a small increase in plasma insulin concentration. After a peak glucose value of 5.3 (0.1) mmol·l^{-1}, glucose concentrations in GAL dropped and remained steady around 4 mmol·l^{-1} for the entire duration of SS. The observed glucose response in the present study is in agreement with other studies, in which ingestion of relatively large amounts of galactose (25–50 g) resulted in plasma glucose increments of only 1 mmol·l^{-1} or less in healthy men (Ercan et al. 1993; Gannon et al. 2001; Stenstam 1946; Williams et al. 1983) and type 2 diabetics (Ercan et al. 1993). The increase in blood glucose concentration following galactose ingestion does not appear to be related to the quantity of galactose ingested (Stenstam 1946). The liver is the major site of galactose uptake and metabolism in humans and practically all circulating galactose is removed by the liver (Williams 1986). Possibly a large part of the galactose is converted to glucose and temporarily stored as glycogen in the liver, which might explain the lower plasma glucose concentrations at some times at rest in GAL compared with GLU and TRE. An interesting finding of the present study was that plasma glucose concentrations in GAL were higher during the final 10 min of SS compared with the other two CHO trials. However, a physiological mechanism for this finding is not known and it did not seem to have affected subsequent performance.

The plasma glucose and insulin responses at rest in TRE followed a similar pattern as in GLU with the only difference that, at rest, the glucose and insulin concentrations in TRE were on average 10% and 50% lower, respectively, compared with GLU. However, during exercise no differences were observed in plasma glucose and insulin concentrations between GLU and TRE. The lack of a difference in plasma glucose concentration between TRE and GLU during SS exercise may be partly explained by the absence of a substantial rebound hypoglycaemia in GLU. In several other studies hypoglycaemia was observed when glucose was ingested in the hour before exercise (Costill et al. 1977; el-Sayed et al. 1997; Koivisto et al. 1981; Mitchell et al. 2000; Scott van Zant and Lemon 1997). Although a significant fall in plasma glucose concentration was observed in GLU, the lowest mean plasma glucose concentration during exercise was 3.9 (0.3) mmol·l^{-1} and did not reach a glucose concentration that could be defined as hypoglycaemia (Jentjens et al. 2002). However, hypoglycaemia in GLU did occur in four subjects of whom two reached nadirs in plasma glucose concentration of 2.6 and 2.7 mmol·l^{-1}, respectively. Only one subject in TRE reached a plasma glucose concentration of less than 3.5 mmol·l^{-1} while none of the subjects in GAL had hypoglycaemic plasma glucose concentrations. The present data indicate that the prevalence of rebound hypoglycaemia in some subjects can be substantially reduced when CHO with a low to moderate GI (20–70) is ingested prior to exercise compared with the pre-exercise ingestion of CHO with a high GI. This supports the findings of previous studies which showed that pre-exercise ingestion of fructose resulted in more steady blood glucose concentrations than pre-exercise ingestion of glucose and did not cause a rebound hypoglycaemia during exercise (Hargreaves et al. 1987; Koivisto et al. 1981; Scott van Zant and Lemon 1997). Despite the fact that rebound hypoglycaemia was observed during SS exercise in some subjects in the present study, this did not negatively affect their subsequent performance. In addition, no differences were observed in RPE for whole body and legs during SS exercise between the trials, indicating that subjects did not experience any detrimental effects of the hypoglycaemia. It is therefore likely that if subjects had directly commenced the TT, rebound hypoglycaemia during the early stage of the TT would also not have affected their performance.

In the present study no difference was found in R, CHO_{tot} and FAT_{tot} between the three CHO trials. This is in agreement with studies in which glucose and galactose or glucose and trehalose was provided during exercise (Leijssen et al. 1995; M. Gleeson and N. Bishop 2000, unpublished observations). In addition, studies that compared the ingestion of glucose and fructose before exercise did not find a difference in R and $\dot{V}O_2$ during exercise, which suggests a similar type of fuel selection (Décombaz et al. 1985; Hargreaves et al. 1985, 1987; Scott van Zant and Lemon 1997).

Plasma lactate concentrations in GAL were higher at rest compared with GLU and TRE. Previous studies have demonstrated an increase in plasma lactate concentration at rest after intravenous galactose injection (Royle et al. 1978) and oral galactose intake (Gannon et al. 2001). Similar data were also found after ingestion of fructose (Décombaz et al. 1985; Koivisto et al. 1981; Nuttall et al. 2000). The ingestion of galactose has also been shown to result in a rapid increase and higher R at rest compared with glucose (Macdonald 1984). Although the mechanism for this effect is not known, a higher R and elevated lactate concentrations at rest may suggest increased glycogenolysis. Leijsen et al. (1995) found higher plasma lactate concentrations during prolonged exercise at 65% $\dot{W}_{max}$, when galactose was

ingested during exercise compared with the ingestion of an equal amount of glucose. In the present study, however, there were no differences in plasma lactate concentrations during exercise between GAL and the other two CHO trials, possibly because of a higher exercise intensity in the present study.

In conclusion, the results of the present study showed that the ingestion of trehalose and galactose leads to lower glucose and insulin responses prior to exercise and reduces the prevalence of rebound hypoglycaemia compared with the ingestion of glucose. However, the type of CHO ingested before exercise, varying in GI between 20 and 100, did not affect TT performance. Furthermore, the present data further suggest that there are no detrimental effects of pre-exercise ingestion of glucose on performance when 75 g of glucose is ingested 45 min before the start of exercise.

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