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Comparison of exogenous glucose, fructose and galactose oxidation during exercise using ^{13}C-labelling

Yan Burelle[1], Marie-Catherine Lamoureux[1], François Péronnet[1], Denis Massicotte[2]* and Carole Lavoie[3]

[1]*Department of Kinesiology, University of Montreal, CP 6128 Centre Ville, Montreal, Quebec, H3C 3J7, Canada*
[2]*Department of Kinanthropology, University of Quebec at Montreal, Montreal, Quebec, H3C 3P8, Canada*
[3]*Department of Science of Physical Activity, University of Quebec at Trois-Rivières, Quebec, G9A 5H7, Canada*

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Six subjects exercised for 120 min on a cycle ergometer (65 (SE 3) % $\dot{V}_{O_{2max}}$) when ingesting a placebo or glucose, fructose or galactose (100 g in 1000 ml water) labelled with ^{13}C. The oxidation of energy substrates including exogenous hexoses was compared using indirect respiratory calorimetry and $^{13}CO_2$ production at the mouth. Total carbohydrate progressively decreased and total fat oxidation increased over the 120 min exercise period in the four experimental situations. During the 120 min of exercise, the amount of fructose oxidized (38·8 (SE 2·6) g; 9·0 (SE 0·6) % energy yield) was not significantly (approximately 4 %) lower than that of exogenous glucose (40·5 (SE 3·4) g; 9·2 (SE 0·8) % energy yield), while that of galactose (23·7 (SE 3·5) g; 5·5 (SE 0·9) % energy yield) was only 59 % and 61 % that of glucose and fructose, respectively. When compared with the placebo, the ingestion and oxidation of the three hexoses did not significantly modify fat oxidation or total carbohydrate oxidation, but it significantly reduced (9–13 %) endogenous carbohydrate oxidation. The present data indicate that fructose and exogenous glucose ingested during exercise could be oxidized at a similar rate, but that the oxidation rate of galactose was only approximately 60 % that of the exogenous glucose and fructose, presumably because of a preferential incorporation of galactose into liver glycogen (Leloir pathway). The reduction in endogenous carbohydrate oxidation was, however, similar with the three hexoses.

Stable isotope: Exogenous hexoses: Calorimetry

When they are ingested in large amounts (1·8–2·4 g/min) during prolonged moderate exercise, the oxidation of exogenous glucose or glucose polymers could exceed 1 g/min and could provide up to 30–40 % of the energy yield (Couture *et al.* 2002; Jentjens *et al.* 2004*a*,*b*; Wallis *et al.* 2005). When compared with that of exogenous glucose or starch, the oxidation of fructose has been shown to be 15 % higher (Décombaz *et al.* 1985), similar (Slama *et al.* 1989; Burelle *et al.* 1997) or approximately 20–25 % lower (Massicotte *et al.* 1986, 1989, 1990, 1994; Guézennec *et al.* 1989; Jandrain *et al.* 1993; Adopo *et al.* 1994), and both hexoses have been shown to reduce endogenous carbohydrate (CHO) oxidation (Massicotte *et al.* 1986, 1989, 1990, 1994; Wagenmakers *et al.* 1993; Jeukendrup *et al.* 1999; Couture *et al.* 2002; Jentjens *et al.* 2004*a*,*b*). As for galactose, Stellaard *et al.* (2000) have observed at rest a 35 % lower recovery of $^{13}CO_2$ following the ingestion of galactose compared with glucose (40 g). In the only study available during exercise, the oxidation rate of galactose was approximately 48 % that of exogenous glucose (Leijssen *et al.* 1995), but no control situation (i.e. exercise with ingestion of water) was included to ascertain the effect of galactose ingestion and oxidation on endogenous substrate utilization.

The purpose of the present study was to further compare the oxidation rate of glucose, fructose and galactose, and the associated changes in endogenous substrate oxidation, during prolonged exercise. Based on data from various studies concerning fructose oxidation (Décombaz *et al.* 1985; Massicotte *et al.* 1986, 1989, 1990, 1994; Guézennec *et al.* 1989; Jandrain *et al.* 1993; Burelle *et al.* 1997; Adopo *et al.* 1994), and from the studies by Leijssen *et al.* (1995) and Stellaard *et al.* (2000) concerning the oxidation of galactose, we hypothesized that the oxidation rate of fructose would be similar to or slightly lower than that of exogenous glucose, whereas that of galactose would be only about half that exogenous glucose. We also hypothesized that the reduction in endogenous CHO oxidation would be lower with galactose than with glucose or fructose.

Methods

Subjects

The experiments were conducted on six active healthy male subjects who gave their informed written consent to participate in the study, which was approved by the Institutional Board on the Use of Human Subjects in Research. Their mean age, body mass, height, fasting plasma glucose concentration and maximal oxygen uptake on a cycle ergometer were

Abbreviations: CHO, carbohydrate; $\dot{V}_{CO_2}$, carbon dioxide production; PDB-1, Pee Dee Bilemnitella.

* **Corresponding author:** Dr Denis Massicotte, fax +1 514 987 6616, email massicotte.denis@uqam.ca

21 (SE 1) years, 65 (SE 2) kg, 172 (SE 2) cm, 4·76 (SE 0·21) mmol/l and 4·40 (SE 0·06) l/min, respectively.

Experimental protocol

The $\dot{V}_{O_{2max}}$ and experimental workloads on a cycle ergometer (Ergomeca, La Bayette, France) were determined for each subject during a preliminary test session using open-circuit spirometry (1100 Medical Gas Analyser; Marquette Electronics, Milwaukee, Wisconsin, USA). Subsequently, at 1-week intervals beginning at 13.00 hours, all subjects performed four exercise sessions of 120 min duration at a workload corresponding to 65 (SE 3) % $\dot{V}_{O_{2max}}$ (218·3 (SE 10·6) W). The last evening meal (19.00 hours: 5450 kJ, approximate values 55 % CHO, 30 % fat, 15 % protein), the morning breakfast (07.30 hours: 3350 kJ, approximate values 60 % CHO, 30 % fat, 10 % protein) and a small snack ingested 2 h before the beginning of exercise (11.00 hours: 2100 kJ, approximate values 50 % CHO, 35 % fat, 15 % protein) were standardized.

In addition, in order to keep a low background ^{13}C enrichment of expired CO_2, the ingestion of CHO from plants with a C_4 photosynthetic cycle, which are naturally enriched in ^{13}C (Lefèbvre, 1985), was avoided during the period of experiments. Subjects also refrained from exercising, and from drinking coffee and alcohol for 2 d before each experiment.

During the exercise period, the subjects ingested 1000 ml water at room temperature with a low-calorie sweetener (Aspartam; Nabisco, Etobicoke, Ontario, Canada) as a placebo, or 100 g glucose, fructose or galactose in 1000 ml water. The solutions were given in five 200 ml doses taken immediately before the beginning of exercise and at 20, 40, 60 and 80 min during the exercise period. The hexoses ingested were artificially labelled with ^{13}C. Glucose, fructose (Biopharm, Laval, Quebec, Canada; −11·03 and −10·91 ‰ δ ^{13}C Pee Dee Bilemnitella (PDB-1), respectively) and galactose (Sigma Chemicals, St Louis, MO, USA; −23·4 ‰ δ ^{13}C PDB-1) were respectively enriched with [U $^{13}C_6$]-glucose, [U $^{13}C_6$]-fructose and [1 $^{13}C_1$]-galactose (ICN Pharmaceuticals Inc, Costa Mesa, CA, USA) in order to achieve final isotopic compositions close to +25 ‰ δ ^{13}C PDB-1 (actual values measured by mass spectrometry +24·5, + 24·1 and +24·2 ‰ δ ^{13}C PDB-1, for glucose, fructose and galactose, respectively).

Measures and calculations

Observations were made at rest immediately before exercise and every 30 min during the exercise period. Fat and CHO oxidation were computed from oxygen consumption ($\dot{V}_{O_2}$) and carbon dioxide production ($\dot{V}_{CO_2}$) using open-circuit spirometry (10 min collection periods). For the measurement of ^{13}C:^{12}C in expired CO_2, 80 ml samples of expired gas were collected and stored in Vacutainers (Becton Dickinson, Franklin Lakes, NJ, USA). Finally, 8 ml blood samples were withdrawn through a catheter (Baxter Health Care Corp., Valencia, CA, USA) inserted into an antecubital vein 30 min before the beginning of experiment, for the measurement of plasma glucose (Sigma Diagnostics, Sigma, Mississauga, Ontario, Canada) and insulin (KTSP-11 001; Immunocorp Sciences, Montreal, Quebec, Canada) concentrations. Plasma samples were stored at −80 °C until analysis.

Measurements of ^{13}C:^{12}C in expired CO_2 and in CO_2 from the combustion of ingested glucose, fructose and galactose were performed by mass spectrometry (Prism; VG, Manchester, UK) following cryodistillation as previously described (Adopo *et al.* 1994). The isotopic composition was expressed in ‰ difference by comparison with the PDB-1 Chicago Standard: ‰ δ ^{13}C PDB-1 = $((R_{spl}/R_{std}) - 1) \times 1000$, where R_{spl} and R_{std} are the ^{13}C:^{12}C ratio in the sample and standard (1·1237 %), respectively (Craig, 1953).

The oxidation of CHO (expressed in g glucose/min) and fat were calculated from $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ as follows (Péronnet & Massicotte, 1991):

$$\text{CHO} = 4{\cdot}59\,\dot{V}_{CO_2} - 3{\cdot}23\,\dot{V}_{O_2} \quad (1)$$

$$\text{Fat} = 1{\cdot}69\,(\dot{V}_{O_2} - \dot{V}_{CO_2}) \quad (2)$$

with mass in g and gas volume in L_{STPD}. The oxidation rate of the exogenous hexose (m_{exo}; g/min) was calculated as follows:

$$m_{exo} = \dot{V}_{CO_2}\,[(R_{exp} - R_{ref})/(R_{exo} - R_{ref})]/k \quad (3)$$

where $\dot{V}_{CO_2}$ is in L_{STPD}/min, R_{exp} is the ^{13}C:^{12}C observed in expired CO_2, R_{ref} is the ^{13}C:^{12}C in expired CO_2 in the control trial, R_{exo} is the ^{13}C:^{12}C in the exogenous glucose, fructose or galactose ingested, and k (0·747 l/g) is the volume of CO_2 provided by the complete oxidation of glucose, fructose or galactose. Endogenous CHO oxidation (g glucose/min) was calculated by the difference between total CHO and exogenous hexose oxidation. The contribution of the oxidation of the various substrates to the energy yield was computed from their respective energy potential.

These calculations are made based on the observation that, in response to exercise, ^{13}C is not irreversibly lost in pools of tricarboxylic acid cycle intermediates (Ruzzin *et al.* 2003) and/or bicarbonate (Trimmer *et al.* 2001), and that $^{13}CO_2$ recovery in expired gases is thus complete or almost complete. In addition, because ^{13}C:^{12}C in expired CO_2 equilibrates only slowly with ^{13}C:^{12}C in the CO_2 produced in the tissues (Pallikarakis *et al.* 1991), the absolute value for exogenous hexose oxidation could be somewhat underestimated at the beginning of exercise. This phenomenon probably does not, however, compromise the comparison of the availability of the three hexoses for oxidation.

Statistics

Data are presented as means with their standard errors. The main effects of time and exogenous substrate ingested, as well as time–substrate interactions, were tested by repeated-measures ANOVA (Statistica package; StatSoft, Tulsa, OK, USA). Newman–Keuls *post hoc* tests were used to identify the location of significant differences ($P<0{\cdot}05$) when ANOVA yielded a significant F ratio.

Results

Over the 120 min of exercise, $\dot{V}_{O_2}$, which was stable, and RER, which significantly decreased, were not significantly different in the four experimental situations (Table 1). At rest before ingestion of the placebo or the ^{13}C-hexose, the ^{13}C:^{12}C in expired CO_2 was not significantly different in the

Table 1. Oxygen consumption and respiratory exchange ratio (RER) during the exercise period

	0–60 min		60–120 min	
	Mean	SE	Mean	SE
Placebo				
$\dot{V}_{O_2}$ (l/min)	2·886	0·083	2·856	0·065
RER	0·916	0·007	0·887	0·007
Glucose				
$\dot{V}_{O_2}$ (l/min)	2·890	0·126	2·845	0·101
RER	0·916	0·012	0·892	0·010
Fructose				
$\dot{V}_{O_2}$ (l/min)	2·717	0·103	2·764	0·068
RER	0·927	0·006	0·908	0·008
Galactose				
$\dot{V}_{O_2}$ (l/min)	2·796	0·109	2·803	0·088
RER	0·909	0·012	0·880	0·013

four experimental situations and averaged −22·99 (SE 0·03; pooled data, *n* 24) (Fig. 1). In response to exercise with ingestion of the placebo, ^{13}C:^{12}C in expired CO_2 increased slightly, as commonly observed (Burelle *et al.* 2001), and reached a plateau after 30 min exercise. The increase in ^{13}C:^{12}C was much higher when ^{13}C-enriched hexoses were ingested, but the values observed were significantly higher beginning at 60 min with the ingestion of glucose and fructose than with the ingestion of galactose (main effect).

Substrate oxidation over the exercise period is presented in Table 2. Total CHO and fat oxidation decreased and increased, respectively, from the first to the second hour of exercise in the four experimental situations but was not significantly different with the placebo and the ingestion of the three hexoses. The amounts of exogenous hexoses oxidized significantly increased

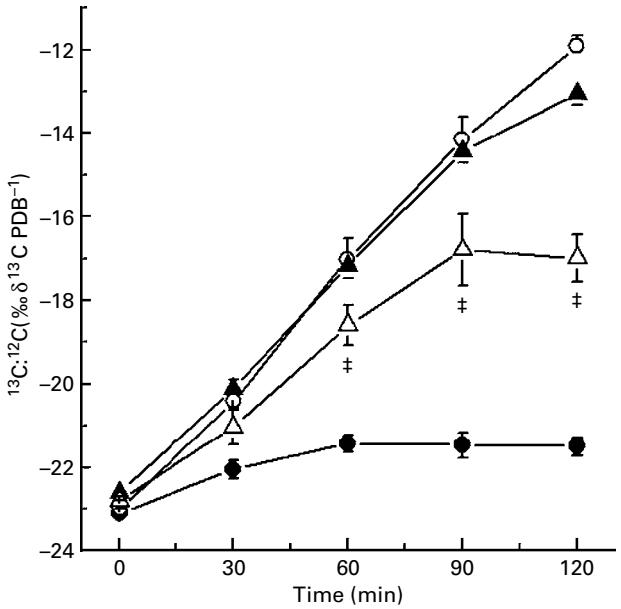

Fig. 1. Changes in ^{13}C:^{12}C in expired CO_2 in response to exercise with ingestion of placebo and ^{13}C-labelled hexoses (means and their standard errors; *n* 6). ‡Values were significantly different from those for glucose and fructose ($P<0·05$). –●–, Placebo; –○–, glucose; –▲–, fructose; –Δ–, galactose.

Table 2. Substrate oxidation (g) during the exercise period (Means and their standard errors, *n* 6)

	0–60 min		60–120 min	
	Mean	SE	Mean	SE
Placebo				
CHO total	168·6	6·0	144·2*	5·1
Exogenous hexose	0	0	0	0
Endogenous CHO	168·6	6·0	144·2*	5·1
Fat	23·1	2·4	31·2*	2·3
Glucose				
CHO total	164·1	10·2	147·2*	10·0
Exogenous hexose	9·0	1·4	31·5*	2·2
Endogenous CHO	155·1	10·4	115·8*†	10·8
Fat	22·7	3·9	29·7*	3·3
Fructose				
CHO total	167·1	6·2	55·2*	6·4
Exogenous hexose	9·2	1·2	29·6*	1·5
Endogenous CHO	158·0	6·1	125·6*†	6·3
Fat	18·6	2·1	24·4*	2·4
Galactose				
CHO total	157·3	7·6	135·0*	7·9
Exogenous hexose	5·6	1·1	18·1*†‡	2·3
Endogenous CHO	151·6	7·3	116·9*†	5·9
Fat	24·6	4·2	33·0*	4·7

CHO, carbohydrate.
* Values were significantly different from the 0–60 min period ($P<0·05$).
† Values were significantly different from those for the placebo ($P<0·05$).
‡ Values were significantly different from those for glucose and fructose ($P<0·05$).

from the first to the second hour of exercise, with no significant difference between exogenous glucose and fructose oxidation. In contrast, the amount of exogenous galactose oxidized was significantly lower (approximately 60 %) than that of exogenous glucose and fructose over both the first and second hour of exercise. As a consequence, galactose oxidation only contributed 5·5 (SE 0·9) % to the energy yield (significantly lower than the percentage energy yield from the oxidation of exogenous glucose and fructose: 9·2 (SE 0·8) and 9·0 (SE 0·6) %, not significantly different). The oxidation of endogenous glucose, which significantly decreased from the first to the second hour of exercise, was significantly lower when exogenous hexoses rather than placebo were ingested. The amount of fat was not significantly modified by ingestion and oxidation of the three hexoses, and was not significantly different, respectively, in the four experimental situations (Fig. 2).

No significant differences were observed between trials for basal plasma glucose and insulin concentration (Fig. 3). Throughout the exercise period, plasma glucose concentration remained at or slightly above basal values (approximately 5·5 mmol/l) when glucose or fructose was ingested. Although this did not reach significance, plasma glucose concentration steadily decreased when placebo or galactose was ingested, and the values observed were significantly lower than with glucose or fructose ingestion at 80 min. The plasma insulin concentration significantly decreased in response to exercise but was not significantly different between the four situations (main effect).

Discussion

The results from the present experiment show that, compared with glucose and for similar amounts ingested during exercise,

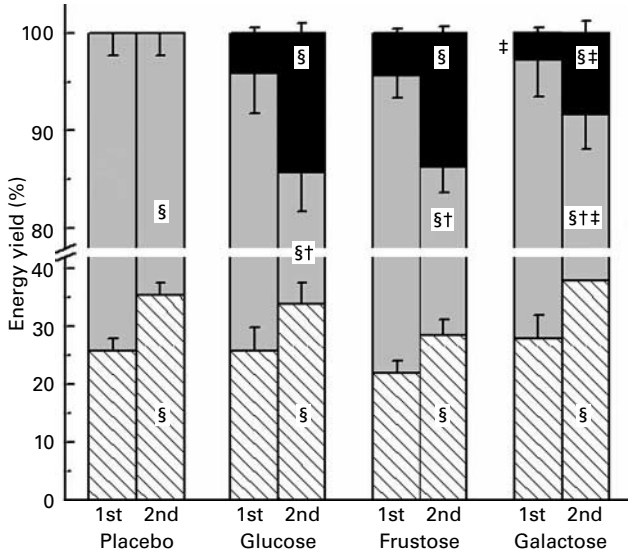

Fig. 2. Percentage contribution to the energy yield of fat (▨), endogenous CHO (■) and exogenous hexoses (■) over the first and second hours of exercise (mean and their standard errors; *n* 6). †Values were significantly different from those for the placebo ($P<0·05$). ‡Values were significantly different from those for glucose and fructose ($P<0·05$). §Values were significantly different from the first hour ($P<0·05$).

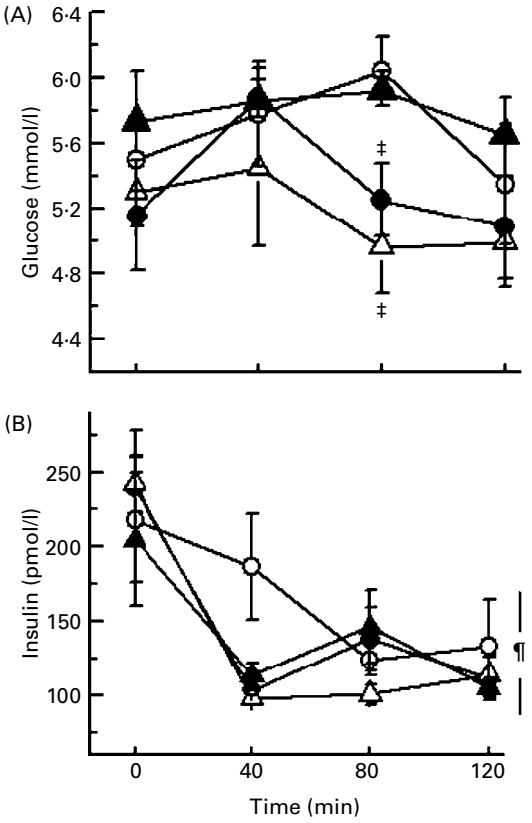

Fig. 3. Plasma concentration of glucose (A) and insulin (B) in the four experimental conditions (mean and their standard errors; *n* 6). ‡Values were significantly different from those for glucose and fructose ($P<0·05$). ¶Values were significantly different from the corresponding value at 0 min ($P<0·05$). –●–, Placebo; –○–, glucose; –▲–, fructose; –Δ–, galactose.

the oxidation of galactose was significantly lower (approximately 40 %), whereas the oxidation of fructose was only about 4 % lower and not significantly different. The ingestion of hexoses did not significantly modify total CHO and fat oxidation, but it significantly reduced (by 9–13 %) endogenous CHO oxidation over the 120 min exercise.

Only one study is available concerning the oxidation of ingested galactose during prolonged exercise (Leijssen *et al.* 1995). In this study, the oxidation rates of 155 g exogenous glucose and galactose were compared during 120 min exercise at 65 % $\dot{V}_{O_{2max}}$ (3·52 l/min). The amount of galactose oxidized over the exercise period (33·4 g) was 46 % that of exogenous glucose (71·7 g). Results from the present experiment are in line with these findings as the amount of galactose oxidized over the 120 min exercise was 23·7 (SE 3·5) g, a value that was 59 % that of exogenous glucose (40·5 *v.* 3·4 g).

In the study by Leijssen *et al.* (1995), when compared with glucose ingestion, the lower oxidation rate of galactose was associated with a compensatory 11 % increase in endogenous CHO oxidation, whereas fat oxidation was similar. However, no control situation with an ingestion of water only was included. In the present study, when compared with the control situation during the 120 min exercise period, the oxidation of galactose significantly reduced endogenous CHO oxidation by 14 % but did not modify fat or total CHO oxidation. When compared with the effect of glucose ingestion, the reduction in endogenous CHO oxidation was very similar with the ingestion of galactose.

The slightly lower oxidation rate of both exogenous glucose and galactose reported in the present experiment when compared with that reported by Leijssen *et al.* (1995), as well as the difference in endogenous glucose oxidation, was probably due to the fact that the amounts of exogenous hexose ingested and the absolute workload were both lower (100 *v.* 155 g, 2·82 *v.* 3·52 l/min, respectively). Indeed, exogenous CHO oxidation has been shown to increase with workload (Wagenmakers *et al.* 1993; Massicotte *et al.* 1994; Pirnay *et al.* 1995), as well as with amount ingested (Adopo *et al.* 1994; Jeukendrup *et al.* 1999; see also Jeukendrup, 2004, for a review).

With exogenous fructose, most studies (Massicotte *et al.* 1986, 1989, 1990, 1994; Guézennec *et al.* 1989; Jandrain *et al.* 1993; Adopo *et al.* 1994; Burelle *et al.* 1997) have shown that, for similar amounts ingested, its oxidation rate during prolonged moderate exercise is lower than that of exogenous glucose or starch. In some of these studies, the difference was large (oxidation rate 64–85 % that of exogenous glucose) and statistically significant (Massicotte *et al.* 1986, 1989, 1990, 1994; Guézennec *et al.* 1989; Jandrain *et al.* 1993; Adopo *et al.* 1994). In contrast, in the studies by Burelle *et al.* (1997) and by Slama *et al.* (1989), the difference was smaller (approximately 5–10 %) and not significantly different, and Décombaz *et al.* (1985) reported a significantly 13 % higher oxidation rate of exogenous fructose than glucose.

In the present experiment, the amount of exogenous fructose oxidized over the 120 min of exercise period was only approximately 4 % lower than that of exogenous glucose, and the difference was not statistically significant. This observation confirms the fact that, in certain situations, fructose oxidation could be similar to exogenous glucose oxidation. On the basis of the limited experimental data available, however, it is difficult to identify the factor(s) explaining how, in certain

situations, fructose oxidation could be lower than, similar to or even higher than that of exogenous glucose. Finally, in the present experiment, in line with several studies comparing the effect of glucose and fructose ingestion (Massicotte *et al.* 1986, 1990, 1994; Jandrain *et al.* 1993; Adopo *et al.* 1994), the reduction in endogenous CHO oxidation observed with fructose was very similar to that observed with ingestion of glucose (12–17 %).

When compared with that of exogenous glucose, the lower oxidation rate of exogenous galactose and fructose, when present, could be due to difference in intestinal absorption, changes in plasma concentration and renal handling, and in metabolism of the three hexoses in the liver and peripheral tissues (mainly muscle during exercise).

No data are currently available for directly comparing the intestinal absorption of glucose, fructose and galactose at rest or exercise in man, and the mechanisms by which each of these three hexoses is absorbed are not fully understood (Kellett, 2001; Santer *et al.* 2003; Wright *et al.* 2003). Glucose, fructose and galactose cross the brush-border membrane of the enterocytes through an active transport mechanism (SGLT1 co-transporter, with a similar affinity for gluctose and galactose but none for fructose; see Wright *et al.* 2003 for a review) or facilitated diffusion (GLUT2 transporters, which have a higher affinity for glucose but can handle all three hexoses (Kellet, 2001; Wright *et al.* 2003); GLUT5 transporters, which are highly specific for fructose) (Ferraris & Diamond, 1997). As for absorption across the basolateral membrane of the enterocyte, the three hexoses appear to share the common facilitated diffusion mediated by GLUT2 transporters (Wright *et al.* 2003). Glucose could also be absorbed following the formation of glucose-6-phosphate, transport within the endoplasmic reticulum, vesicle trafficking and the release of free glucose outside the cell (Stümpel *et al.* 2001; Santer *et al.* 2003).

Taken together, these data suggest that the intestinal absorption of glucose could be slightly more efficient than that of galactose, which in turn could be more efficient than that of fructose. These differences in the intestinal absorption of hexoses could at least partly explain why, in most studies (Massicotte *et al.* 1986, 1989, 1990, 1994; Guézennec *et al.* 1989; Slama *et al.* 1989; Jandrain *et al.* 1993; Adopo *et al.* 1994; Burelle *et al.* 1997), the oxidation rate of exogenous fructose has been shown to be slightly lower (albeit not always statistically significantly lower) than that of exogenous glucose. This does not, however, explain why the oxidation rate of exogenous galactose in response to exercise is much lower than that of exogenous glucose, as shown by Leijssen *et al.* (1995) as well as in the present experiment.

One possible explanation for the much lower oxidation rate of galactose than fructose or glucose is the loss of galactose in the urine. In contrast to what is observed following the ingestion of glucose (Jeukendrup *et al.* 1999; Couture *et al.* 2002; Jentjens *et al.* 2004*a*,*b*) or fructose (Jandrain *et al.* 1993; Burelle *et al.* 1997), the ingestion of galactose results in a marked increase in plasma galactose concentration (e.g. 12 mmol/l at the end of exercise with an ingestion of 155 g galactose; Leijssen *et al.* 1995). Because the renal threshold for galactose could be as low as 0·5 mmol/l (Williams, 1986), this explains why Ganda *et al.* (1979) recovered from the urine approximately 16 % of a 32·5 g dose of galactose ingested at rest (plasma galactose concentration 19 mmol/l). Although this was not measured by Leijssen *et al.* (1995) or in the present study, losses of galactose in the urine could be suspected owing to the large amounts ingested.

Finally, as discussed by Leijssen *et al.* (1995) for galactose, and as shown for fructose (Jandrain *et al.* 1993), the lower oxidation rate of these hexoses compared with glucose during prolonged exercise is probably mainly due to their different metabolic fate in the liver and peripheral tissues. Because of the much higher affinity of muscle hexokinase than liver glucokinase for glucose, and because of the stimulation of muscle glucose uptake in response to exercise (for a review, see Pereira & Lancha, 2004), the oxidation rate of plasma glucose increases markedly, and the oxidation rate of exogenous glucose can reach about 1·2 g/min in as much as the ingestion rate is large enough (Jeukendrup *et al.* 1999; Couture *et al.* 2002; Jentjens *et al.* 2004*a*,*b*).

In contrast, because of the presence of a fructokinase and a galactokinase, fructose and galactose could be preferentially taken up by the liver (Chen & Whistler, 1977; Williams, 1986). Data from Ahlborg & Björkman (1990) indicate that only small amounts of fructose if any are taken up by the muscle, except when the plasma fructose concentration is very high (8·8 mmol/l in their study). Data from Jandrain *et al.* (1993) indicate that exogenous ^{13}C-fructose ingested during exercise, and presumably taken up by the liver, was quickly converted into ^{13}C-glucose, which was released in the blood and became available for oxidation in the muscle. The same phenomenon has been shown following ^{13}C-galactose ingestion at rest (Berry *et al.* 1995), but no data are currently available on possible conversion of exogenous galactose to plasma glucose during exercise.

Because of the presence of the Leloir pathway (Williams, 1986), galactose could be preferentially converted into liver glycogen, thus escaping oxidation. A preferential incorporation of exogenous galactose into liver glycogen during exercise is suggested by data from Leijssen *et al.* (1995). In their study, following a 60 min recovery period after the first 120 min period of exercise, the oxidation rates of ^{13}C-galactose and ^{13}C-glucose were similar during a subsequent 30 min period at 60 % maximal workload. This could reflect the release and oxidation of glucose from liver glycogen synthesized from ^{13}C-galactose during the first exercise period and the subsequent recovery.

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