Oxidation rates of orally ingested carbohydrates during prolonged exercise in men

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WAGENMAKERS, ANTON J. M., FRED BROUNS, WIM H. M. SARIS, AND DAVID HALLIDAY. Oxidation rates of orally ingested carbohydrates during prolonged exercise in men. J. Appl. Physiol. 75(6): 2774–2780, 1993.—Six male volunteers exercised on a cycle ergometer at 65% of maximal work load for 120 min on six occasions while ingesting water (W) only, four doses of maltodextrin (M) [0.92, 1.85, 2.77, and 3.70 g/kg body wt (4, 8, 12, and 16% M, respectively)], and sucrose (S) [1.85 g/kg body wt (8% S)]. Drinks were given during warm-up (8 min), body wt. The rates were each 5 min during exercise (2 ml/kg body wt). M and S were of high 13C natural abundance. Total carbohydrate (CHO) and fat oxidations were calculated from the nonprotein respiratory exchange ratio. M and S increased total CHO oxidation compared with W; no difference was observed between CHO solutions. Total CHO oxidation decreased continuously with time and more rapidly after W than after M or S. Fat oxidation increased continuously in all treatments. Oxidation rates of ingested CHO were 82 ± 19, 76 ± 12, 86 ± 10, and 91 ± 9 g/h for 4, 8, 12, and 16% M, respectively. The oxidation rate of S was 81 ± 10 g/h (not different from 8% M), which indicated that the glucose polymer had no advantage over S. Oxidation rates of M and S increased to a plateau after 90–120 min of exercise. For all solutions except 4% M, the plateau oxidation rate was close to 1.0 g/min. Differences between 8, 12, and 16% M and 8% S were minimal such that ingestion of 8% M or S may well have had an optimal ergogenic effect. With 12 and 16% M, ingestion exceeded the plateau observed during the last 30-min period, which indicated the accumulation of exogenous CHO in the gastrointestinal tract and/or in unidentified endogenous pools.

carbon-13 enrichment; stable isotopes; performance; maltodextrins; sucrose; blood metabolites

CARBOHYDRATES and FATTY ACIDS are the main fuels oxidized by skeletal muscle to provide energy during prolonged exercise (22). To avoid exhaustion, humans have to reduce exercise intensity to ≤50% of maximal work load (W max) when the glycogen stores have been emptied and fatty acids have become the main fuel (22). This implies that exercise at relatively high intensities (>60% W max) can be continued only when a mixture of fat and carbohydrate is oxidized. For this reason also dietary measures that maximize the muscle glycogen store [carbohydrate loading (1)] and carbohydrate ingestion during exercise have been shown to improve performance during prolonged exercise at moderate and high intensities (5–9).

Because of their ergogenic effect, the use of carbohydrate drinks has become common practice in many sports events and disciplines lasting >1–2 h. Recommendations have been given on the optimal rate of carbohydrate ingestion (9), but this remains an issue of debate because of the lack of direct scientific information on oxidation rates of orally ingested carbohydrates as a function of the dose given. Too little intake may not provide enough energy to sustain optimal work rates in muscle (5–9). On the other hand, ingestion of large quantities of carbohydrates and large volumes of solutions may lead to accumulation of carbohydrates and fluid in stomach and gut and lead to the well-known gastrointestinal complaints that can force athletes to give up competition (4).

The use of carbohydrates with a high 13C natural abundance makes it possible to quantitate the oxidation rate of orally ingested carbohydrates (18). Previously this technique has been used for comparison of the oxidation rate of two doses of glucose (200 and 400 g) ingested during 285 min of exercise at 45% of maximal O2 consumption (VO2max) (23).

Doubling the glucose dose given substantially increased the oxidation rate (137 vs. 227 g).

In another study, Rehrr et al. (28) gave 58 and 220 g of glucose in the form of 4.5 and 17% solutions, respectively, to subjects during 80 min of exercise at 70% VO2max. Of the glucose given, only 32 and 42 g, respectively, were oxidized, which suggested that giving four times as much glucose hardly had an advantage in terms of energy provision to the working muscle. The remarkable difference in the metabolic availability of the oral glucose between these studies may be due to differences in exercise duration and intensity. In the present study, oxidation rates of four maltodextrin solutions with a carbohydrate content increasing from ~4 to 16% were measured during 2 h of exercise at 65% W max to define the dose of maltodextrin that gives the maximal oxidation rate. Total amounts of carbohydrates ingested during exercise were 0.92, 1.85, 2.77, and 3.70 g/kg body wt. The oxidation rate of an 8% sucrose solution was also measured to look at a potential effect of the carbohydrate source on the oxidation rate. The effects of carbohydrate ingestion on total carbohydrate and fat oxidations, estimated with indirect respiratory calorimetry, and on changes in blood metabolite concentrations were also investigated.

METHODS

Subjects. Six healthy highly trained male amateur cyclists, competing at national and international level, vol-
The subjects exercised on electromagnetically braked cycle ergometers (Lode) at a freely chosen power output-independent pedaling rate (80–120 rpm) in a temperature-controlled (18 ± 1°C) room. After the Wmax attained during incremental cycle exercise had been determined [403 ± 42 (SD) W] as previously described (17), the subjects were studied on six occasions during 2 h of cycle exercise at 65% Wmax. The experiments were conducted in the morning (9:30 a.m.) after an overnight 14-h fast and a standardized breakfast (identical for all trials) 2 h before exercise. The breakfast included 1 g/kg body wt of white bread and 5 ml/kg body wt of Meritene (Wander, Bern, Switzerland). The breakfast composition was (percent total energy provided) 23.8 protein, 13.2 lipid, and 62.9 carbohydrate, with 29 kJ energy/kg body wt. All carbohydrates in the breakfast were of C4 metabolic origin (low natural enrichment). Six test drinks were given in random order as a bolus (8 ml/kg body wt) during the last 4 min of warm-up before the 2-h exercise period and as repeated boluses (2 ml/kg body wt) each 15 min during exercise. Subjects drank only water once; a cornstarch-derived maltodextrin solution four times [Maldex-20 (Amylum, Aalst, Belgium), a glucose polymer containing the following mixture of (glucose)4 (in percent): 10.6, n = 1; 10.7, n = 2; 13.4, n = 3; 9.6, n = 4; 8.7, n = 5; 12.0, n = 6; 11.1, n = 7; 5.0, n = 8; 2.4, n = 9; 1.4, n = 10; and 15.0, n > 10] with different carbohydrate contents of 4, 8, 12, and 16% (wt/vol) and different total carbohydrate ingestions of 0.92, 1.85, 2.77, and 3.70 g/kg body wt, respectively; and a cane sugar-derived sucrose content 8%; total carbohydrate intake 1.85 g/kg body wt). Warm-up consisted of 3 min of exercise at 100 W, 4 min at 40% Wmax and finally 4 min at 55% Wmax.

Methods. O2 consumption and CO2 production were measured throughout exercise in 3-min blocks every 7.5 min with a SensorMedics 2900 indirect calorimeter system. Forearm venous blood samples were drawn at rest immediately before warm-up and 2 min after completion of the 2-h exercise protocol. Aliquots of plasma were frozen for analysis of glucose (glucose oxidase-Perid method, no. 124 010, Boehringer-Mannheim). Serum was prepared and frozen for analysis of fatty acids (NEFA C test kit no. 994–75409, Wako Chemicals, Neuss, Germany) and glycerol (no. 125 032, Boehringer-Mannheim).

Breath samples were drawn directly from the mixing chamber of the SensorMedics 2900 system and were collected in duplicate in 20-ml vacutainer tubes before warm-up (rest sample) and each 15 min during exercise. 13C enrichment in CO2 was measured with a Finnigan isotope ratio mass spectrometer with on-line breath car- ousel and sample derivatization equipment as previously described (30). The isotopic enrichment of CO2 was expressed as the change per mil difference between the 13C-to-12C ratio of the sample and a known laboratory reference standard according to

\[ \delta^{13}C = \left( \frac{^{13}C/^{12}C \text{ sample}}{^{13}C/^{12}C \text{ standard}} - 1 \right) \times 10^3 \]

The 13C was then related to the international standard Pee Dee Belemnite (PDB). The 13C enrichment of expired CO2 of our subjects at rest (n = 36) was -24.12 ± 0.34% (SD) vs. PDB. The 13C enrichment of the ingested maltodextrin was -9.5% vs. PDB, and that of the sucrose was -9.95% vs. PDB. The increase in 13C enrichment in expired CO2 observed in a given time interval in the exercise test with water ingestion only was subtracted from the increase observed in the tests with carbohydrate ingestion (correction for the background change). The amount of ingested carbohydrates oxidized in a given time interval during exercise was then calculated from the background-corrected 13C enrichment in expired CO2 and that of the ingested carbohydrates according to a computation procedure proposed previously (26). Oxidation rates of the ingested maltodextrin and sucrose are given as grams of glucose equivalents oxidized, assuming that 1.000 g of Maldex-20 provides 1.085 g of glucose and 1.000 g of sucrose provides 1.052 g of glucose equivalents.

The amounts of carbohydrates and lipids oxidized at each time interval were computed from the respiratory exchange ratio taken as the nonprotein respiratory quotient.

Statistical analysis. A one-factor analysis of variance for repeated measures was used to compare differences between the six ingested solutions in substrate oxidation in a given time period and in changes in the concentration of blood metabolites during the 2 h of exercise. The level of significance was set at P < 0.05. A Fisher's protected least-significant difference post hoc test was used to define which treatments were different from each other.

RESULTS

In the control test, plasma concentration of glucose before exercise was 5.09 ± 0.89 mM and the serum concentration of fatty acids was 0.18 ± 0.07. Similar concentrations were observed before exercise in the various carbohydrate treatments (data not shown). Changes in concentration of these metabolites during exercise are shown in Fig. 1. The decrease in glucose concentration during exercise was reduced by the various carbohydrate treatments with the reductions by the 12 and 16% maltodextrin solutions being significant. The three highest
OXIDATION OF ORALLY INGESTED CARBOHYDRATES

Fat oxidation was significantly higher after water ingestion than after ingestion of the 4, 12, and 18% maltodextrin solutions (Table 1). No significant difference existed in this respect between the control, 8% maltodextrin, and 8% sucrose solutions. Fat oxidation gradually increased for subsequent 30-min periods for all treatments with the control treatment showing the highest oxidation rate in all periods (Table 2; data in part not shown). Fat oxidation was significantly lower with the 4 and 16% maltodextrin solutions than with the other treatments in the 30- to 60-, 60- to 90-, and 90- to 120-min periods.

In Fig. 2 the $^{13}$C enrichment of breath CO$_2$ is shown for the control run with water ingestion only and for the four maltodextrin and sucrose treatments compared with the $^{13}$C enrichment of the rest sample taken before warm-up. In the test with only water ingestion, there was no significant increase in $^{13}$C enrichment during exercise and no difference between the $^{13}$C enrichment of the exercise samples and that of the rest sample obtained immediately before exercise.

Oxidation rates of the orally ingested carbohydrates increased slowly but gradually when the maltodextrin dose was increased fourfold, ranging from 52 to 91 g/h. With the lowest maltodextrin dose, 70 ± 18% of the ingested carbohydrates was oxidized in the 2-h period. This figure decreased to 51 ± 7, 39 ± 4, and 31 ± 3 for the 8, 12, and 16% maltodextrin solutions, respectively. The oxidation rate of the lowest maltodextrin dose was lower than those of the 12 and 16% maltodextrin solutions. No significant differences existed between the other treatments, and no significant difference was observed between the 8% maltodextrin and 8% sucrose treatments (Table 1).

The oxidation rates of orally ingested carbohydrates in the first and last 30-min periods were shown in Table 2. Some care should be exerted in interpreting the oxidation rates in the 0- to 30-min period. A delay occurs in the appearance of $^{13}$CO$_2$ in the breath because of dilution of the $^{13}$CO$_2$, originating from the oxidation of orally ingested carbohydrates, in the bicarbonate pools of the body. Oxidation rates increased gradually (data not shown) and tended to plateau toward the end of the 2-h

![Figure 1](image.png)

**FIG. 1.** Effect of carbohydrate ingestion on changes in blood metabolites during prolonged exercise. Values are means ± SD. Six male volunteers exercised on cycle ergometer at 65% of maximal work load for 120 min on 6 occasions during which they ingested water only (control), 4 doses of maltodextrin (M; 0.92, 1.85, 2.77, and 3.70 g/kg body wt given as 4, 8, 12, and 16% solutions, respectively), and sucrose (S; 1.85 g/kg body wt given as 8% solution). Drinks were given as described under Experimental protocol. Forearm venous blood samples were drawn before and at end of exercise. Glucose concentration was measured in plasma (A), and fatty acid concentration in serum was measured as described under Methods (B). *Significantly different from control treatment with water ingestion only (analysis of variance followed by Fisher's protected least-significant difference post hoc test).

maltodextrin treatments significantly reduced the increase in serum fatty acid concentration. No differences were observed in this respect between the sucrose, 4% maltodextrin, and control treatments. Changes similar to those obtained for the fatty acid concentrations were seen for the increase in serum glycerol concentration during exercise (data not shown).

Total carbohydrate oxidation during the 2-h exercise period at 65% $W_{max}$ was significantly higher after carbohydrate ingestion than in the control test with only water ingestion except for the 8% sucrose solution (Table 1). No significant differences were present between carbohydrate treatments. The difference between carbohydrate treatments and water ingestion appears to be due to the fact that total carbohydrate oxidation fell more rapidly in the control test while the subjects exercised (Table 2). No difference was present in the first 30-min period, differences became significant ($P < 0.05$) between 30 and 60 min of exercise (data not shown), and differences increased by 14–23 g/30 min between 90 and 120 min of exercise (Table 2).

**TABLE 1.** Overall substrate oxidation during exercise: effect of ingestion of various CHO solutions

<table>
<thead>
<tr>
<th>Carbohydrate</th>
<th>Fat</th>
<th>Total CHO</th>
<th>Oral CHO</th>
<th>Endogenous CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>123±32</td>
<td>236±22</td>
<td>236±22</td>
<td>236±22</td>
</tr>
<tr>
<td>4% Maltodextrin</td>
<td>99±10*</td>
<td>297±29*</td>
<td>52±19*</td>
<td>298±33</td>
</tr>
<tr>
<td>8% Maltodextrin</td>
<td>118±20</td>
<td>279±58*</td>
<td>76±12*</td>
<td>203±55*</td>
</tr>
<tr>
<td>12% Maltodextrin</td>
<td>107±20*</td>
<td>283±52*</td>
<td>86±10†</td>
<td>197±51†</td>
</tr>
<tr>
<td>16% Maltodextrin</td>
<td>101±10*</td>
<td>295±35*</td>
<td>91±9†</td>
<td>205±31†</td>
</tr>
<tr>
<td>8% Sucrose</td>
<td>114±12</td>
<td>205±29</td>
<td>81±10†</td>
<td>184±22†</td>
</tr>
</tbody>
</table>

Values are means ± SD of 6 subjects. Oxidation rates were measured and calculated as described in METHODS. Oxidation rates of oral carbohydrates (CHO) are given as grams of glucose equivalents derived from oral CHO. Respective value of each total maltodextrin ingestion was 0.92, 1.85, 2.77, and 3.70 g/kg body wt and of sucrose ingestion was 1.85 g/kg body wt. Significantly different from: * control; † 4% maltodextrin [ANOVA and Fisher's protected least-significant difference (PLSD) post hoc test].
TABLE 2. Substrate oxidation during exercise in 0- to 30- and 90- to 120-min periods: effect of ingestion of various CHO solutions

<table>
<thead>
<tr>
<th>Substrate</th>
<th>Period, min</th>
<th>Control</th>
<th>4%</th>
<th>8%</th>
<th>12%</th>
<th>16%</th>
<th>8% Sucrose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pat</td>
<td>0–30</td>
<td>25±5</td>
<td>21±5</td>
<td>23±9</td>
<td>22±6</td>
<td>21±2</td>
<td>25±3</td>
</tr>
<tr>
<td></td>
<td>90–120</td>
<td>33±8</td>
<td>29±2*</td>
<td>32±2</td>
<td>31±6</td>
<td>32±2*</td>
<td>32±4</td>
</tr>
<tr>
<td>Total CHO</td>
<td>0–30</td>
<td>50±14</td>
<td>48±13</td>
<td>50±16</td>
<td>50±14*</td>
<td>50±11*</td>
<td>50±17*</td>
</tr>
<tr>
<td></td>
<td>90–120</td>
<td>42±9</td>
<td>65±8*</td>
<td>60±15*</td>
<td>60±14*</td>
<td>65±11*</td>
<td>65±17*</td>
</tr>
<tr>
<td>Oral CHO</td>
<td>0–30</td>
<td>6±4</td>
<td>9±2*</td>
<td>9±2*</td>
<td>9±2*</td>
<td>9±2*</td>
<td>8±4</td>
</tr>
<tr>
<td></td>
<td>90–120</td>
<td>10±6</td>
<td>26±5*</td>
<td>30±6*</td>
<td>32±4*</td>
<td>32±6*</td>
<td>32±5</td>
</tr>
<tr>
<td>Endogenous CHO</td>
<td>0–30</td>
<td>80±15</td>
<td>78±13</td>
<td>78±14</td>
<td>78±17</td>
<td>78±19</td>
<td>78±18</td>
</tr>
<tr>
<td></td>
<td>90–120</td>
<td>42±9</td>
<td>47±5</td>
<td>34±13</td>
<td>30±11</td>
<td>32±8</td>
<td>30±5</td>
</tr>
</tbody>
</table>

Values are means ± SD of 6 subjects. Oxidation rates were measured and calculated as described in METHODS. Oxidation rates of oral CHO are given as grams of glucose equivalents derived from oral CHO. Respective value of each total maltodextrin ingestion was 0.92, 1.85, 2.77, and 3.70 g/kg body wt and of sucrose ingestion was 1.85 g/kg body wt. * Significantly different from control (ANOVA with Fisher PLSD post hoc test).

Period at ~30 g/30 min (1 g/min). Between 30 and 120 min, the oxidation rate of the lowest maltodextrin dose was lower than those for all other treatments (Table 2; data in part not shown). No significant difference existed between the other treatments in the 30- to 60-, 60- to 90-, or 90- to 120-min periods (Table 2; data in part not shown).

The oxidation rate of the endogenous carbohydrates (i.e., liver and muscle glycogen, glucose made by gluconeogenesis from endogenous carbon skeletons) was calculated by subtracting the oxidation rate of ingested carbohydrates from the total carbohydrate oxidation. The values that were obtained are shown in Tables 1 (per 2-h period) and 2 (per 30-min period). No difference was observed between endogenous carbohydrate oxidation in the control run with water ingestion only and the 4% maltodextrin treatment. The three highest maltodextrin treatments and the sucrose treatment significantly reduced oxidation of endogenous carbohydrates compared with the control and 4% maltodextrin solutions. No differences were observed in this respect between the three highest maltodextrin and sucrose treatments in the 2-h period or in any of the 30-min periods.

DISCUSSION

Many previous studies have quantitated the rate of oxidation of orally ingested substrates with the use of substrates with a high 13C natural abundance (10, 11, 14, 15, 18–20, 23–29, 31). However, Péronnet et al. (26) raised concerns about possible overestimates of endogenous substrate oxidation rates after they observed increases in the 13C enrichment of breath CO2 in exercise tests with only water ingestion. Most of the earlier reports ignored these changes and used the 13C enrichment of breath CO2 in the resting sample as a reference in calculating oxidation rates. Recently it has been shown that in subjects who adhere to a traditional European diet no changes occur in the 13C enrichment of breath CO2 during exercise with only water ingestion (31). This was confirmed again in the present study (Fig. 2). It has been pointed out that the concerns raised by Péronnet et al. apply primarily to subjects on North American diets (31). Both differences in the type of carbohydrate in the diet (more C4 plant-derived carbohydrates are consumed in North America than in Europe) and differences in the European and North American total food chains (21) may contribute to the greater increases in the North American diet of 13C enrichment of breath CO2 during exercise tests with only water ingestion.

Potential advantages of carbohydrate ingestion, with respect to energy provision to the working skeletal muscle, become more clear with prolonged exercise when the endogenous carbohydrate stores become smaller. For this reason, gradual changes are to be expected during 2 h of exercise at 65% Wmax. We therefore analyzed our data for both the 2-h and 30-min periods. Our data show that carbohydrate ingestion during exercise has clear advantages with respect to maintenance of blood glucose (Fig. 1) and maintenance of rates of carbohydrate oxidation, especially toward the end of the 2-h exercise period (Table 2). Total carbohydrate oxidation decreased more in the control than after carbohydrate supplementation. This finding was primarily due to the fact that the oxidation of the ingested carbohydrates increased gradu-
Oxidation of orally ingested carbohydrates

July during exercise (Table 2). In the last 30-min period, oral carbohydrate oxidation represented ~50% of total carbohydrate oxidation with the 8, 12, and 16% solutions (Table 2). An even higher contribution (37.5%) of oral carbohydrate oxidation to total carbohydrate oxidation has been reported by Pallikarakis et al. (23) for the last hour of a 4-h exercise bout at 45% $\text{V}_\text{O}_2\text{max}$ with ingestion of 50 g of glucose each 30 min.

An important observation in the present study was that maximal oxidation rates and the maximal carbohydrate sparing effect were reached with the 8% maltodextrin and 8% sucrose solutions (mean carbohydrate intake 148 g/2 h). A further increase in the maltodextrin content to 12 and 16% (mean intakes of 222 and 296 g/2 h, respectively) did not lead to a significant increase in oxidation of the ingested carbohydrates or a further reduction in the oxidation of endogenous carbohydrates. Our data therefore suggest that an intake of 148 g/2 h according to the suggested ingestion protocol (52 g at start followed by 13 g each 15 min) has an optimal ergogenic effect when the exercise conditions studied here apply (2 h of cycling at 65% W$_\text{max}$).

With intake rates of ≥148 g/2 h, oxidation rates of the ingested carbohydrates reached values between 0.9 and 1.1 g/min in the last 30-min period (Table 2). A similar value was reported by Pallikarakis et al. (23) in the last hour of a 4-h exercise bout at 45% $\text{V}_\text{O}_2\text{max}$ with ingestion of 50 g of glucose each 30 min. Higher values have never been reported in studies using naturally enriched $^{13}$C-labeled carbohydrates (10, 11, 14, 15, 18–20, 24–29). A value of 1.8 g/min has recently been reported for the later stages of 90 min of exercise at 70% $\text{V}_\text{O}_2\text{max}$ in a study using $^{14}$C-labeled carbohydrates (13), but this value, as admitted by the authors (12), is most probably an overestimation due to different solubility characteristics of tracer and tracee leading to preferential oxidation of the tracer (more correct values are given in Ref. 29). The oxidation rate of 1.1 g/min is approaching the maximal rate of blood glucose utilization during exercise at 67% $\text{V}_\text{O}_2\text{max}$ to fatigue, which was estimated at 1.2 g/min from femoral arteriovenous glucose fluxes (2). The value also approaches the whole body disposal rate of glucose in glycogen-depleted subjects that was measured by Coggan and Coyle (6), who used the euglycemic clamp technique (1.13 g/min). The value of 1.1 g/min for ingested carbohydrate oxidation also approaches the total carbohydrate oxidation rate observed after 120 min of exercise in the control test with only water ingestion (1.4 g/min). This finding may imply that the ingested carbohydrate delivers carbohydrate at such a rate that muscle glycogen is not required to sustain muscle glucose oxidation rates at exercise intensities of 65% W$_\text{max}$.

No differences were observed in this study between 8% maltodextrin and 8% sucrose solutions with respect to oxidation of the ingested carbohydrates, effects on total carbohydrate and fat oxidation, and sparing of endogenous carbohydrates. Massicotte et al. (20) previously also did not find a difference in the oxidation rate of glucose or a glucose polymer (7% solution; 1.33 g/kg body wt each 20 min) during 120 min of cyclic exercise at 55% $\text{V}_\text{O}_2\text{max}$. Rehrer et al. (28) did not find any difference in the oxidation rate of glucose or maltodextrin solution (both 17% solutions; 5 ml/kg body wt during warm-up followed by 3 ml/kg body wt each 20 min) during 80 min of exercise at 70% $\text{V}_\text{O}_2\text{max}$. Guezennec et al. (11) reported similar oxidation rates for ingested starch suspensions and glucose solutions given in similar dosages, and Saris et al. (29) found similar mean and peak oxidation rates (1.1 g/min) as reported here for a soluble starch and reduced rates for an insoluble starch during 150 min of exercise at 60% W$_\text{max}$ (16% starch suspensions; 6 ml/kg body wt during warm-up followed by 2.5 ml/kg body wt each 15 min). Together, these data indicate that glucose polymer solutions (both maltodextrins and starches) do not offer an advantage over free glucose or sucrose solutions in terms of energy provision for the working muscle (see also Ref. 24 for a review).

The percentages of the orally ingested carbohydrates that were found to be oxidized in the present study with the two lower maltodextrin solutions (70 and 51% oxidized per 2 h) and sucrose solution (55% oxidized) are in agreement with earlier reports on the oxidation of carbohydrates of a high $^{13}$C natural abundance (11, 14, 19, 20, 23–29). However, here we also confirm that very large doses of carbohydrate lead to a much lower percentage of the ingested carbohydrates being oxidized.

In this study, drinks were given as a larger bolus (8 ml/kg body wt) at the start of exercise followed by repeated smaller boluses (2 ml/kg body wt) each 15 min during exercise. This protocol was chosen to simulate the way cyclists tend to drink during competition and also to create a constant supply of carbohydrates that potentially could lead to a plateau in the oxidation rate. Table 3 shows the calculated ingestion rates of carbohydrates via the small repeated boluses during the last 105 min of exercise, which we compared with the oxidation rates observed in the last 30-min period. In the case of the 4% maltodextrin solution, exogenous carbohydrates were oxidized at a rate that exceeded the rate of ingestion. This finding most likely indicates that part of the original larger bolus was still being oxidized and appeared in the breath in this period. In the case of the 8% maltodextrin and sucrose solutions, the rates of oxidation and ingestion were equal. If this situation were maintained when exercise is continued for ≥1 h, then a situation would exist in which oxidation and ingestion are in perfect balance. With the 12 and 16% maltodextrin solutions, more carbohydrates were ingested than oxidized (Table 2). This observation implies that carbohydrates were accumulating somewhere in the body. One possibility is that it accumulates in the stomach or gut. Rehrer et al. (28) showed that 30–40% of the ingested fluid and carbohydrates accumulated in the stomach at the end of 80 min of exercise at 70% $\text{V}_\text{O}_2\text{max}$ with the use of 17% glucose and 17% maltodextrin solutions and a comparable ingestion protocol as was used in this study. With a 4.5% glucose solution, this value was only 5%. To investigate the presence of gastrointestinal problems (4), we asked the subjects to indicate whether they suffered from full stomach feelings, belching, nausea, or gastrointestinal pain or cramps during and/or after exercise. With the 16% maltodextrin solution, five of six subjects suffered from a full stomach feeling and belching and one subject complained of nausea, whereas with the 4% maltodextrin solution only one subject complained of a full stomach feeling and two experienced belching. Intermediate scores were obtained with the 8 and 12% maltodextrin
solutions. With 8% sucrose, the only complaint was belching in two subjects.

Rehrer et al. (28) showed that only ~30% of the carbohydrate that was emptied from the stomach was oxidized with 17% carbohydrate solutions. This observation suggests that part of the missing carbohydrates was remaining in the intestine and/or was incorporated into endogenous pools in the body. In the latter case, provision of more carbohydrates could have advantages with respect to energy provision to the working skeletal muscle, especially in ultraendurance events. Glycogen synthesis has previously been reported to occur during exercise in subjects consuming a 25% maltodextrin solution (17) and in rats after oral ingestion of 20% glucose (16). It also cannot be excluded that fat synthesis occurs during exercise when large amounts of carbohydrates are taken. Changes in blood metabolites (2, 3; Fig. 1) and lipolytic hormones (3) indeed are such that they may stimulate lipogenesis during exercise more with carbohydrate ingestion than with water ingestion only.

In conclusion, this study shows that ingestion of ~50 g of maltodextrin or sucrose at the start of exercise followed by 12–13 g each 15 min leads to close to maximal oxidation rates of total and ingested carbohydrates and to a maximal sparing effect on the endogenous carbohydrate stores during 2 h of cycle exercise at 65% $W_{max}$ under laboratory conditions. Higher intake rates could have small advantages with respect to energy provision to the working skeletal muscle but may lead to gastrointestinal problems and distress due to accumulation of carbohydrates and fluid in the stomach (and maybe the intestine).

This work was supported by an Leostar Science grant from Sandoz Nutrition, Bern, Switzerland.

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Received 7 December 1992; accepted in final form 4 October 1993.

REFERENCES
23. PALIKARAKIS, N., B. JANDREINA, P. FRYNA, P. MOSORA, M. LACROIX, A. LACOR, AND P. LEFEBVREE. Remarkable metabolic avail-


