The liver directly through the portal vein, whereas long-chain triacylglycerols are not. These physical properties, MCTs are rapidly absorbed and enter the lymphatic system (9). Studies with stable isotopes have shown that medium-chain fatty acids are rapidly oxidized when the body is at rest (10) and during exercise (7, 11). We showed that carbohydrates coingested with MCTs accelerated the oxidation of MCTs during the first 90 min of exercise (7). By adding MCTs to a carbohydrate solution, the oxidation rates of these carbohydrates will not be impaired (8), and the MCTs can serve as additional fuel for the working muscle (6, 7). However, in recent studies we found that MCTs, although rapidly oxidized, contributed only marginally to total energy expenditure (6, 7) and did not spare muscle glycogen (8). In these studies, the amount of MCTs ingested was small (∼30 g) and no rise in plasma fatty acids was observed after ingestion.

Studies in which high plasma fatty acid concentrations were induced by giving either a high-fat meal or an intravenous infusion of a triacylglycerol emulsion (Intralipid; Kabi Pharmacia, Wördern, Germany) contributed only marginally to total energy expenditure (6, 7) and did not spare muscle glycogen (8). In these studies, the amount of MCTs ingested was small (∼30 g) and no rise in plasma fatty acids was observed after ingestion.

Accordingly, the aim of this study was to examine the metabolic effects of ingesting a large quantity of MCTs (85 g) both alone and in combination with carbohydrates compared with the effects of carbohydrate or water ingestion during 2 h of cycling. A second purpose was to investigate the effect of the ingestion of carbohydrates, MCTs, carbohydrates plus MCTs, or water on subsequent time-trial cycling performance.

KEY WORDS
Medium-chain triacylglycerol, exercise, maximal oxygen uptake, substrate utilization, carbohydrate, men.
SUBJECTS AND METHODS

Subjects

Seven endurance-trained male cyclists who regularly trained > 2 h/d, 4–7 d/wk, were recruited for this study. Five to seven days before the first experimental trial, \( V_{\text{O}2\text{max}} \), maximal work rate (Wmax), and peak heart rate (HRpeak) were determined in a stepwise incremental exercise test to exhaustion as described by Kuipers et al (16).

Maximal work test

After a warm-up period of 5 min at 100 W, the work rate was increased by 50 W every 2.5 min until a heart rate of 160 beats/min was reached. Then the work rate was increased by 25 W every 2.5 min. The Wmax (in W) was determined by the following equation:

\[
W_{\text{max}} = W_{\text{out}} + \left( \frac{t}{150 \text{ s}} \right) \times 25 \text{ Watts} \tag{1}
\]

where \( W_{\text{out}} \) is the last completed stage and \( t \) is the time (in s) in the final stage. All exercise tests were performed on an Excalibur Sport electromagnetically braked cycle ergometer (Lode, Groningen, Netherlands). Heart rate was measured continuously by a Polar Vantage heart rate monitor (Polar, Kempele, Finland). Mean \( \pm \) SEM \( V_{\text{O}2\text{max}}, W_{\text{max}}, \) and HRpeak were 74 ± 1 mL·kg\(^{-1}\)·min\(^{-1}\), 401 ± 17 W, and 187 ± 1 beats/min, respectively. The study was approved by the Maastricht University and Academic Hospital Maastricht Medical Ethical Committee.

Procedure

On the day of the test, subjects reported to the laboratory at 1200 after consuming a standardized breakfast in the morning. At the laboratory a standardized lunch was provided. At 1315, a polytetrafluoroethylene catheter (Baxter Quick Cath; Utrecht, Netherlands) was inserted into an antecubital vein and connected to a three-way stopcock (Discofix-3; Braun, Meisungen, Germany). At 1330, a resting blood sample was drawn. Resting breath gases were collected for the measurement of oxygen consumption and carbon dioxide production (Oxycon, Mt. Mijnhard, Netherlands). At 1355, subjects cycled for 5 min at 100 W as a warm-up. At 1400, subjects started cycling at 60% \( V_{\text{O}2\text{max}} \) for 2 h, and in the first minute they had to drink an initial bolus (8 mL/kg) of one of the test drinks. Thereafter, every 15 min a beverage volume of 2 mL/kg was provided. Blood samples (10 mL) were collected every 30 min and expiratory gases were collected every 15 min cycling at 75% of their maximal work load) as fast as possible. The measurement of performance was the trial of = 15 min duration was performed as described previously (17). Subjects were asked to perform a certain amount of work (equal to = 15 min cycling at 75% of their maximal work load) as fast as possible. The measurement of performance was the time to complete the set amount of work. This total amount of work was based on Wmax and was calculated as follows:

\[
\text{Total amount of work} = 0.75 \times W_{\text{max}} \times 900 \text{ s} \tag{2}
\]

For this trial, the ergometer was set in the pedaling rate dependent mode. In this mode, power varies with RPM according to the following formula:

\[
W = L \times (\text{RPM})^2 \tag{3}
\]

in which \( W \) is the work rate (in W), RPM is the pedaling rate, and \( L \) is a coefficient (\( W/\text{RPM}^2 \)) that is dependent on the subject's \( V_{\text{O}2\text{max}} \). \( L \) was chosen in such a way that, at a pedaling rate of 90 RPM, 70% Wmax was elicited. Thus, the coefficient \( L \) was calculated by solving equation 3 for \( L \) in terms of \( W_{\text{max}} \) and pedaling rate (ie, \( L = 0.70 W_{\text{max}} / (90 \text{ RPM})^2 = 0.70 W_{\text{max}}/8100 = 8.64 \times 10^{-5} W_{\text{max}} \)). The ergometer was connected to a computer (Apple Macintosh Powerbook 5300cs; software developed by L Wouters and A Jeukendrup) that recorded work rate and time on-line and calculated the amount of work performed as previously described (17). The only information the subjects received was the amount of work performed and the percentage of work performed relative to the preset amount of work (0% at the start and 100% at the end of the trial). Subjects did not receive information regarding work rate, pedaling rate, time, or heart rate. During all tests, environmental conditions were standardized, temperature was kept at 20°C, and an electric fan circulated air to minimize thermal stress. Subjects were encouraged by the same person in every test. This performance test has been shown to produce highly reproducible results: the CV of this test was 3.35% (17).

To avoid any influence of circadian variance, subjects always performed their tests at the same time of day. Subjects were not allowed to train on the day before the test. Two days before the test, training was not allowed to be exhaustive. Additionally, all subjects were instructed to eat the same evening meal, which was controlled by a registration procedure, before the test and were advised to eat a carbohydrate-rich meal such as pasta. So that we could further control carbohydrate and energy intake, subjects provided food records for the day before the test and for the day of the test.

Beverages

Subjects received four drinks on four different occasions, subsequent tests being separated by ≥ 5 d. The drinks consumed were 1) a 10% carbohydrate solution (CHO; 170 ± 6 g glucose), 2) a 10% carbohydrate solution with 5% MCTs (CHO + MCT; 170 ± 6 g glucose, 85 ± 3 g MCTs), 3) a 5% MCT solution (85 ± 3 g MCTs), or 4) a placebo consisting of artificially colored and flavored water. The carbohydrates in the CHO + MCT and CHO drinks consisted of corn-derived glucose (Amylum; Aalst, Belgium) and the 13C enrichment of these carbohydrates was −11.3 δ per mil versus Pee Dee Bellemnitella (PDB) standard as determined by combustion isotope-ratio mass spectrometry (Carlo Erba–Finnigan MAT 252; Bremen, Germany). Ninety-nine percent of the fatty acids in the MCT had a chain length of eight carbons (Triocanooate, Estasun GT8-99; Unichema, Barcelona, Spain). To each 1L of drink 5 g fructose, 730 mg citric acid, 160 mg aspartame, and 640 mg of a flavor substance (orange) were added to make the taste comparable in all trials. Subjects ingested 8 mL/kg of one of the drinks during the first minute of the warm-up period and 2 mL/kg every 15 min thereafter. Such a drinking pattern was reported previously to result in high rates of gastric emptying throughout exercise (18). Drinks were given in a randomized order and subjects were unaware of the content of the drink.

During the CHO and CHO + MCT trials, subjects ingested a glucose solution with a high natural abundance of 13C so that oral
glucose oxidation could be studied. The placebo trial was used to allow for correction for changes in breath $^{13}$CO$_2$ background enrichment during exercise. To avoid background shifts, standard procedures were followed (5–7, 19). Subjects were instructed not to consume any products with a high natural abundance of $^{13}$C during the entire experimental period. This was done to minimize a shift in background enrichment due to changes in endogenous substrate utilization. Furthermore, subjects were instructed to keep their diets as constant as possible on the days before the trials.

**Analysis**

Blood (5 mL) was collected into EDTA-containing tubes and centrifuged at 1500 × g at 4°C for 4 min. Aliquots of plasma were frozen immediately in liquid nitrogen and stored at −40°C until concentrations of glucose (Uni Kit III, 0710970; Hoffman-La Roche, Basel, Switzerland), lactate (20), β-hydroxybutyrate (21), fatty acids (Wako FFA-C test kit; Wako Chemicals, Neuss, Germany), and glycerol (GPO-trinder 337; Sigma Diagnostics, St Louis) were analyzed, with use of the COBAS BIO semi automatic analyzer (Hoffman-La Roche). From indirect calorimetry (respiratory quotient and VO$_2$) and stable isotope measurements ($^{13}$CO$_2$/12CO$_2$; continuous flow IRMS; Finnigan MAT 252, Bremen, Germany), total energy expenditure and oxidation rates of total fat, total carbohydrate, and exogenous glucose were calculated.

**Calculations**

From carbon dioxide production ($\dot{V}$CO$_2$) and VO$_2$, carbohydrate and fat oxidation rates were calculated by using stoichiometric equations (22).

\[ \text{Glucose oxidation} = 4.585 \dot{V}\text{CO}_2 - 3.226 \dot{V}\text{O}_2 \]  
\[ \text{Fat oxidation} = 1.695 \dot{V}\text{O}_2 - 1.701 \dot{V}\text{CO}_2 \]

The isotopic enrichment was expressed as the δ per mil difference between the $^{13}$C and $^{12}$C ratios of the sample and a known laboratory reference standard according to the formula of Craig (23):

\[ \delta^{13}\text{C per mil} = \left[ \frac{\text{[C}/\text{C\text{standard}}} - 1 \right] \times 10^3 \]

The δ $^{13}$C was then related to the international standard Pee Dee Bellemnitella (PDB-1).

The amount of glucose oxidized was calculated according to the formula:

\[ \text{Exogenous glucose oxidation} = \dot{V}\text{CO}_2 \times \left( \frac{\delta\text{Exp} - \delta\text{bkg}}{\delta\text{ing} - \delta\text{bkg}} k \right) \]

in which δbkg is the $^{13}$C enrichment of expired air in the background trial (the placebo served as the background correction trial), δExp is the $^{13}$C enrichment of expired air during exercise at different time points, δing is the $^{13}$C enrichment of the ingested glucose, and k is the amount of CO$_2$ (in L) produced by the oxidation of 1 g glucose ($k = 0.7467$ L CO$_2$/g glucose).

Endogenous glycogen utilization was calculated as follows:

\[ \text{Endogenous glycogen utilization} = \frac{\text{total carbohydrate oxidation} - \text{exogenous glucose oxidation}}{\text{exogenous glucose oxidation}} \]

**RESULTS**

**Time-trial cycling performance**

Times to complete the preset amount of work in the four trials are presented in Table 1 and were not significantly different among the CHO, CHO + MCT, and placebo trials. The time to accomplish the preset amount of work in the MCT trial was significantly longer than in the other trials. There was no effect of test order. Average work rate during the time trial was 17–18% lower in the MCT trial than in the other trials (Table 1). The mean heart rate was also significantly lower during the MCT trial than in the other trials, in which the mean heart rates were comparable. No statistical differences were observed in peak heart rate during the time trial or in ratings of perceived exertion.

**Blood indexes**

During the 120 min of steady state exercise, plasma glucose concentrations initially rose from resting values of 4.6–5.1 mmol/L to 5.8 mmol/L during the CHO and CHO + MCT trials, whereas in both the placebo and MCT trials plasma glucose concentrations tended to decline (Figure 1). Plasma glucose concentrations during the MCT trials (and the placebo trial at 60 min) were significantly ($P < 0.05$) lower during the 2 h of exercise than were the concentrations during the two CHO trials (CHO and CHO + MCT). Plasma glucose concentrations increased during the time trial but this increase was significant only in the MCT and placebo trials (Figure 1).

Plasma lactate concentrations during the 120 min of exercise were stable and similar in all trials, and average values were < 1 mmol/L (0.7–0.9 mmol/L; Figure 1). Plasma lactate increased during the time trial to high concentrations during the CHO, CHO + MCT, and placebo trials (8.0–10.3 mmol/L) and increased less during the MCT trials (3.6 mmol/L; Figure 1).

Plasma fatty acid concentrations increased from resting values of between 213 and 292 mmol/L to 406 ± 87 mmol/L during CHO trials, to 506 ± 87 mmol/L during the CHO + MCT trials, and to values > 700 mmol/L at the end of 2 h of exercise when no carbohydrates were ingested (722 ± 146 mmol/L during the
placebo trials and 820 ± 134 mmol/L during the MCT trials). During exercise, plasma fatty acid concentrations were significantly higher ($P < 0.05$) during the MCT trials than during the CHO or CHO + MCT trials (except at 120 min). Also, during the time trial plasma fatty acids were higher in the MCT trial than in the other trials (Figure 1).

Glycerol concentrations rose gradually during exercise in all trials. Although glycerol concentrations seemed to be somewhat lower by the end of 2 h of exercise in the CHO trials, this difference was not statistically significant. Only at 120 min were glycerol concentrations during the placebo trials significantly ($P < 0.05$) higher than during the CHO or CHO + MCT trials. During the time trials, glycerol concentrations further increased and concentrations remained lower in the CHO feeding trials ($P < 0.05$; Figure 1).

β-Hydroxybutyrate concentrations showed a small rise from 200–250 to 222–380 μmol/L during the CHO, CHO + MCT, and placebo trials but increased markedly during the MCT trials (to

<table>
<thead>
<tr>
<th>Trial</th>
<th>Duration</th>
<th>Average work rate</th>
<th>Mean HR</th>
<th>Max HR</th>
<th>RPE$^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHO</td>
<td>14.18 ± 0.57</td>
<td>314.4 ± 18.5</td>
<td>180 ± 2</td>
<td>187 ± 2</td>
<td>9.14 ± 0.34</td>
</tr>
<tr>
<td>CHO + MCT</td>
<td>14.02 ± 0.32</td>
<td>313.9 ± 13.0</td>
<td>181 ± 2</td>
<td>187 ± 2</td>
<td>9.29 ± 0.29</td>
</tr>
<tr>
<td>MCT</td>
<td>17.33 ± 1.10$^4$</td>
<td>263.1 ± 22.4$^4$</td>
<td>170 ± 4$^4$</td>
<td>182 ± 6</td>
<td>9.00 ± 0.44</td>
</tr>
<tr>
<td>PLAC</td>
<td>14.43 ± 0.70</td>
<td>311.6 ± 17.8</td>
<td>182 ± 2</td>
<td>187 ± 2</td>
<td>8.57 ± 0.81</td>
</tr>
</tbody>
</table>

$^1 \bar{x} ± SEM; \ n = 7$. bpm, beats per minute; CHO, glucose solution; CHO + MCT, glucose and medium-chain triacylglycerol solution; MCT, medium-chain triacylglycerol solution; PLAC, artificially colored and flavored water solution.

$^2$ Scored from 1 to 10.

$^4$ Significantly different from the other trials, $P < 0.05$. 

**FIGURE 1.** Plasma glucose, lactate, fatty acids, glycerol, and β-hydroxybutyrate during 120 min of exercise at 60% of maximal oxygen uptake ($\bar{V}O_2$max) followed by a time trial (<15 min) with ingestion of a 10% carbohydrate solution (CHO), a 10% carbohydrate solution with 5% medium-chain triacylglycerols (CHO + MCT), a 5% MCT solution (MCT), or a placebo consisting of artificially colored and flavored water (PLAC). $\bar{x} ± SEM; \ n = 7$. Means with different superscript letters are significantly different ($P < 0.05$) as follows: a, significant difference between CHO and MCT; b, significant difference between CHO + MCT and MCT; c, significant difference between CHO and placebo; d, significant difference between CHO + MCT and placebo; e, significant difference between MCT and placebo; and f, significant difference between CHO and CHO + MCT.
883 μmol/L; P < 0.05). During the time trial, β-hydroxybutyrate concentrations also significantly increased (to 1.1–1.7 mmol/L; Figure 1).

Indirect calorimetry

During the second hour of exercise, R values averaged 0.88–0.91 in the CHO and CHO + MCT trials and were significantly lower in the MCT and placebo (0.83–0.88) trials. Carbohydrate ingestion reduced fat oxidation in both the CHO and CHO + MCT trials compared with the placebo and MCT trials and maintained carbohydrate oxidation rates of > 2 g/min (Figure 2). Without carbohydrate ingestion, carbohydrate oxidation rates decreased to 1.5 g/min (Figure 2). No differences in carbohydrate or fat oxidation were observed between the CHO and CHO + MCT trials or between the placebo and MCT trials.

Exogenous glucose oxidation

Background 13C enrichment measured from the resting breath samples was −26.1 ± 0.2 δ per mil versus PDB-1. With ingestion of the corn-derived glucose in the CHO and CHO + MCT trial, the rise in 13C was significant, reaching a δ per mil versus PDB-1 difference of ≈3.5 toward the end of 120 min of exercise (compared with resting breath sample). The changes in background enrichment during exercise in the placebo trial were ≈10% of the 13C enrichment provoked by the exogenous glucose in the CHO and CHO + MCT trials. Therefore, a background correction was made for the calculation of exogenous glucose oxidation, by using the data from the placebo trial. No differences were observed in background enrichment between MCT and placebo. Exogenous glucose oxidation showed a gradual increase over time and leveled off after ≈75 min (Figure 3).

Maximal oxidation rates were 0.81 ± 0.03 g/min in the CHO trials and 0.78 ± 0.03 g/min in the CHO + MCT trials. No differences in oral carbohydrate oxidation between the CHO and CHO + MCT trials were observed. Endogenous carbohydrate (liver and muscle glycogen) oxidation rates as calculated from total carbohydrate oxidation minus exogenous glucose oxidation were similar in the CHO and CHO + MCT trials (Figure 3).

Gastrointestinal discomfort

Gastrointestinal complaints were registered by means of a questionnaire. Selected complaints are displayed in Table 2. Vomiting and diarrhea were not reported during exercise. Two subjects vomited after the MCT trial and three complained of diarrhea afterward. Nausea, headache, and dizziness were not significantly different among the trials. Belching and a bloated feeling in the stomach were more often reported when the CHO, CHO + MCT, or MCT beverage was ingested compared with the placebo. Gastrointestinal cramps occurred significantly more often when the MCT beverage was ingested, and this was the most serious complaint reported during exercise.

DISCUSSION

Fat supplementation during exercise is believed to be undesirable for several reasons. First, endogenous fat stores are very large and therefore fat ingested during exercise would not add appreciable triacylglycerol to the total body content. Second, the digestion and absorption of fat is slow and the appearance of ingested long-chain triacylglycerols in the bloodstream may take 3–4 h. Dietary fat contains long-chain triacylglycerols that, after being hydrolyzed first in the intestine, diffuse as long-chain fatty acids into the intestinal mucosa cells for reesterification to triacylglycerols. These long-chain triacylglycerols are then incorporated into chylomicrons and are transported through the lymphatic system, which ultimately drains them into the systemic circulation. Third, fat will reach the circulation in chylomicrons, which are generally believed to not be a major energy source for the working muscle. Aside from this, long-chain triacylglycerols are known to be potent inhibitors of gastric emptying. For these reasons, fat supplementation during exercise is not recommended.

The effects of MCTs, however, differ from those of long-chain triacylglycerols. It has been shown that MCTs do not slow gastric emptying (24), are rapidly hydrolyzed and absorbed, and enter the systemic circulation directly through the portal vein (9). It has also been shown that ingested MCTs are oxidized at high rates relative to their rate of ingestion (6, 7, 11, 25). In addition to this, we recently showed that MCTs coingested with carbohydrates were more rapidly oxidized (ie, they reached maximal MCT oxidation rates more quickly) than were MCTs alone (7). Furthermore, we showed that 30 g MCT coingested with carbohydrates did not significantly affect exogenous carbohydrate oxidation during 3 h of exercise at ≈60% VO2 max (8).
Only a few studies have investigated the effect of MCT ingestion on exercise performance. Satabin et al (26) studied the effect of MCTs ingested before exercise on exercise capacity and found no effect of a single 45-g bolus of MCTs or an equienergetic amount of glucose on time to fatigue at 60% $\dot{V}O_2\text{max}$. Recently, van Zeyl et al (15) reasoned that the amount of MCTs provided was too small to detect performance effects of MCT ingestion and they gave their subjects 86 g of an MCT solution in repeated small boluses during exercise. After 120 min of submaximal exercise at 60% $\dot{V}O_2\text{max}$, their subjects performed a simulated 40-km time trial. Performance decreased with MCT ingestion compared with carbohydrate ingestion, but simultaneous ingestion of carbohydrates and MCTs resulted in increased performance compared with ingestion of carbohydrates alone (ie, average speed during the time trial). One of these investigators’ hypotheses was that ingestion of carbohydrates in conjunction with MCTs improved performance compared with ingestion of carbohydrates alone because of an increased availability of plasma fatty acids and a subsequent sparing of muscle glycogen (15). Such effects have been suggested to favorably influence exercise capacity (27, 28). However, the study by van Zeyl et al (15) included no placebo control group; therefore, the separate effect of MCTs compared with control conditions was not investigated. Thus, the present study was aimed at investigating the effect on time-trial cycling performance of ingestion of large amounts of MCTs (85 g) with or without carbohydrates compared with ingestion of carbohydrates alone or a placebo. As in the study of van Zeyl et al (15), drinks were ingested during 2 h of submaximal exercise at 60% $\dot{V}O_2\text{max}$ and thereafter a time trial was performed. Interestingly, we found no differences in performance when subjects ingested CHO, CHO + MCT, or placebo. However, when subjects ingested MCTs, performance was significantly decreased.

One of the possible explanations for the fact that in this study MCTs added to a glucose solution did not have the positive effect reported by van Zeyl et al (15) is the difference in the duration of the time trial. We used a 15-min time trial in contrast with the 60-min time trial used by van Zeyl et al (15); we chose this duration after pilot studies showed that subjects developed serious gastrointestinal discomfort when the time trial lasted

### TABLE 2

Gastrointestinal (GI) discomfort after 120 min of exercise at 60% $\dot{V}O_2\text{max}$ (ie, the start of the time trial) and directly after the time trial

<table>
<thead>
<tr>
<th>Beverage</th>
<th>Nausea</th>
<th>Belching</th>
<th>Bloating feeling</th>
<th>Headache, dizziness, or both</th>
<th>Gastrointestinal cramping</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHO</td>
<td>1.0 ± 1.0</td>
<td>1.4 ± 1.0⁡</td>
<td>1.4 ± 1.4⁡</td>
<td>0.7 ± 0.7</td>
<td>0.0 ± 0.0</td>
</tr>
<tr>
<td>CHO + MCT</td>
<td>1.0 ± 1.0</td>
<td>0.0 ± 0.0</td>
<td>1.4 ± 1.4⁡</td>
<td>1.0 ± 0.1</td>
<td>0.0 ± 0.0</td>
</tr>
<tr>
<td>MCT</td>
<td>1.3 ± 1.2</td>
<td>1.2 ± 0.7⁡</td>
<td>2.5 ± 1.5⁡</td>
<td>1.5 ± 0.9</td>
<td>2.0 ± 1.3³⁴</td>
</tr>
<tr>
<td>PLAC</td>
<td>0.1 ± 0.1</td>
<td>0.3 ± 0.3⁡</td>
<td>1.7 ± 1.1⁡</td>
<td>1.3 ± 1.2</td>
<td>1.8 ± 1.2³⁴</td>
</tr>
</tbody>
</table>

1 Values are average scores on a questionnaire given at completion of the time trial, in which on a 10-point scale 1 was “no complaints” and 10 was “very serious complaints.” x ± SEM; n = 7. CHO, glucose solution; CHO + MCT, glucose and medium-chain triacylglycerol solution; MCT, medium-chain triacylglycerol solution; PLAC, artificially colored and flavored water solution.

2 Significantly different from PLAC, P < 0.05.

3 Significantly different from CHO, P < 0.05.

FIGURE 3. Exogenous carbohydrate oxidation during 120 min of exercise at 60% of maximal oxygen uptake ($\dot{V}O_2\text{max}$) with ingestion of a 10% carbohydrate solution (CHO) and a 10% carbohydrate solution with 5% medium-chain triacylglycerols (CHO + MCT) and endogenous carbohydrate oxidation with ingestion of CHO, CHO + MCT, MCT (5% MCT solution), or PLAC (placebo consisting of artificially colored and flavored water). x ± SEM, n = 7.
longer than 15 min. However, the test-to-test CV of a time trial lasting ≈1 h was similar to the CV of a 15-min time trial (17). Both performance tests have been shown to be highly reproducible (17).

Other differences between the study by van Zeyl et al (15) and ours may include the type of carbohydrate used (short-chain glucose polymers rather than glucose), the type of MCT used (a mixture of C8 and C10 rather than pure C8), and the time of the day at which experiments were performed (morning rather than early afternoon). How these differences would explain the different findings, however, is unclear.

The most likely explanation for the differences between the study by van Zeyl et al (15) and the present study may be the differences in gastrointestinal discomfort. In this study, subjects more often reported gastrointestinal cramping during the MCT trials, which was negatively correlated to their performance. Because of this gastrointestinal discomfort, subjects reported that they could not push as hard as they could without gastrointestinal discomfort. This is supported by a lower mean heart rate (Table 1) and lower plasma lactate and β-hydroxybutyrate concentrations (Figure 1) during the MCT trials, which are the results of a lower absolute work rate (Table 1). Similar gastrointestinal complaints were reported by Ivy et al (29). The placebo drinks provoked the least gastrointestinal discomfort, whereas the CHO drinks, given in these relatively large quantities (on average 85 g/h), resulted in a bloated feeling. This may be the explanation for the lack of performance effect after ingestion of CHO drinks. Van Zeyl et al (15) however, did not report major gastrointestinal problems during exercise when carbohydrates and MCTs were ingested.

Interestingly, we did not observe a positive effect on time-trial performance of carbohydrates compared with placebos. Previously, we showed that carbohydrate ingestion during exercise may improve time trial performance lasting ≈1 h (3). Here the time trial was relatively short (14 min), which may have prevented a performance effect. However, we strongly feel that the bloated feeling as reported by the subjects after CHO ingestion was the major reason for the lack of time-trial performance improvement. The amount of carbohydrates provided was 75 g/h in the previous trials for 1-h trials (3), whereas it was 170 g/h in the present study for a 2-h trial. This larger total amount of carbohydrates ingested may be responsible for the bloated feeling reported by the subjects and the lack of a performance effect compared with the placebo. However, other factors, such as the short duration of the performance trial or the small number of subjects (n = 6) may also have played a role in the lack of a significant performance effect of carbohydrates compared with the placebo.

In the present study, the exercise intensity increased drastically during the time trial compared with the 120 min of submaximal exercise at 60% VO2max. Glycogenolysis was stimulated and lactate was formed and accumulated in plasma (Figure 1). Plasma glucose concentrations increased (significantly so in the MCT and placebo trials) as a result of the high exercise intensity. This was shown before and was attributed to increased circulating plasma epinephrine concentrations, which increase hepatic glucose production (30). Apparently, the availability of acetyl-CoA units in the liver increased more than the utilization that caused the large increase in plasma β-hydroxybutyrate concentration. Responses seemed to be somewhat less pronounced in the MCT trial, in which the absolute work rate was somewhat less.

As reported previously (1, 7, 31), carbohydrate ingestion markedly blunted plasma fatty acid and total fat oxidation and maintained high rates of carbohydrate oxidation. Although the amount of MCTs ingested was threefold higher than in previous studies from our lab (6–8) and by others (11, 25, 29), in agreement with these studies, we found no significant differences in total fat or carbohydrate utilization with or without MCT ingestion (Figure 2). Although statistically not significant, however, fat oxidation seemed to be slightly higher with ingestion of carbohydrates in combination with MCTs than with ingestion of carbohydrates alone. Because we did not measure exogenous MCT oxidation or plasma medium-chain fatty acid concentrations in this study, it cannot be determined whether this higher fat oxidation is the result of MCT oxidation or a reduced suppression of endogenous fat oxidation induced by the ingested carbohydrates.

Increases in plasma fatty acids and glycerol were blunted by carbohydrate ingestion, but MCT ingestion had no effect on plasma fatty acid or glycerol concentrations. We reported before that glycerol concentrations were not significantly elevated after MCT ingestion and we suggested that this was due to the capacity of the liver to use glycerol for gluconeogenesis (7).

Also, in agreement with previous findings (8), MCTs did not influence exogenous or endogenous carbohydrate oxidation rates. It has been suggested that MCT ingestion may elevate the plasma fatty acid concentration and subsequently spare muscle glycogen (12–14). However, several studies have shown that ingestion of 25–30 g MCTs did not lower the rate of muscle glycogen breakdown (8, 25, 33). Recently, van Zeyl et al (15) suggested that the amount of MCTs in the above mentioned studies was too small to affect muscle glycogen breakdown. Therefore, they gave their subjects large amounts of MCTs; tracer data suggested that glycogen breakdown was reduced. However, these data need to be interpreted with caution. MCT feeding elevated plasma ketone concentrations to high levels and it is known that ketogenesis may fixate oxygen in the production of β-hydroxybutyrate. Because the estimates of glycogen breakdown are dependent on VCO2 and VCO2, this may have decreased the calculated carbohydrate oxidation rates in the MCT trial.

Although we did not measure muscle glycogen concentrations, tracer methods did not show a sparing of endogenous carbohydrate stores (in the liver and in muscle glycogen) after MCT ingestion (8). Because van Zeyl et al (15) reported no differences in plasma glucose oxidation with ingestion of MCTs, it is unlikely that we would have observed glycogen sparing in this study, as total carbohydrate oxidation rates were similar.

In conclusion, the present study showed that large amounts of MCTs (85 g) coingested with carbohydrates did not significantly affect total rates of carbohydrate and fat oxidation and had no positive effect on performance. When only MCTs were ingested, performance decreased by 17–18%, probably as a result of gastrointestinal distress.

REFERENCES


