Effect of carbohydrate or carbohydrate plus medium-chain triglyceride ingestion on cycling time trial performance

DAMIENT H. ANGUS, MARK HARGREAVES, J. DANCEY, AND MARK A. FEBBRAIO
1Exercise Physiology and Metabolism Laboratory, Department of Physiology, The University of Melbourne, Parkville, Victoria 3052; and 2School of Health Sciences, Deakin University, Burwood, Victoria 3125, Australia

Angus, Damien J., Mark Hargreaves, J. Dancey, and Mark A. Febbraio. Effect of carbohydrate or carbohydrate plus medium-chain triglycerides on metabolism and cycling performance. J. Appl. Physiol. 88: 113–119, 2000.—This study examined the effectiveness of ingesting a carbohydrate or carbohydrate + medium-chain triglycerides (MCT) on metabolism and cycling performance. Eight endurance-trained men [peak O2 uptake \(\dot{V}O_2{\text{peak}}\) \(= 4.71 \pm 0.09\) (SE) \(\text{L/min}\)] completed a set amount of work in both C and C+M compared with P by 7 and 5%, respectively (C: 166 \pm 7 min; C+M: 169 \pm 7 min; P: 178 \pm 11 min; \(P < 0.01\)). Plasma glucose concentration was maintained at or above resting values throughout both C and C+M trials but decreased \((P < 0.05)\) below resting values in P at the completion of the TT. The estimated rate of carbohydrate oxidation was not different during the first 90 min of exercise but thereafter was reduced \((P < 0.05)\) in P compared with both C and C+M. These data demonstrate that carbohydrate ingestion during exercise improves 100-km TT performance compared with a sweet placebo, but the addition of MCT does not provide any further performance enhancement.

endurance exercise; glucose

**DURING PROLONGED ENDURANCE exercise, the body must carefully manage the balance between substrate availability and fuel utilization. Liver and muscle glycogen reserves are relatively small, and depletion of these stores has been associated with fatigue during constant-load exercise (3, 7). It is not surprising that research has focused on carbohydrate (CHO)-feeding strategies during exercise, which have been shown to maintain rates of CHO oxidation, prevent hypoglycemia, and significantly increase exercise performance (3–5, 7, 8, 22–24). In some previous studies, the measure of exercise performance was the time taken for a subject to be unable to maintain the desired power output (3–5, 7, 8). This measure of exercise performance does not accurately reflect a conventional competitive environ-**
ing 2 h of exercise at 65% peak O₂ uptake (V₀₂peak), ingested MCT could be oxidized and contributed a similar proportion to total energy expenditure compared with an isocaloric CHO beverage. During the last hour of a 3-h cycling bout at 50% maximal work rate, it was found that when MCT were ingested with CHO the contribution of oxidized MCT to total energy expenditure increased from 3.2 to 6.4% (16).

Only two studies have examined MCT and CHO feeding during exercise and subsequent TT performance. Van Zyl et al. (28) found that ingestion of a 4.3% MCT + 10% CHO beverage during 2 h of exercise at 60% V₀₂peak improved performance in a subsequent simulated 40-km TT, compared with a CHO solution alone. The authors observed that ingestion of large amounts (~85 g) of MCT during the steady-state exercise period increased free fatty acid (FFA) concentration, reduced CHO oxidation, and spared muscle glycogen stores, resulting in the improved TT performance. In contrast, a recent study (17) adopted a similar research design and observed neither significance of GI cramping, or improvement in subsequent exercise performance with MCT + CHO administration. The purpose of the present study was to compare the ingestion of a CHO, or a CHO+MCT solution, with a sweet placebo during a simulated 100-km (2.5- to 3-h) cycling TT. This proposed protocol would determine whether the favorable performance benefits of MCT+CHO ingestion could be replicated when subjects were required to engage in the cycling TT for the entire experimental trial, rather than subsequent to a bout of lower intensity, steady-state exercise. Despite MCT-containing products now being commercially available, there is no study to date that has examined the effectiveness of this purported ergogenic aid at sustained exercise intensities similar to those in competitive endurance events. Furthermore, GI distress has been associated with MCT ingestion (14, 17), and this may adversely effect TT performance, because high-intensity exercise (>70% V₀₂peak) reduces blood flow to the splanchic and GI tract (26). We reasoned that CHO feeding would produce faster TT performances but that addition of MCT to the ingested beverage would not produce any further augmentation in exercise performance.

METHODS

Subjects. Eight male endurance-trained cyclists/triathletes [22 ± 0.5 yr; 72 ± 2 kg; 177 ± 1 cm; V₀₂peak = 4.71 ± 0.09 (SE) l/min] volunteered to serve as subjects for the investigation, which had been approved by The University of Melbourne Human Research Ethics Committee. Subjects were made fully aware of the procedures and risks associated with the study, both verbally and in writing. All subjects completed a medical questionnaire and provided written informed consent. To determine VO₂peak, each subject performed incremental cycling (Lode, Groningen, The Netherlands) to volitional fatigue at least 7 days before the first experimental trial.

Preexperimental protocol. Subjects were provided with a food parcel (~15.6 MJ), 71% CHO, 15% protein, 14% fat) for the 24 h before an experimental trial. They were instructed to adhere to the diet, consume water ad libitum, and abstain from exercise, alcohol, tobacco, and caffeine in this period. On the morning of a trial, subjects arrived at the laboratory 2 h after a breakfast (1.3 MJ, 66% CHO) similar to one that is normally ingested before competition (2 muesli bars, 250 ml of orange juice, and 300 ml of tap water). We found that these pretrial exercise and lifestyle controls resulted in reproducible metabolite and hormonal levels in all subjects before each experimental trial (see baseline values).

Experimental trials. Subjects were weighed on arrival in the laboratory, and a catheter (Turumo, Toyoko, Japan) was inserted into an antecubital vein of one forearm for the collection of blood samples. The weight recorded during the first trial was used to calculate the required work for all trials. After a 5-min warm-up at a fixed workload of 200 W, the ergometer was placed in a mode at which power output was dependent on pedal frequency. In this ergometer mode, an increase in cycling cadence resulted in a proportional elevation in the required workload. Subjects were required to complete 35 kJ/kg of body weight “as quickly as possible” in each trial. Total work completed by the subject was recorded every 15 min during the TT. To replicate a competitive environment in the laboratory, subjects were rewarded with prizes for low aggregate TT times as an alternative incentive to completing the exercise trials in the shortest possible time. In addition, care was taken to ensure that each subject was encouraged to a similar extent by the same investigator in each trial. All trials were performed in mild environmental conditions (20–22°C), and an electric fan circulated air to minimize thermal stress. Subjects were provided with 250 ml of the test beverage at the start of exercise, and a further 250 ml every 15 min until completion of the TT.

Test beverages. The beverage provided to the subjects consisted of either a 6% (wt/vol) CHO solution (C; Gatorade, Quaker Oats); a 6% CHO plus a 4.3% MCT (C+M; Mead Johnson) solution; or a sweet placebo (P; defizzed diet soft drink). The MCT liquid comprised triglycerides containing 71% caprylic (C₈) and 23% capric (C₁₀) medium-chain fatty acids. All test solutions were lemon flavored (to mask the addition of CHO and MCT), well mixed, and devoid of carbonation. Beverages were administered in opaque containers in a double-blind and randomized fashion for the three experimental trials.

Heart rate and gas-exchange measurements. Every 30 min during exercise, heart rate (Electro, Polar) was recorded and expired gas was collected into two Douglas bags. Expired-gas samples were analyzed for oxygen and carbon dioxide (Applied Electrochemistry 5-3A/I and CD-3A, Ametek, Pittsburgh, PA) concentration. These analyzers were calibrated by using commercial gases of known composition. The volume of expired air was measured on a Parkinson-Cowan gas meter calibrated against a Tissot spirometer. Determination of oxygen uptake (VO₂), RER, and ventilation was performed by using conventional equations. Assuming a nonprotein respiratory quotient (25), an estimation of the whole body CHO and fat oxidation was calculated by indirect calorimetry by using the following equations

\[ \text{CHO oxidation} = 4.585 \cdot \dot{V}_{\text{CO}_2} - 3.226 \cdot \dot{V}_{\text{O}_2} \]

\[ \text{Fat oxidation} = 1.695 \cdot \dot{V}_{\text{O}_2} - 1.701 \cdot \dot{V}_{\text{CO}_2} \]

where \( \dot{V}_{\text{CO}_2} \) is carbon dioxide production.

Blood metabolite and hormone analysis. A blood sample was obtained at rest, every 30 min during exercise, and at the completion of the TT to measure plasma glucose, lactate, FFA, and insulin. An aliquot (~1.5 ml) of the blood sample
was placed into a tube containing a preservative (EGTA and GSH). The plasma was separated by centrifugation and frozen at −20°C for later analysis of FFA concentration by a spectrophotometric assay (FFA-C test kit, Wako Chemicals, Neuss, Germany). The remaining whole blood was placed in fluoride heparin tubes, spun to extract plasma, and stored frozen for later analysis of plasma glucose, using an automated glucose oxidase method (YSI 2300, Yellow Springs, OH). Plasma insulin was measured by radioimmunoassay (Incstar, Stillwater, MN), whereas plasma for lactate determination was deproteinized in 8% perchloric acid, spun, and stored frozen for analysis by using a standard enzymatic technique (18).

Statistical analysis. The data from the three trials were compared by using a two-factor (time and treatment) ANOVA with repeated measures. A one-way ANOVA was used to compare TT completion times, with significance set at P < 0.05. Specific differences were located by using the Student-Newman-Keuls post hoc test when ANOVA revealed a significant interaction. A Biomedical data processing software package was used to compare these statistics, and comparative data are reported as means ± SE.

RESULTS

When subjects consumed either of the two CHO-containing beverages (C or M), the time to complete the simulated 100-km TT was reduced (P < 0.05) by 7 and 5%, respectively, compared with P (C: 166 ± 7 min; M: 169 ± 7 min; P: 178 ± 11 min; Fig. 1). Subjects maintained an average power output of 251 ± 1 W for the first 135 min of the TT, irrespective of the beverage consumed (Fig. 1). In the final portion of the TT, however, power output decreased (P < 0.05) to 212 ± 18 W in P, while being maintained in both C and M (257 ± 7 and 252 ± 8 W, respectively). As reported in Table 1, the first 2 h of exercise required an average VO2 of 3.5 ± 0.11/l/min in all trials. This equates to 75 ± 1% of VO2peak. A final respiratory measurement was obtained near the end of the TT (average 158 min) and revealed a reduced VO2 in the P trial (P < 0.05), which was maintained in both C and C+M trials (Table 1). Likewise, heart rate was similar in all trials, with the exception of the final measurement, where HR was lower (P < 0.05) in the P trial than in both C and C+M trials (Table 1). RER declined (P < 0.05) in all trials, irrespective of the beverage consumed (Table 1). The rate of decline, however, was different (P < 0.05) among trials, with final measurements in P being lower (P < 0.05) than in C and C+M (0.84 ± 0.02, 0.91 ± 0.01, and 0.90 ± 0.01 for P, C, and C+M, respectively)

Rates of whole body CHO oxidation were −3.5–4.0 g/min for the first 90 min of exercise and were not different among trials (Fig. 2). Thereafter, CHO oxidation was lower (P < 0.05) in the P trial, compared with in both the C and C+M trials (Fig. 2). Whole body fat oxidation (Fig. 2) was found to increase over the course of the TT in all trials (P < 0.05). In addition, there was a significant treatment effect observed (P < 0.05), with a higher rate of fat oxidation measured in P compared with either C or C+M, but no specific time and treatment interaction (P = 0.12).

Plasma glucose concentration increased (P < 0.05) at the onset of exercise in all trials (Fig. 3). After 30 min of exercise, and for the remainder of the trial, plasma glucose concentration in C was higher (P < 0.05) compared with in P. Similarly, plasma glucose concentration was higher (P < 0.05) in C+M than in P after 60 min, 120 min, and at the completion of the TT. At the end of the TT, plasma glucose concentration was higher in C+M than in C (P < 0.05). In the P trial, plasma glucose concentrations steadily decreased (P < 0.05) throughout the TT, reaching a nadir of 3.9 ± 0.2 mmol/l, a value lower (P < 0.05) than that measured before exercise. Plasma lactate levels increased (P < 0.05) after 30 min of exercise, to an average of 4.6 ± 0.3 mmol/l (Fig. 3). For the following 90 min, plasma lactate was maintained at an average of 3.7 ± 0.1 mmol/l, with an increased (P < 0.05) lactate concentration at the completion of the TT with all beverages tested. There was no significant effect of beverage type on plasma lactate concentration.

Plasma FFA concentrations increased during the TT to a similar extent in all three trials (Fig. 4; P < 0.05).
After C, plasma FFA concentrations were lower ($P < 0.05$) than in the P trial from 120 min to the end of the TT ($P < 0.05$). Similarly, after C1M, plasma FFA concentrations were lower ($P < 0.05$) than in the P trial from 90 min until TT completion ($P < 0.05$). Plasma insulin concentrations fell ($P < 0.05$) during exercise in all trials (Fig. 4; $P < 0.05$). In the P trial, plasma insulin concentrations were lower ($P < 0.05$) throughout the TT, compared with in C and C1M.

DISCUSSION

The major finding of this study was that ingestion of a 6% CHO beverage improved simulated 100-km TT.

Table 1. Heart rate, oxygen uptake, and RER during a simulated 100-km TT when consuming a placebo, carbohydrate, or carbohydrate and medium-chain triglyceride beverage

<table>
<thead>
<tr>
<th></th>
<th>30 min</th>
<th>60 min</th>
<th>90 min</th>
<th>120 min</th>
<th>Final</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, beats/min</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>162 ± 5</td>
<td>161 ± 5</td>
<td>163 ± 3</td>
<td>162 ± 3</td>
<td>158 ± 4</td>
</tr>
<tr>
<td>C</td>
<td>164 ± 3</td>
<td>164 ± 3</td>
<td>164 ± 4</td>
<td>165 ± 5</td>
<td>174 ± 3*</td>
</tr>
<tr>
<td>C + M</td>
<td>165 ± 3</td>
<td>165 ± 3</td>
<td>165 ± 3</td>
<td>167 ± 3</td>
<td>175 ± 4*</td>
</tr>
<tr>
<td>$\dot{V}O_2$, l/min</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>3.5 ± 0.1</td>
<td>3.5 ± 0.1</td>
<td>3.6 ± 0.1</td>
<td>3.5 ± 0.1</td>
<td>3.0 ± 0.2</td>
</tr>
<tr>
<td>C</td>
<td>3.5 ± 0.1</td>
<td>3.5 ± 0.1</td>
<td>3.5 ± 0.1</td>
<td>3.5 ± 0.1</td>
<td>3.8 ± 0.1*</td>
</tr>
<tr>
<td>C + M</td>
<td>3.5 ± 0.1</td>
<td>3.5 ± 0.1</td>
<td>3.5 ± 0.1</td>
<td>3.6 ± 0.1</td>
<td>3.7 ± 0.1*</td>
</tr>
<tr>
<td>RER</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>0.94 ± 0.01</td>
<td>0.93 ± 0.01</td>
<td>0.92 ± 0.01</td>
<td>0.88 ± 0.01</td>
<td>0.84 ± 0.02</td>
</tr>
<tr>
<td>C</td>
<td>0.95 ± 0.01</td>
<td>0.95 ± 0.01</td>
<td>0.92 ± 0.01</td>
<td>0.92 ± 0.00*</td>
<td>0.91 ± 0.01*</td>
</tr>
<tr>
<td>C + M</td>
<td>0.95 ± 0.01</td>
<td>0.93 ± 0.01</td>
<td>0.92 ± 0.01</td>
<td>0.91 ± 0.01*</td>
<td>0.90 ± 0.01*</td>
</tr>
</tbody>
</table>

Values are means ± SE; $n = 8$ subjects. $\dot{V}O_2$, oxygen uptake; RER, respiratory exchange ratio; P, C, C + M: placebo, carbohydrate, and carbohydrate and medium-chain triglyceride beverage, respectively; final measurement, nearing end of time trial. *Significantly different ($P < 0.05$) compared with P.

After C, plasma FFA concentrations were lower ($P < 0.05$) than in the P trial from 120 min to the end of the TT ($P < 0.05$). Similarly, after C + M, plasma FFA concentrations were lower ($P < 0.05$) than in the P trial from 90 min until TT completion ($P < 0.05$). Plasma insulin concentrations fell ($P < 0.05$) during exercise in all trials (Fig. 4; $P < 0.05$). In the P trial, plasma insulin concentrations were lower ($P < 0.05$) throughout the TT, compared with in C and C + M.
V\textsubscript{O2peak}. This is in contrast to a value of 1 M beverage.

\( P \) irrespectively of beverage composition, was 75 different from C (\( P \), Symbols for beverages are defined as in Fig. 1. *\( P \) is significantly different from C (\( P \), Table 1 vs. 0.89; Madsen et al. The difference in RER indicates a contribution of exercise [30-min values; 0.95; Table 1 vs. 0.89; Madsen et al.]. The average intensity selected in the first \( 2 \) h of exercise in the present study,是怎么的？

Subjects in both the present and previous (19) studies selected in the first \( 2 \) h of exercise in the present study, how-ever, is in contrast to a recent study that reported no benefit of CHO ingestion compared with ingestion of a sweet placebo (19). There are several possible explanations for the opposite findings with regard to exercise performance and CHO ingestion in a prolonged TT. Subjects in both the present and previous (19) studies were able to self-select their exercise workload, completed an estimated 100-m TT, and produced similar completion times with CHO feeding [166 ± 7; Fig. 1 vs. 160 ± 4 min; Madsen et al. (19)]. The average intensity selected in the first \( 2 \) h of exercise in the present study, irrespective of beverage composition, was 75 ± 1% of \textit{VO2peak}. This is in contrast to a value of <70% in the study by Madsen et al. As would be expected, the elevated exercise intensity of the subjects in the present study resulted in an augmented RER throughout exercise [30-min values; 0.95; Table 1 vs. 0.89; Madsen et al.]. The difference in RER indicates a contribution of CHO to total energy expenditure of 83 and 63% for the present and previous (19) study, respectively. A 20% reduction in CHO oxidation reduces the likelihood of endogenous CHO store depletion and negates the performance benefit of exogenous feedings (19). A reduced CHO availability in the absence of CHO feeding in the present (Fig. 1) and previous studies (3–5, 7, 8) contributed to decreased TT performances. Plasma glucose concentrations was significantly decreased in the absence of CHO feeding (Fig. 3) and reached a nadir of 3.9 ± 0.4 mmol/l, a value lower than measured before exercise. In contrast, Madsen et al. (19) reported the maintenance of euglycemia and no differences in RER during the prolonged TT, irrespective of exogenous CHO feeding. These differences in metabolic data suggest that subjects in the present study exercised at a higher percentage of their \textit{VO2peak}, derived more energy from CHO, and depleted their endogenous stores of CHO.

The average workload for the subjects was remarkably similar for the first 120 min of exercise (Fig. 1), suggesting prudent selection of an appropriate workload for the prolonged TT. Average plasma lactate concentrations were at least 3.7 mmol/l (Fig. 3) during the TT, demonstrating the maintenance of a high work rate and the high endurance capacity of this subject group. It was only in the final segment of the TT that a reduction in CHO oxidation, RER, and plasma glucose combined to reduce the amount of work performed if exogenous CHO was not provided. Hence, CHO ingestion allowed the subjects to maintain their work rate at the end of the TT, thereby reducing the time to complete the TT. Interestingly, despite strict pretrial instructions to abstain from exercise, the consumption of a high-CHO diet, and additional CHO feeding 2 h before exercise, TT performance was still limited by CHO availability during the control trial.

Recently, Van Zyl et al. (28) administered a CHO and MCT beverage during a 2-h steady-state cycle (at 57% \textit{VO2peak}) and reported an improvement in subsequent 40-km TT performance, compared with ingestion of CHO alone. The proposed mechanism of augmented TT performance was that the ingested MCT elevated plasma FFA concentrations, increased fat oxidation, and spared muscle glycogen. When FFA levels were elevated by infusions of intralipid and heparin (6, 29) or fat ingestion (29), increased fat oxidation, muscle glycogen sparing, and fatigue delay were observed. Jeukendrup et al. (17) performed an experiment similar to that by Van Zyl et al. (28) and observed no improvement in subsequent 15-min TT performance, no increase in fat oxidation, and no significant differences in FFA concentration with CHO and MCT administration compared with CHO alone, although there was a tendency for higher FFA concentrations in CHO and MCT feeding compared with CHO alone (17).

In the present study, no differences were observed in RER (Table 1), total fat oxidation (Fig. 2), or in plasma FFA (Fig. 4) during the TT when subjects ingested C+M, compared with C. These results suggest that the
exercise intensity may have been too severe for the medium-chain fatty acids to be absorbed into the bloodstream, or to be oxidized, as CHO was the preferred substrate during the TT (contributing 83% to total energy expenditure over the first 2 h). High-intensity exercise has been shown to reduce splanchnic blood flow by up to 70% (26), illustrating a possible explanation for a reduction in absorption of ingested MCT during the TT in the present study. Furthermore, of the eight subjects tested, four reported symptoms of GI discomfort after the MCT-feeding trial, ~2 h postexercise (stomach fullness, belching, and resultant loose stools), with two of these subjects experiencing more severe symptoms (vomiting and/or diarrhea). GI distress has been reported in other studies in which subjects have ingested MCT (14, 17). Although efforts were made to deliver the MCT in several small but frequent administrations to minimize discomfort, the potential for GI discomfort is an important practical finding, because it is likely that MCT ingestion would be adopted during TT situations.

Another possible obstacle to lipid mobilization and oxidation during this TT was the powerful effect of insulin on blunting lipolysis and elevating CHO oxidation. Athletes often consume a high-CHO meal 2–4 h before competition. In this study, subjects were instructed to consume a pretrial breakfast 2 h before they reported to the laboratory. Of note, the preexercise plasma insulin concentrations were higher than values typical for the postabsorptive state (Fig. 4), and elevated insulin concentrations have been found to reduce rates of fat oxidation (12). The elevation in resting insulin concentration as a consequence of consuming the preexercise meal in this study may affect the ability of all lipids to be oxidized, including the ingested MCT.

In summary, this study demonstrated that CHO ingestion during a simulated 100-km TT in well-trained athletes does enhance exercise performance. The addition of MCT to the ingested CHO beverage, however, neither alters plasma FFA concentrations nor provides any additional reduction in TT time.

This study was supported by a research grant from the Gatorade Sports Science Institute. Address for reprint requests and other correspondence: M. Febbraio, Exercise Physiology and Metabolism Laboratory, Dept. of Physiology, The Univ. of Melbourne, Parkville, Victoria 3052, Australia (E-mail: m.febbraio@physiology.unimelb.edu.au).

Received 25 February 1999; accepted in final form 1 September 1999.

REFERENCES


