Enhanced postprandial energy expenditure with medium-chain fatty acid feeding is attenuated after 14 d in premenopausal women[1–3]

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ABSTRACT

Background: Medium-chain triacylglycerols (MCTs) are reported to enhance human energy expenditure (EE), although few studies have involved women and the duration of such effects is only known for periods of ≈7 d.

Objective: This study was conducted to determine whether women consuming mixed, MCT-enriched or long-chain triacylglycerol (LCT)–enriched diets showed changes in EE or substrate oxidation after 7 and 14 d.

Design: Twelve nonobese, premenopausal women were fed isoenergetic mixed diets enriched in either MCTs or LCTs during separate, 14-d feeding periods. Each meal contained 40% of energy as fat (80% of which was the treatment fat), 45% as carbohydrate, and 15% as protein. On days 7 and 14 of each trial, basal metabolic rate (BMR, kJ/min), total energy expenditure (TEE, kJ/min), and thermic effect of feeding (ΔkJ/min) after a standardized breakfast were measured by respiratory gas exchange.

Results: On day 7, the mean (±SEM) BMR (3.58 ± 0.11 kJ/min) with the MCT diet was greater ($P = 0.0003$) than that with the LCT diet (3.43 ± 0.11 kJ/min). The mean postprandial TEE on day 7 was significantly greater ($P = 0.04$) with the MCT diet (4.36 ± 0.04 kJ/min) than with the LCT diet (4.23 ± 0.04 kJ/min); by day 14, postprandial TEE was still greater with the MCT diet, but not significantly so. No significant differences in the thermic effect of feeding were evident between diets.

Conclusions: Results from this longest controlled MCT feeding study to date suggest that short-term feeding of MCT-enriched diets increases TEE, but this effect could be transient with continued feeding.   *Am J Clin Nutr* 1999;69:883–9.

KEY WORDS   Premenopausal women, medium-chain triacylglycerols, long-chain triacylglycerols, thermic effect of food, respiratory quotient, fat oxidation, carbohydrate oxidation, energy expenditure

INTRODUCTION

The composition of dietary fat is thought to influence postprandial energy expenditure (EE) and substrate oxidation rates in rodents (1) and humans (2–6). Specifically, the fatty acid chain length is thought to be a determinant of the rate of postprandial substrate oxidation and total EE (7, 8). Over the postprandial period, the thermic effect of food (TEF) and fat oxidation were shown by Hill et al (4) to be greater when medium-chain triacylglycerols (MCTs) than when long-chain triacylglycerols (LCTs) were ingested. In hospitalized patients receiving total parenteral nutrition, similar responses were observed (5): patients showed no metabolic responses to LCT infusions, but significant increases in the TEF and in fat oxidation after MCT infusions. Additionally, a diet containing low-to-moderate amounts of MCT was shown to stimulate 24-h EE by 5% more than a diet with an isoenergetic amount of LCT (6). These studies support the postulate that both MCT and LCT ingestion will increase postprandial EE, but that the increase is greater after MCT ingestion, possibly because of enhanced postprandial fat oxidation.

The evidence supporting increases in postprandial EE and fat oxidation after MCT ingestion (2–6) is not without exception. Flatt et al (9) showed that postprandial EE and fat and carbohydrate oxidation were similar after breakfasts that were low in fat, MCT-enriched, or LCT-enriched. Similarly, MacDougall et al (10) showed no significant differences in mean postprandial EE and substrate oxidation on days 8 and 11 after a breakfast enriched in either MCT or LCT. After consumption of longer-term, low-energy diets (11, 12), women showed similar weight losses with diets enriched in either MCT or LCT. The results from studies with feeding of high-MCT–containing diets suggest that a positive effect on EE and fat oxidation is possible for periods up to ≈7 d (4–6). It is questionable, however, whether longer feeding periods would show these differences (11, 12). In addition, only 2 studies report comparisons of MCT and LCT feeding in women (11, 12); most other studies were restricted to men (2–6, 9).

The purpose of the present study was to determine whether fatty acid chain length influences EE and substrate oxidation in

women being fed controlled, isoenergetic, North American diets that were consumed under supervision in a clinical research unit. Feeding diets enriched in either MCT or LCT over 2 wk allowed the subjects' responses to be compared over a period at least twice as long as reported previously (4–6).

SUBJECTS AND METHODS

Subjects

Twelve women participated in the study and sample size was calculated by using the tables of Machin and Campbell (13). The criteria for selection were that subjects were normolipidemic, menstruating regularly, nonobese, and between 18 and 30 y of age. The subjects selected were 22.8 ± 2.2 y of age ($\bar{x} \pm$ SE) and had a mean weight, height, and body mass index (kg/m^2) of 56.9 ± 5.6 kg, 1.63 ± 4.0 m, and 21.4 ± 2.0, respectively. All subjects were informed of the inherent risks of the study and were instructed that they could withdraw their participation at any time without prejudice. The protocol was approved by the McGill University Human Ethics Committee and all subjects gave their signed, informed consent before the study.

Diets

Two mixed diets were used in the study. An MCT-enriched and an LCT-enriched diet were prepared in the Mary Emily Clinical Nutrition Research Unit (CNRU) of the School of Dietetics and Human Nutrition on the MacDonald Campus of McGill University, Ste-Anne-de-Bellevue, Canada, and included typical North American foods. The MCT- and LCT-enriched diets provided 40% of energy as fat (80% of which was the treatment fat), 45% as carbohydrate, and 15% as protein. Butter and coconut oil were the fats used to increase the proportion of MCTs in the diet and beef tallow was the fat used to increase the proportion of LCTs. During food preparation, all foods were weighed to the nearest 0.1 g. The proportions of fatty acids in each of the MCT- and LCT-enriched diets are given in **Table 1**. The isoenergetic intake was calculated with the equation of Mifflin et al (14), from which the basal metabolic rate (BMR) was determined and then multiplied by an individualized activity factor (15). The factor of Bell et al (15), adjusted for athletically active subjects by using a table adapted from Passmore and Durnin (16), gave a mean activity factor of 1.72 ± 0.05. The mean energy intake of

TABLE 1
Fatty acid composition of the 2 test diets[1]

Fatty acid chain length	Diet	
	MCT	LCT
	% of total fatty acids	
8:0	3.9	0
10:0	4.0	0.4
12:0	17.7	1.4
14:0	13.3	4.4
16:0	25.4	27.3
16:1n−7	1.6	2.9
18:0	8.1	16.9
18:1n−9	19.3	35.9
18:1n−7	0.6	0.8
18:2n−6	4.8	8.0

[1]MCT, medium-chain triacylglycerol; LCT, long-chain triacylglycerol.

the subjects was $10\,682 \pm 184$ kJ and the subjects' weights were maintained at 56.6 ± 0.4 kg in the MCT-diet group and at 56.5 ± 0.4 kg in the LCT-diet group. The diets for each treatment were isoenergetic for each of the breakfast, lunch, and dinner meals. Feeding was closely supervised throughout the trials to ensure that all food provided was consumed. Subjects showed excellent tolerance of these diets and consumed all of the prescribed food. More than 99% of the meals were eaten in the CNRU; on only a few occasions were prepackaged meals consumed outside the CNRU.

Replicate portions of the 2 diets were homogenized by using a commercial blender and relative percentages of the fatty acids were determined by gas-liquid chromatography after lipid extraction (17) and boron trifluoride methylation. The gas chromatograph (model 5890 series II; Hewlett-Packard, Palo Alto, CA) was equipped with an autosampler and flame ionization detectors. Separation was achieved on an SP2330 (Supelco, Bellefonte, PA) 30-m capillary column with an internal diameter of 0.2 mm and a 0.25-μm film thickness. The split ratio was 50:1. The gas chromatograph was programmed to an initial temperature of 80°C, to increase by 10°C/min to 160°C and hold for 10 min, to then increase by 10°C/min to 220°C and hold for 12 min, and then to increase by 10°C/min to a maximum of 240°C and hold for 5 min. Individual fatty acids were identified against standards by using retention times.

Study design

Each diet was fed for a 14-d period, separated by a 14-d washout period during which time subjects resumed their habitual eating patterns. Subjects were blinded to diet type and were randomly assigned in equal numbers to either the MCT- or LCT-enriched diet before the first 14-d feeding period began. After consuming a standardized breakfast (ie, isoenergetic in total energy and fat), subjects' expired gases were continuously collected with a ventilated hood over 5.5 h; short washroom breaks were permitted. The standardized breakfast contained one-third of each subject's predetermined daily energy intake. Throughout the postprandial period, subjects relaxed in a semirecumbent position and read or watched television.

Measurements

A Deltatrac metabolic monitor (Sensormedics, Anaheim, CA) was used to determine oxygen consumption and carbon dioxide production, both expressed at standard temperature and pressure dry. On days 6 and 13 of each diet, subjects slept at the CNRU and on the following morning, at ≈0700 after a 10–12-h fast, BMR was measured over 30 min. After the standardized breakfast, a transparent ventilated hood was positioned over the subject's heads with Collins tubing connecting the hood to the monitor and expired gases were continuously collected over 5.5 h; short washroom breaks were permitted. After a warm-up period of ≥30 min, a reference gas (5% CO_2, 95% O_2) was used to calibrate the oxygen and carbon dioxide analyzers. Validation of Deltatrac analyzers against a lung model has shown an accuracy of 1.9% for oxygen consumption and 1.5% for carbon dioxide production (18). Readings from the metabolic monitor were collected every minute with a personal computer.

Core temperatures increase from ≈0.5 to 1°C after ovulation (during the luteal phase) and for each degree Celsius increase in body temperature, resting metabolic rate increases by 10–12% (19). This effect has been shown to elevate 24-h EE by 8–16%

(20) and postovulatory TEF is lower than preovulatory TEF (21). To control for this change in body temperature, we used a 14-d feeding period followed by a 14-d washout period. Because the subjects had menstrual cycles ≈28 d in length, they were on the same day of their cycles when the second treatment began.

EE was expressed as postprandial total EE (TEE, kJ/min). The TEF (ΔkJ/min) was calculated as the difference between the postprandial EE and EE measured under basal conditions before breakfast (ie, the BMR). Mean postprandial TEE, mean fat (g/min), and mean carbohydrate oxidation (g/min) rates across the entire postprandial period were calculated for each 30-min period. A mean value was calculated for each 30-min period after breakfast until the end of the measurement period. The integrated values for total carbohydrate (g) and total fat (g) oxidized over the postprandial period are also given. The respiratory quotient (RQ) was calculated as carbon dioxide production divided by oxygen consumption. Weir's (22) formula was used unmodified to give an estimate of fat and carbohydrate oxidation because the error due to the oxidation of MCT rather than LCT was estimated at <1% (3). A constant oxidation of 0.7 g protein/kg fat-free mass was assumed because an error in protein oxidation ≤30% would have no significant effect on substrate oxidation rates (23).

Statistical analyses

A crossover, repeated-measures analysis of variance was used. The factors used were diet (MCT or LCT), day (day 7 or day 14), and hour (different times between 0.5 and 5.5 h). A sequence factor for diet was added to control for crossover effects, as recommended for this type of repeated-measures design (24). Post hoc comparisons at selected 30-min intervals were made between treatment conditions by using contrasts. For multiple post hoc comparisons, Bonferroni correction was applied to control for the level of type I error. To assess body weight, a repeated-measures ANOVA (diet, day, and diet-by-day-interaction) was used. SUPERANOVA (Abacus Concepts, Inc, Berkeley, CA) was used for the analyses.

RESULTS

A comparison of mean EEs on days 7 and 14 as a function of dietary fat type is shown in **Figure 1**. The mean rates of EE measured under basal conditions are given at 0 h on both days. The main effect of diet (MCT and LCT) on BMR was significant ($F = 16.11$, $P = 0.001$), but there was no diet-by-day (days 7 or 14) interaction for the model. On day 7, the BMR was greater ($P = 0.003$) with the MCT (3.58 ± 0.11 kJ/min) than with the LCT (3.43 ± 0.11 kJ/min) diet. On day 14, the BMR was also greater ($P = 0.06$) with the MCT (3.63 ± 0.08 kJ/min) than with the LCT (3.49 ± 0.09 kJ/min) diet.

Postprandial TEE was greater with the MCT than with the LCT diet on day 7, with an attenuation of this effect on day 14. The main effect of diet on TEE was significant ($F = 30.87$, $P = 0.001$) and there was a significant diet-by-day interaction ($F = 5.22$, $P = 0.02$). After the standardized breakfast on day 7, post hoc mean comparisons showed that postprandial TEE was significantly greater with the MCT than with the LCT diet at different time points between 0.5 and 3 h (Figure 1). These differences are reflected on day 7 by a mean TEE rate of 4.36 ± 0.04 kJ/min with the MCT diet that was greater ($P = 0.04$) than the mean rate with the LCT diet (4.23 ± 0.04 kJ/min). These differences in TEE were attenuated on day 14, as supported by the significant diet-by-day interaction for TEE and by the mean rate of TEE on day 14 with the MCT diet (4.38 ± 0.03 kJ/min), which was not significantly different from that with the LCT diet (4.29 ± 0.03 kJ/min).

On day 7, the integrated TEE across the 330-min postprandial period with the MCT diet (1439.43 ± 34.63 kJ) was not significantly greater than that with the LCT diet (1394.76 ± 28.38 kJ). On day 14, the integrated postprandial TEE was 1444.56 ± 24.88 kJ
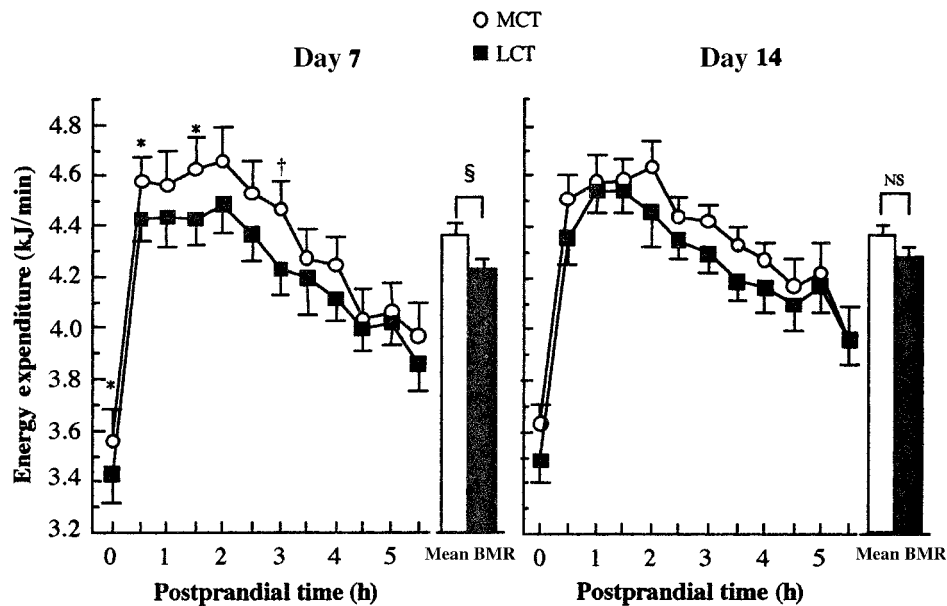

FIGURE 1. Comparison of mean (±SEM) postprandial energy expenditure and basal metabolic rate (BMR) on days 7 and 14 between diet conditions [moderate amounts of medium-chain triacylglycerols (MCT) or long-chain triacylglycerols (LCT)] in nonobese women. Measurements were made after consumption of a breakfast standardized with MCT or LCT. Significantly different from LCT diet: $^{\S}P < 0.05$, $^{*}P < 0.004$, $^{\dagger}P < 0.001$.

with the MCT diet, which was not significantly different from that with the LCT diet (1413.46 ± 27.32 kJ). Neither the main effect of diet nor the diet-by-day interaction for TEF (ie, TEE − BMR) were significant. On day 7, the mean TEF was 0.79 ± 0.03 kJ/min with the MCT diet and 0.80 ± 0.03 kJ/min with the LCT diet. On day 14, the mean TEF was 0.75 ± 0.04 kJ/min with the MCT diet and 0.79 ± 0.03 kJ/min with the LCT diet.

For the RQ, the main effect of diet ($F = 34.72$, $P = 0.0001$) and the diet-by-day interaction ($F = 6.98$, $P = 0.009$) were both significant. No significant differences in the RQ during basal conditions were evident between the 2 diets. At different time points after the standardized breakfast on day 7, the RQ with the LCT diet was greater than that with the MCT diet (**Figure 2**).

The main effect of diet ($F = 43.57$, $P = 0.0001$) and the diet-by-day interaction ($F = 8.69$, $P = 0.004$) were both significant for fat oxidation. This main effect of diet was evident at different time points in the postprandial period on day 7, when the rate of fat oxidation was greater with the MCT diet than with the LCT diet (Figure 2). The main effect of diet ($F = 18.77$, $P = 0.001$) and the diet-by-day interaction ($F = 4.23$, $P = 0.04$) for carbohydrate oxidation were also significant. On day 7, carbohydrate oxidation was lower with the MCT diet than with the LCT diet. On day 14, as evidenced by the significant diet-by-day interaction, substrate oxidation and RQ were similar with both diets.

There was no effect of diet or diet-by-day interaction for total fat or total carbohydrate oxidation. In the postprandial period,
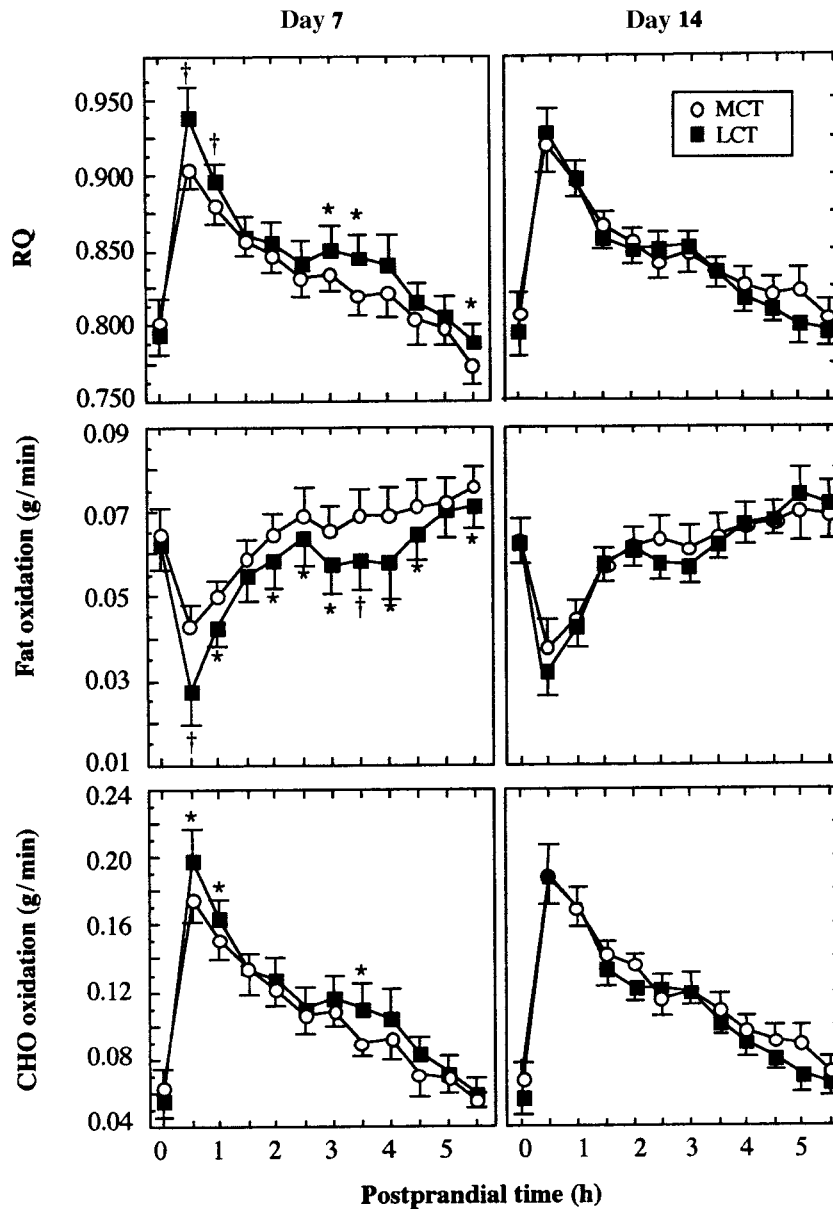

FIGURE 2. Comparison of mean (±SEM) preprandial (0 h) and postprandial respiratory quotients (RQ), fat oxidation, and carbohydrate (CHO) oxidation between diet conditions [moderate amounts of medium-chain triacylglycerols (MCT) or long-chain triacylglycerols (LCT)] on days 7 and 14 in nonobese women. Measurements were made after consumption of a breakfast standardized with MCT or LCT. Significantly different from MCT diet: *$P < 0.004$, †$P < 0.001$.

total fat oxidation on day 7 was 20.88 ± 1.48 g with the MCT diet and 18.31 ± 1.82 g with LCT diet. By day 14, total fat oxidation values were nearly identical with both diets (MCT diet: 19.07 ± 1.48 g; LCT diet: 19.09 ± 1.30 g). On day 7, carbohydrate oxidation rates were 36.04 ± 2.41 g with the MCT diet and 39.32 ± 3.44 g with the LCT diet. On day 14, total carbohydrate oxidation rates were 40.49 ± 2.51 g with the MCT diet and 38.63 ± 2.33 g with the LCT diet.

There was no main effect of diet during the postprandial period on day 7 after mean fat oxidation rates were corrected for basal fat oxidation rates. Mean values were similar: 0.001 ± 0.002 and -0.007 ± 0.002 g/min with the MCT and LCT diets, respectively. However, there was a diet-by-day interaction

($F = 4.34$, $P = 0.04$) for corrected fat oxidation. On day 14, postprandial fat oxidation rates were -0.007 ± 0.002 g/min with the MCT diet and -0.005 ± 0.002 g/min with the LCT diet. There was no main effect of diet or diet-by-day interaction for carbohydrates when corrected for basal oxidation rates. Carbohydrate oxidation rates on day 7 were 0.044 ± 0.004 and 0.062 ± 0.004 g/min with the MCT and LCT diets, respectively. On day 14, carbohydrate oxidation rates corrected for basal oxidation rates were the same with both diets: 0.059 ± 0.004 g/min. The RQ and substrate oxidation rates were grouped by diet and compared between days 7 and 14 (**Figure 3**). No main effect of diet or diet-by-day interaction for substrate oxidation rates and RQ were observed for within-diet, between-day comparisons.
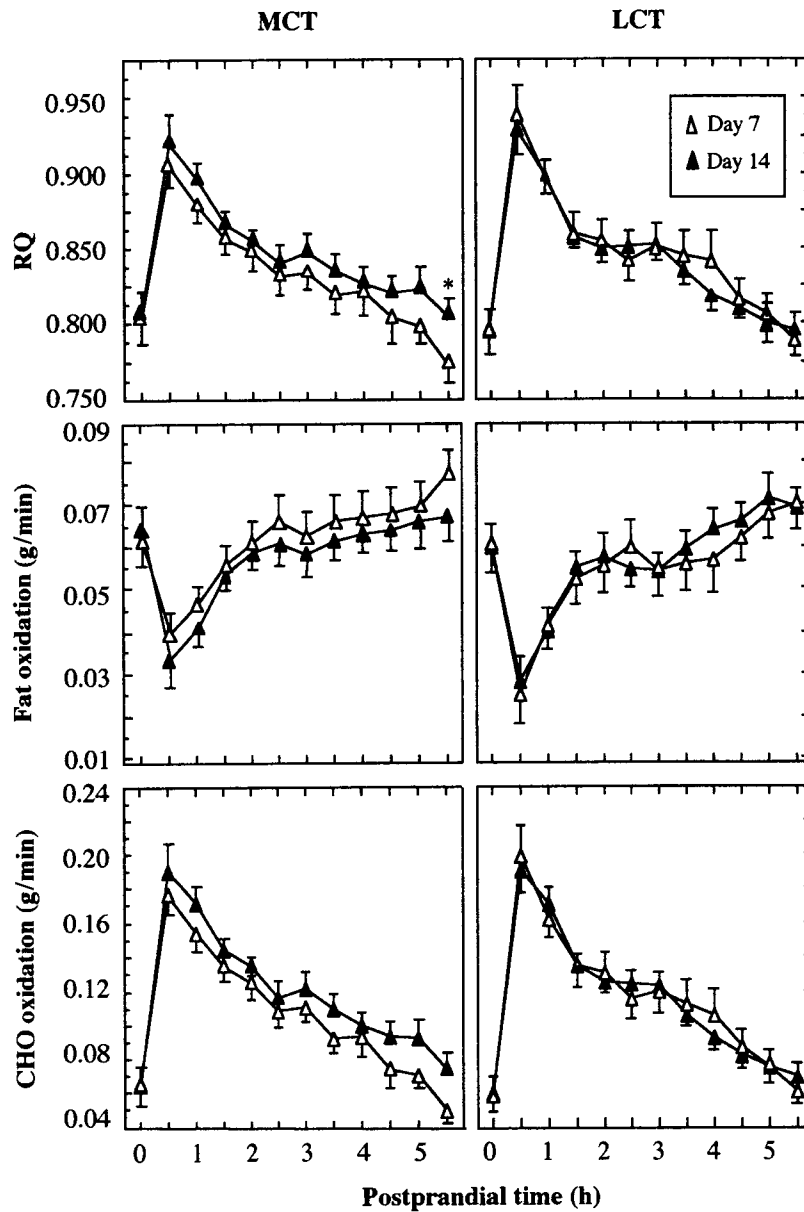

FIGURE 3. Comparison of mean ($\pm$SEM) preprandial (0 h) and postprandial respiratory quotients (RQ), fat oxidation, and carbohydrate (CHO) between testing day (days 7 and 14) in nonobese women consuming a diet with moderate amounts of medium-chain triacylglycerols (MCT) or long-chain triacylglycerols (LCT). Measurements were made after consumption of a breakfast standardized with MCT or LCT. *Significantly different from day 7, $P < 0.004$.

DISCUSSION

The effect of controlling the fatty acid chain length in the fat component of a mixed diet for these women showed a time-dependent effect on both EE and substrate oxidation. On day 7, the increases in BMR and postprandial TEE with the MCT diet were small but significantly greater than those with the LCT diet (Figure 1). In addition, on day 7, fat oxidation rates at different time points over the postprandial period were significantly greater with the MCT than with the LCT diet (Figure 2). This effect of MCT on both postprandial TEE and fat oxidation was attenuated by day 14 of the feeding periods (Figures 1 and 2). The BMR, in contrast, showed a trend for a sustained greater elevation with the MCT than with the LCT diet on day 14 (Figure 1). Postprandial TEE values on day 7 are consistent with results from studies using a mixed, weight-maintenance diet before a single fat load, indicating a greater increase in postprandial EE with the MCT than with the LCT treatment (2–5). Short-term prefeeding with the treatment fats (ie, MCT or LCT) for ≈1 wk produced similar results (4, 5). The results on day 14 are supported by the findings of studies of prolonged but uncontrolled (outpatient) feeding that showed a diminishing effect on energy balance as the duration of MCT-enriched dietary treatments was extended beyond a few days to several weeks (11, 12, 25). Specifically, similar weight losses were reported in women fed MCT-enriched or LCT-enriched hypoenergetic diets (11, 12) and in subjects fed MCT-enriched or non-MCT–enriched hypoenergetic diets (25). Together, the evidence suggests that the positive effects of MCT on energy balance diminished with long-term feeding.

Studies reporting resting metabolic rates or BMRs of subjects fed MCT- and LCT-enriched diets for 1–28 d showed both no effect (4, 10) or greater increases in preprandial EE with an MCT than with an LCT infusion (5) or a non-MCT hypoenergetic diet (25). Subjects supplemented with MCT oils during a 28-d hypoenergetic diet (25), despite a weight loss of 10 kg, maintained their prediet BMRs. Concurrently, the non-MCT oil control group (25) showed the same 10-kg loss and the associated decrease in BMR that is normally seen during such weight reductions (26, 27). Patients receiving total parenteral feeding with MCT and LCT oils showed a greater elevation in resting metabolic rate with the MCT than with the LCT infusion on days 1, 3, and 5 of the feeding (5), supporting the view that MCTs increase preprandial EE. Thus, results on the effects of MCTs in the preprandial period differ, with some studies showing no effect (4, 10) and others a positive effect (5, 25). The results of the present study indicate that MCT feeding resulted in a greater BMR than did LCT feeding after 7 d, and possibly after 14 d as well (Figure 1); however, this topic clearly merits further study to resolve these discrepancies. An effect of MCTs on BMR has considerable importance concerning weight maintenance because basal metabolic processes make up most of the daily EE.

Ravussin and Swinburn (28) showed that the CVs for repeated TEF responses varied between 4% and 48%. This large variability in TEF responses may cast light on discrepancies for TEF responses to MCT or LCT feeding. In the present study, there were no significant differences in TEF between the MCT and LCT groups; similar feeding studies showed the same results (9, 10). Some studies, however, showed a greater TEF with MCT than with LCT feeding (2–5). These discrepancies in TEF response may have been due to differences in the quantity of MCT fed or to the use of prefeeding protocols in some studies and not in others. Generally, for studies in which treatment fats were prefed (9, 10), including the present study, no differences in TEF were apparent between the MCT and LCT groups. In contrast, studies that showed a greater elevation in TEF after a single MCT load than after a single LCT load did not use a prefeeding protocol (2, 3, 5). In addition, studies that showed marked differences in TEF between MCT- and LCT-diet groups (2–5) used commercial MCT oils providing from 75% (5) to 100% (2) of the total fat energy intake. Such quantities of MCTs are considerably greater than those used in the present study (Table 1). Together the evidence suggests that an increase in TEF is greatest after a single fat load including a high proportion of MCTs (2–5) and when there is no prefeeding of MCTs (2, 3, 5). These differences in protocols and the highly variable nature of TEF responses (28) suggest that firm conclusions cannot be made about the effects of MCT and LCT feeding on the TEF. These findings apply equally to substrate oxidation rates corrected for basal rates of oxidation.

A short-term effect of MCT feeding on postprandial fat and carbohydrate oxidation was apparent during this study, but, overall, the evidence is not compelling. On day 7, greater rates of fat oxidation and lower rates of carbohydrate oxidation were evident with the MCT diet than with the LCT diet (Figure 2). There is evidence to support greater postprandial fat oxidation rates with MCT feeding than with LCT feeding (3–5), although the results of studies differ (2, 9, 10). When higher amounts of MCT than those used in the present study are fed, indirect calorimetry should be used with caution when measuring fat oxidation because the substrate mix may contain ketones (29, 30).

The diet-by-day interaction term was significant for RQ, fat oxidation, and carbohydrate oxidation. The source of these interactions appeared to be due to a time dependence of the diet factor. This time dependence was apparent from the differences evident in the postprandial period for each of RQ, fat oxidation, and carbohydrate oxidation on day 7, which diminished by day 14 (Figure 2). These interactions were evident despite maintained values of each variable within each diet (**Figure 3**). Studies of MCT feeding show improved glycemic control after 5 d, as evidenced by the increased amount of glucose needed for a euglycemic clamp (11, 31) and improved glucose tolerance, as judged by a higher glucose disappearance rate (32). There is also a higher expression of hepatic lipogenic enzymes after long-term feeding of MCT-enriched diets (33, 34). In one study, there was a 3-fold increase in total plasma triacylglycerols between days 1 and 6 of an MCT-enriched diet in humans (35). These studies (33–35) suggest increased hepatic lipogenesis from the medium-chain fatty acid (MCFA) moieties, a finding that supports decreased fat oxidation as MCT feeding is prolonged. Together the evidence suggests that postprandial fat oxidation decreases and glucose tolerance improves with longer-term feeding of MCTs (8).

The reason for elevated postprandial EE with MCT feeding but not with LCT feeding appears to be due to the different metabolic fates of MCT and LCT (8, 33). After absorption, most MCFAs are weakly bound to albumin (36) in the portal circulation and are preferentially oxidized to acetyl-CoA (8). No single mechanism accounting for the elevation in postprandial EE after MCT feeding was evident, but several hypotheses have been proposed (8). LCT, in contrast with MCT, is incorporated mostly into chylomicrons and enters the circulation through the lymphatic system. LCTs from chylomicrons are taken up mostly by peripheral adipose tissue for storage; this is less costly energetically than the ketogenesis or lipogenesis from MCFAs (37).

Based on the greater increase in EE on day 7 with the MCT diet than with the LCT diet across the 5.5-h postprandial period, projected to 24 h, the increase in energy expenditure would be ≈669 kJ (160 kcal), or 6% of a typical daily intake of ≈10500 kJ (2500 kcal) for these subjects. This value is supported by the findings of Dulloo et al (6), who showed by using whole-body indirect calorimetry that 24-h EE was 5% higher after MCT than after LCT feeding. If such an increase in 24-h EE were maintained over ≈20 d, it would contribute to a loss of energy approximately equivalent to that provided by a pound of fat (0.45 kg). This potential benefit over the longer term, however, is questionable because the effect of the MCT diet on EE was diminished by day 14 of the study.

In conclusion, on day 7 of a 14-d feeding trial of moderate amounts of MCT in a mixed diet, there were increases in BMR, postprandial EE, and fat oxidation in nonobese, college-aged women. These positive effects of the MCT diet on EE were diminished when feeding was extended to 14 d, a period longer than was studied previously. Evidence suggests that moderate amounts of dietary MCT increase EE, but this effect could be transient.

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