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PAPER

Greater rise in fat oxidation with medium-chain triglyceride consumption relative to long-chain triglyceride is associated with lower initial body weight and greater loss of subcutaneous adipose tissue

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OBJECTIVE: Medium-chain triglyceride (MCT) consumption has been shown to increase energy expenditure (EE) and lead to greater losses of the adipose tissue in animals and humans. The objective of this research was to examine the relationship between body composition and thermogenic responsiveness to MCT treatment.
DESIGN: Randomized, crossover, controlled feeding trial, with diets rich in either MCT or long-chain triglyceride (LCT) (as olive oil) for periods of 4 weeks each.
SUBJECTS: A total of 19 healthy overweight men aged ($x \pm$ s.e.m.) 44.5 ± 2.5 y with a body mass index of 27.8 ± 0.5 kg/m^2.
MEASUREMENTS: EE and body composition were measured using indirect calorimetry and magnetic resonance imaging, respectively, at the baseline and end point of each feeding period. EE was measured for 30 min before consumption of a standard meal and for 5.5 h following the meal.
RESULTS: Body weight (BW) decreased ($P<0.05$) by 1.03 ± 0.25 kg with MCT consumption compared to 0.62 ± 0.29 kg with LCT consumption. The difference in average EE between MCT and LCT consumptions was related to initial BW, such that men with lower initial BW had a greater rise in EE with MCT consumption relative to LCT on day 28 ($r=-0.472$, $P=0.04$) but not day 2 ($r=-0.368$, $P=0.12$). Similar results were obtained with fat oxidation on day 28 ($r=-0.553$, $P=0.01$). The greater rise in fat oxidation with MCT compared to LCT consumption on day 2 tended to be related to greater loss of BW after MCT *vs* LCT consumption ($r=-0.4075$, $P=0.08$).
CONCLUSION: These data suggest that shunting of dietary fat towards oxidation results in diminished fat storage, as reflected by the loss of BW and subcutaneous adipose tissue. Furthermore, MCT consumption may stimulate EE and fat oxidation to a lower extent in men of greater BW compared to men of lower BW, indicative of the lower responsiveness to a rapidly oxidized fat by overweight men.
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Keywords: medium-chain triglycerides; body weight; adipose tissue; energy expenditure; magnetic resonance imaging

Introduction

Obesity is a worldwide problem, with prevalence rates reaching over 30% in the American adult population,[1] and is linked to a plethora of disorders, ranging from type II diabetes to cardiovascular disease, hypertension, and certain cancers. Dietary treatments to prevent and reverse obesity have largely been unsuccessful, with the majority of dieters regaining the lost weight.[2,3] In a recent meta-analysis, subjects regain, on average, 77% of the weight lost within 5 y of the weight loss program.[4] Another approach to weight loss involves tipping the energy balance scale towards greater energy output. This can be achieved through increased physical activity, but has also been observed with dietary

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modifications. Medium-chain triglycerides (MCTs), which do not require chylomicron formation for absorption into the circulation, travel directly to the liver, where they have been shown to be rapidly and almost completely oxidized.[5,6] Human studies have shown that replacing the more common dietary long-chain triglycerides (LCTs) for MCT leads to increased energy expenditure (EE) and fat oxidation,[7–14] resulting in body adipose tissue loss.[14,15] However, studies linking increased EE with MCT consumption to body composition changes are scarce[12,14] and the underlying mechanisms relating these factors are not well developed.

The aim of the current trial was to verify whether replacement of LCT by MCT in a typical North American diet would lead to changes in EE and body composition in overweight men. The objectives of this trial were, therefore, to examine whether differences in EE and substrate oxidation between MCT and LCT consumptions would be related to initial body weight (BW) and composition, as well as to the different degree of body composition change after consumption of isocaloric diets for 4 weeks each.

Methods

Subjects

In all, 24 healthy, overweight men, aged ($x \pm$s.e.m.) 43.1 ± 2.3 y with a body mass index (BMI) of 28.2 ± 0.4 kg/m^2, participated in this trial (Table 1). Subjects were recruited from newspaper advertisements. The inclusion criteria were BMI between 25 and 31 kg/m^2, and total cholesterol and triglyceride concentrations below 7.0 and 3.0 mmol/l, respectively. Subjects were excluded if they had previously been diagnosed with cardiovascular disease, diabetes, hypertension, and gastrointestinal disorders, and if they were taking cholesterol-lowering medications or had unusual eating patterns. The study protocol was reviewed and accepted by the Human Ethical Review Committee of the Faculty of Agriculture and Environmental Sciences of McGill University. Eligible subjects signed informed consent forms in the presence of the study coordinator prior to entry into the study.

Study design

This randomized, crossover, controlled feeding trial involved subjects consuming two different diets differing only in the type of added fat for two periods of 28 days each (Table 2). Experimental phases were separated by a 4-week washout period to allow for measured parameters to return to basal values. Diets were designed to maintain BW and each subject's energy requirement was calculated using the Mifflin equation,[16] multiplied by an activity factor of 1.7. This factor was previously shown to adequately estimate energy requirements for weight maintenance.[17] Both diets contained 40% of energy as fat, 55% as carbohydrates, and 15% as protein. Of the total amount of fat provided by the diets, 25% was intrinsic to foods common to both experimental diets and the remaining 75% was added fat. For the MCT diet, two-thirds of the added fat was MCT oil (Neobee 1053, Stepan Company, Northfield, USA) and the rest was provided by olive oil, coconut oil, flaxseed oil, and canola oil. The MCT oil was composed of 55% octanoic and 44% decanoic acids extracted from coconut or palm kernel oil. For the LCT diet, all of the added fat was olive oil. Energy intakes were increased or decreased by 2% in the first week of the first phase to account for any BW variations that may occur when one adapts to a new diet. After this initial 1-week period, energy intakes remained constant throughout the rest of the first phase and this caloric load was used for the second phase.

Subjects were required to eat two meals per day under supervision at the Mary Emily Clinical Nutrition Research Unit (CNRU) of McGill University, and could eat the third meal away from the CNRU. Subjects were repeatedly instructed to consume all foods provided and nothing else, during the two experimental phases. In addition, subjects were asked not to deviate from habitual physical activity level and to maintain a constant pattern of physical activity during the two experimental phases.

Table 1 Subject characteristics at screening

Characteristic	*Average (s.e.m.)*
Age (y)	43.1 (2.3)
Weight (kg)	87.2 (1.9)
Height (m)	1.76 (0.01)
Body mass index (kg/m^2)	28.2 (0.4)

Table 2 Fatty acid composition of the MCT and LCT treatment fats.

Fatty acid	*MCT[a] (%)*	*LCT (%)[b]*
6:0	0.17	0
8:0	36.95	0
10:0	30.35	0
12:0	3.61	0
14:0	1.06	0
16:0	3.52	14
16:1	0.23	0
18:0	0.65	3
18:1	13.81	71
18:2(n-6)	4.62	10
18:3(n-3)	4.94	Tr
20:0	0.05	0
Total SFA	76.2	17
Total MUFA	14.0	71
Total PUFA	9.6	10

[a]LCT, long-chain triglyceride; MCT, medium-chain triglyceride; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids. [b]From Jones PJH, Kubow S. Lipids, sterols, and their metabolites. In: Shils ME, Olson JA, Shike M, Ross AC (eds.). *Modern Nutrition in Health and Disease*, 9th edn. Lippincott, Williams and Wilkins: MA, USA. 71pp.

Body composition measurements

Body weights were measured every day before breakfast using a standard scale. On days 1 and 29 of each

experimental phase, body composition was assessed using magnetic resonance imaging (MRI). The MRI protocol is described in detail elsewhere.[12] Briefly, images were acquired using a Siemens 1.5 T MRI scanner (Siemens, Mississauga, Canada) using a T-1 weighted, spin-echo sequence with a 210 ms repetition time and a 17 ms echo time. Subjects were required to lie in the magnet in a prone position with their arms above their head while images were being acquired. Using the intervertebral space between the fourth and fifth lumbar vertebrae (L4–L5) as the point of origin, transverse images with 10 mm slice thickness were obtained every 40 mm from hand to foot, resulting in a total of approximately 45 images for each subject. The entire MRI protocol took approximately 45 min. Data were analyzed using specially designed MRI analysis software (Tomovision Inc, Montreal, Canada). Details of the data analysis procedure have been previously published.[12]

EE measurements

EE measurements were taken for 19 of the subjects (age 44.5 ± 2.5 y and BMI 27.8 ± 0.5 kg/m^2) on days 2 and 28, after a 12 h overnight fast. On each testing day, subjects were required to arrive at the CNRU approximately 1 h prior to the start of the measurement period, for a resting period designed to allow the subject's metabolic rate to return to near-basal state. After this initial resting period, the resting metabolic rate (RMR) was assessed using indirect calorimetry with ventilated hood methodology (Delta Trac, Sensor Medics, Anaheim, USA). The metabolic cart was calibrated daily, after an overnight warm-up period, at an ambient pressure with gas containing 96% O_2 and 4% CO_2. Following RMR measurement, subjects were provided with a standard breakfast to be consumed in a 30 min period, after which EE measurements were resumed. All subjects were provided with the 30 min breakfast break during which they were required to consume the entire breakfast meal during both EE measurement periods. EE was measured 30 min every hour for 5.5 h following breakfast consumption. This measurement period was determined to be appropriate to capture most of the thermic effect of a meal.[18] Subjects were permitted to go to the bathroom, watch television or read in between active EE measuring periods. They were not permitted to walk around the house or do any other type of activity. EE and fat and carbohydrate oxidation rates were calculated for every minute using standardized Eqs.[19]

Statistical analyses

EE values for days 2 and 28 for each correlation analysis used the average EE values over each 6.5-h EE measurement period. The change in BW was calculated as the change between the average last 3 days and first 3 days of each experimental period. The body composition values used for correlation analyses include total adipose tissue (TAT), subcutaneous adipose tissue (SAT), upper body adipose tissue (AT), visceral adipose tissue (VAT), and lean tissue (LT). Upper body AT was computed by summing all AT compartment volumes from images starting at L4–L5 and above. Initial LT and VAT volumes were calculated by averaging volumes at the baseline of both dietary phases. Correlations were computed to determine the body weight at which MCT intake was most beneficial to effect increases in EE and fat oxidation, and to verify that these thermogenic responses were linked to the degree of weight loss observed with MCT consumption relative to LCT. All correlations were performed using SAS system for windows version 8.2 (SAS Institute, Cary, USA). Data are reported as means $\pm$ s.e.m. Statistical significance was set at a P-value of 0.05.

Results

Body weights decreased with both MCT and LCT consumption. Body weight, body composition, and EE data with MCT and LCT consumption are reported elsewhere[14]. In short, MCT consumption resulted in increased EE and fat oxidation relative to LCT consumption (Figure 1). Furthermore, MCT consumption reduced TAT, SAT, and upper body AT by 0.83 ± 0.25, 0.54 ± 0.16, and 0.67 ± 0.26 kg, respectively, but only the change in upper body AT was significantly different from the change that occurred with LCT consumption (Figure 2). The corresponding body composition variations with LCT consumption, 0.31 ± 0.30, 0.17 ± 0.19, and 0.02 ± 0.19 kg, were not significant.

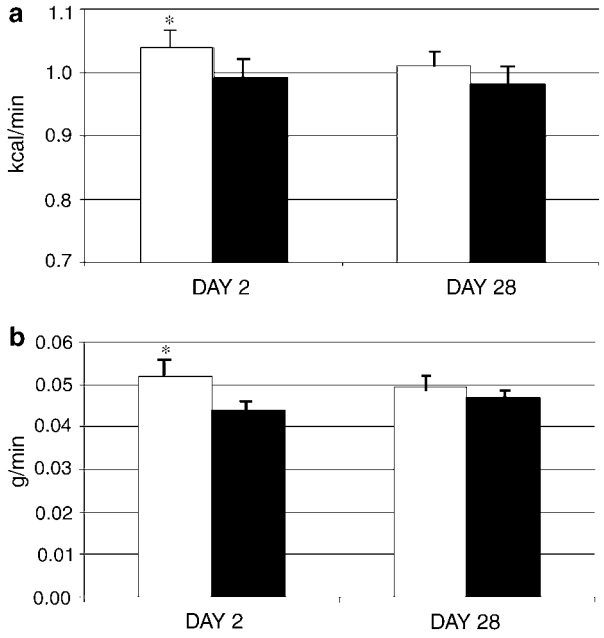

Figure 1 EE (a) and fat oxidation rates (b) with MCT (white bars) and LCT (black bars) consumption on days 2 and 28 of each experimental phase. *Significant diet difference, $P<0.05$, $n=19$.

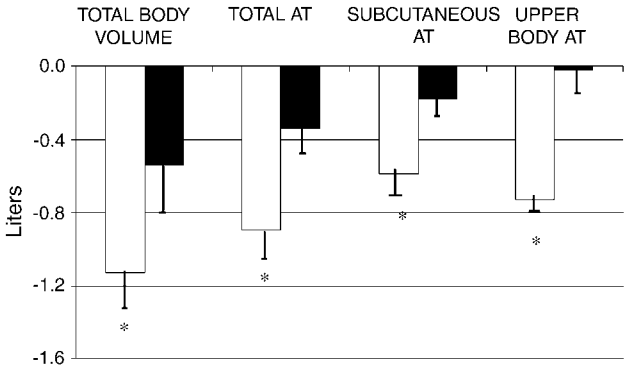

Figure 2 Change in body adipose tissue compartments with MCT (white bars) and LCT (black bars) consumption for 28 days. *Significant within diet effect, $P<0.05$, $n=24$.

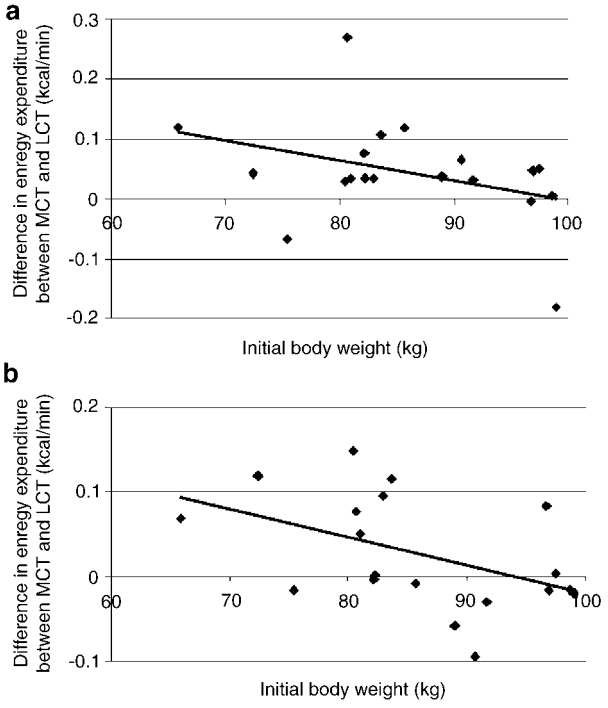

Figure 3 Correlation between difference in EE with MCT and LCT consumption (MCT–LCT) on day 2 (a; $r=-0.368$, $P=0.12$) and day 28 (b; $r=-0.472$, $P=0.04$) and initial BW, $n=19$.

Figure 3 shows the relationships between differences in EE between MCT and LCT consumption and initial BW. Correlation analyses show that the difference in EE between MCT and LCT on day 2 tended to be related to initial BW ($r=-0.368$, $P=0.12$). This inverse relationship became significant on day 28 ($r=-0.472$, $P=0.04$). The relationship between the difference in EE between MCT and LCT and initial BW was largely driven by relationships between EE and LT mass. The differences in EE between MCT and LCT on days 2 and 28 were inversely related to LT volume

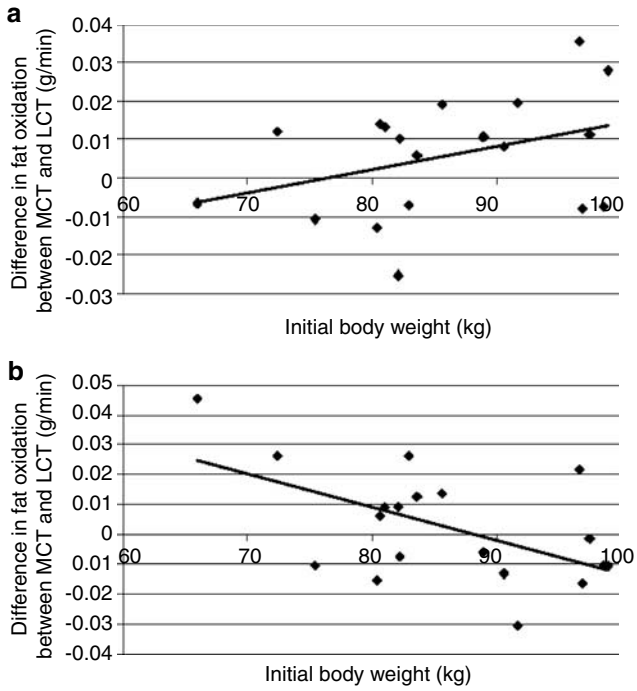

Figure 4 Correlation between difference in fat oxidation with MCT and LCT consumption (MCT–LCT) on day 2 (a; $r=0.367$, $P=0.12$) and day 28 (b; $r=-0.553$, $P=0.01$) and initial BW, $n=19$.

($r=-0.478$, $P=0.038$ for day 2; $r=-0.590$, $P=0.0079$ for day 28). Correlations for differences in fat oxidation between MCT and LCT consumption and initial BW are shown in Figure 4. Similar results as those seen with EE were obtained when correlating differences in fat oxidation and initial BW for day 28 ($r=-0.553$, $P=0.01$), but the reverse trend was found for day 2 ($r=0.367$, $P=0.12$). In addition, initial VAT and LT volumes were inversely correlated with difference in fat oxidation between MCT and LCT ($r=-0.496$, $P=0.031$ and $r=-0.613$, $P=0.0053$, for VAT and LT, respectively) on day 28.

Responsiveness to MCT feeding by increased EE relative to LCT on both days 2 and 28 was not related to differences in BW and body composition changes between MCT and LCT feeding periods. There was a trend on day 2 for the increase in fat oxidation with MCT consumption relative to LCT to be inversely related to changes in BW between MCT and LCT feeding periods ($r=-0.408$, $P=0.08$). This trend was no longer apparent when the same correlation was computed using fat oxidation data from day 28.

Data from both treatment phases were merged to assess whether BW and body composition were correlated with EE and fat oxidation. This analytical method is considered appropriate since it is not expected that diet treatment would modify the effect of BW and body composition on EE or fat oxidation. Results from these analyses showed that EE on day 2 and 28 were positively correlated with initial BW ($r=0.592$, $P<0.0001$ and $r=0.545$, $P=0.0004$, for days 2 and

28, respectively). This correlation was largely driven by LT volume since AT compartments, either total, subcutaneous or visceral, were not related with EE or fat oxidation rates. As a result, EE was positively correlated with LT volume on day 2 ($r=0.654$, $P<0.001$) and 28 ($r=0.439$, $P=0.0058$). When examining the relationship between fat oxidation and LT, correlation coefficients were smaller, 0.376 ($P=0.020$) for day 2 and 0.230 ($P=0.165$) for measurements taken on day 28. The rate of fat oxidation was not correlated with initial BW or AT volumes.

Again with data from both treatment phases combined, EE on day 2, but not day 28, was significantly inversely related to change in BW ($r=-0.351$, $P=0.03$). EE was not significantly correlated with change in any of the AT compartments. Fat oxidation, however, was significantly and inversely correlated with change in BW for day 2 ($r=-0.342$, $P=0.04$). On day 28, a trend was observed relating fat oxidation and change in BW ($r=-0.316$, $P=0.054$), yet, as for EE, was not related to changes in body AT compartments.

When each treatment phase was examined separately, EE with MCT consumption was not related to body composition changes for any of the parameters measured. However, fat oxidation with MCT consumption on day 2 was related to change in BW ($r=-0.607$, $P=0.006$) and SAT ($r=-0.478$, $P=0.04$). Correlations when day 28 values for fat oxidation were used were weaker and no longer statistically significant. For LCT consumption, only EE and fat oxidation on day 2 were related to change in BW ($r=-0.582$, $P=0.009$ and $r=-0.500$, $P=0.029$, for EE and fat oxidation, respectively). Changes in AT compartment volumes with LCT consumption were not related to EE or fat oxidation on either day 2 or 28.

We then examined whether subjects with greater volumes of LT had more pronounced changes in body composition. With data from both treatment phases grouped, LT volume on day 2 was not associated with any change in AT compartment. When correlations were done for each treatment separately, there was a correlation between LT volume on day 2 and change in intramuscular AT, but only during LCT consumption ($r=0.524$, $P=0.0082$).

Discussion

This trial is the first to link responsiveness to a thermogenic oil to initial BW as well as to changes in BW and body compartment volumes. Results from this study show that, in overweight men, those with lower BW had the greatest rise in EE and fat oxidation when replacing dietary LCT with MCT. It was previously suggested that MCT could be used in the treatment of obesity;[10] however, the results reported here seem to indicate that MCT may be a better tool in the prevention of weight gain when BW is not yet highly elevated.

Few trials have compared the effects of LCT substitution by MCT in overweight and lean subjects.[10,20] In the study by Scalfi *et al*,[10] normal weight and morbidly obese men did not differ in their postprandial response to MCT compared to LCT; both groups had similar increases in postprandial EE over RMR values. However, as observed in the present analyses, postprandial respiratory quotient was reduced with MCT consumption relative to LCT, indicative of greater fat oxidation, in the normal weight but not in the obese subjects. In a similar experiment in women,[20] cumulative exogenous lipid oxidation over 6 h increased from 3.2 g with LCT consumption to 8.1 g with MCT consumption in obese and from 6.0 to 9.2 g with LCT compared to MCT consumption, respectively, in normal weight subjects. When total lipid oxidation was compared between normal weight and obese subjects, it was found that in the normal weight, but not obese women, total lipid oxidation was greater after the MCT-containing challenge than after the LCT challenge. Again, these results are in agreement with those observed in this experiment, where subjects of lower BW had a greater rise in fat oxidation with replacement of LCT by MCT in the diet compared to those with higher initial BW. This may be due to enhanced uptake of dietary fatty acids by the greater adipose tissue mass of overweight subjects, in whom lipoprotein lipase activity is increased.[21] Release of exogenous fatty acids into plasma has previously been shown to be defective in obese women compared to normal weight women,[20] possibly due to greater uptake by elevated AT stores. The results from this study show that fat oxidation and EE were enhanced to a greater degree with MCT consumption compared to LCT in men of lower BW. This may be related to the quantity of VAT stores, which would favor greater fat uptake for storage rather than oxidation.

It is unknown, at this time, why a trend towards greater rise in fat oxidation with MCT consumption relative to LCT was linked to greater initial BW early in the trial, whereas a significant correlation in the opposite direction was seen after 4 weeks of dietary treatment. It may be that MCT helps to restore rates of fat oxidation early on, but that a thermogenic adaptation occurs whereby differences in fat oxidation between MCT and LCT are diminished. In the study by Binnert *et al*,[20] it was reported that MCT oxidation is similar in obese and normal weight women, whereas LCT oxidation seems to be defective in obese compared to normal weight women. Since their observations only extended over a single meal, it is not known whether long-term feeding would have resulted in maintenance of the improved fat oxidation noted with MCT consumption.

Although not unexpected, data showing that subjects with the greater rise in fat oxidation had the greatest loss of BW and SAT support the purported mechanism of action of MCT on BW. It has been hypothesized that the direct transport of MCT to the liver by the portal circulation diminishes their potential for deposition into AT.[5] MCT are rapidly and almost completely oxidized by the liver, further reducing the probability that they will be elongated and returned to the circulation, where deposition into AT is more likely.[5,22] The results reported here show that, in fact, greater fat oxidation

with MCT was correlated with smaller SAT depots. Moreover, subjects with the greatest difference in fat oxidation between MCT and LCT consumption also tended to have the greatest loss of BW. However, these data must be interpreted with caution since we observed no significant difference in loss of SAT between the two diets. Nevertheless, MCT consumption led to a significant decrease in TAT, SAT, and upper body AT, whereas LCT consumption did not.

Our research demonstrates that an oil rich in MCT increases EE and fat oxidation and leads to beneficial body composition changes. Previous studies from our group have shown that MCT, when combined with flaxseed oil and phytosterols, also improve plasma lipid profiles in men[23] and women.[24] These effects make this oil combination a suitable candidate for classification as a functional food. A functional food is a food that is similar in appearance to a conventional food, but that provides demonstrated health benefits or reduces the risk of chronic disease above and beyond its basic nutritional functions. Our MCT-containing oil meets this definition since it increases EE and fat oxidation to a greater extent than a conventional LCT-containing oil, helps reduce body fatness, and decreases plasma lipid concentrations. These effects can lead to lower risk of developing obesity and cardiovascular disease, two prevalent debilitating conditions in Western societies. However, MCT are not readily available in the American diet but are found in small quantities in coconut oil and dairy fats. Sevenhuysen *et al*[25] reported that middle-aged women consume on an average 15 g/day of butter as added fat on bread and potatoes. If we consider that 3.5% of fatty acids in butter are the medium-chain fatty acids, octanoic and decanoic acids,[26] this would represent an intake of MCT of approximately 0.52 g/day. However, the versatility of MCT oil, and its tasteless and odorless properties[22] make it suitable for incorporation into a variety of different food items.

Finally, the replacement of dietary LCT with MCT in a typical North American diet for a period of 28 days leads to significant increases in EE and fat oxidation. In addition, BW and AT compartment volumes were diminished when MCT was consumed in place of LCT. Furthermore, correlation analyses show that the rise in fat oxidation with MCT relative to LCT consumption was responsible for the greater loss of BW. Also, subjects with the greatest EE and fat oxidation rates also had the greatest loss of BW. These beneficial effects of LCT replacement by MCT on EE and fat oxidation were correlated with initial BW. Therefore, these results show that substitution of dietary LCT for MCT can be beneficial in the prevention of weight gain in subjects with lower BW, but may not be as useful to those with greater BW.

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