Diacylglycerols affect substrate oxidation and appetite in humans¹–³

Marleen MJW Kamphuis, David J Mela, and Margriet S Westerterp-Plantenga

ABSTRACT

Background: Meals rich in diacylglycerols (DGs) instead of triacylglycerols (TGs) show beneficial effects on lipid metabolism and energy balance. These effects are probably attributable to differences in DG and TG metabolism, especially postprandial fat oxidation.

Objective: We assessed the effects of partial replacement of TGs with DGs on substrate oxidation, energy expenditure (EE), relevant blood variables, and appetite.

Design: Twelve healthy, dietarily unrestrained women participated in a single-blind, placebo-controlled, randomized trial with crossover design. For 3 d before and throughout a 36-h stay in a respiration chamber, subjects were fed energy-maintenance amounts of a diet consisting of 55% of energy from carbohydrate, 15% from protein, and 30% from fat. In the respiration chamber, 40% of the fat was consumed as DG-rich (80% DGs) oil or as TG-based control oil with a similar fatty acid profile.

Results: Fat oxidation was significantly higher with DG treatment than with TG treatment. Appetite profiles during day 1 (24 h) did not differ significantly between the DG and TG treatments; however, feelings of hunger, appetite, estimated prospective food intake, and desire to eat were all significantly lower on day 2 (12 h) with DG treatment. Mean plasma β-hydroxybutyrate tended to be higher with DG treatment, and the difference between the 2 treatments was significant at 1130 on day 2. Plasma lipid concentrations and resting and 24-h EE did not differ significantly between the 2 treatments.

Conclusion: Consumption of DGs in place of TGs does not alter EE but produces metabolic effects, particularly increases in fat oxidation, which may be associated with improved appetite control and energy balance. Am J Clin Nutr 2003;77:1133–9.

KEY WORDS Substrate oxidation, thermogenesis, energy expenditure, appetite, diacylglycerols, respiration chamber, women

INTRODUCTION

The incidence and prevalence of obesity are increasing in the United States (1) and Europe (2) and throughout the developed and developing world. Obesity is the result of an imbalance between energy intake (EI) and energy expenditure (EE), by which surplus EI is stored as triacylglycerol in adipose tissue. Prevention and treatment of obesity are major public health challenges. Although weight loss can readily be achieved experimentally, long-term weight maintenance is often unsuccessful (3–5). Therefore, food ingredients affecting energy metabolism, eg, EE, fat oxidation, satiety, and EI, may help people to successfully control or manage their weight.

One of those ingredients may be diacylglycerols (DGs). Fat consumed in the diet normally consists of triacylglycerols (TGs), although small amounts of DGs are usually present. Studies in rats and humans suggest that modest intakes of DGs may have a beneficial effect on lipid metabolism. Compared with consumption of TGs, consumption of DGs may produce less postprandial elevation in plasma TG concentrations in humans (6, 7) and lower fasting serum TG concentrations in rats (8, 9) and humans (10). Consumption of DGs also reduces total body fat accumulation (11) and visceral fat accumulation (12) in rats and humans (13, 14). Although the mechanisms are still speculative, these effects are most likely attributable to differences in DG utilization, especially promotion of enhanced (postprandial) β-oxidation (9, 14). This enhanced β-oxidation is probably due to the enhanced postabsorptive availability of free fatty acids (FFAs) in the portal circulation. Notably, DG oil has the same digestion and absorption routes that comparable TG oils have and has bioavailability and a physiologic fuel value that are similar to those of comparable TG oils (15).

The aim of the present study was to determine whether DG and TG (as control) treatment have different effects on substrate oxidation, energy metabolism, postprandial plasma TG and β-hydroxybutyrate (BHB) concentrations, and appetite in humans. We assessed these variables under controlled conditions, ie, in a respiration chamber. We hypothesized that DG treatment would lead to greater fat oxidation and therefore lower respiratory quotients (RQs) than would TG treatment. Furthermore, we hypothesized that in comparison with TG treatment, DG treatment would produce lower postprandial TG concentrations and higher BHB concentrations as a consequence of greater hepatic fatty acid oxidation. Because hepatic fat oxidation is inversely associated with appetite and food intake (16, 17), we hypothesized that DGs and TGs also differentially affect measures of appetite.

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Table 1: Baseline characteristics of the 12 female subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>34.5 ± 9.4</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>71.3 ± 7.4</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.69 ± 0.06</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.9 ± 1.8</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>32.5 ± 5.3</td>
</tr>
<tr>
<td>F1 score</td>
<td>7.8 ± 3.5</td>
</tr>
<tr>
<td>F2 score</td>
<td>5.3 ± 2.9</td>
</tr>
<tr>
<td>F3 score</td>
<td>3.6 ± 2.5</td>
</tr>
<tr>
<td>HP score</td>
<td>14.3 ± 2.6</td>
</tr>
</tbody>
</table>

Total body water was obtained by dividing the measured deuterium dilution space by 1.04 (21). Percentage of body fat was calculated by using the 3-compartment model of Siri (22).

Table 2: Fatty acid composition of diacylglycerol (DG) and triacylglycerol (TG) oils

<table>
<thead>
<tr>
<th>Fatty Acid</th>
<th>DG</th>
<th>TG</th>
</tr>
</thead>
<tbody>
<tr>
<td>16:0</td>
<td>3.1</td>
<td>5.7</td>
</tr>
<tr>
<td>18:0</td>
<td>1.2</td>
<td>2.7</td>
</tr>
<tr>
<td>Total SFA</td>
<td>5.0</td>
<td>9.1</td>
</tr>
<tr>
<td>18:1</td>
<td>37.3</td>
<td>36.3</td>
</tr>
<tr>
<td>18:2</td>
<td>48.3</td>
<td>48.4</td>
</tr>
<tr>
<td>18:3</td>
<td>8.2</td>
<td>5.2</td>
</tr>
</tbody>
</table>

\(^{1}\)SFA, saturated fatty acids.

SUBJECTS AND METHODS

Subjects

Twelve healthy, nonsmoking women were recruited by advertisements in local newspapers. Subjects had to have a body mass index (in kg/m²) of 23–30, be in good health, and report a stable medication and were at most moderate alcohol users. The degree of dietary restraint was measured by using Dutch language versions of the Three-Factor Eating Questionnaire (18, 19) and the Herman-Polivy restraint questionnaire (20).

Anthropometry

Anthropometric measurements were made at baseline. After the subjects had fasted overnight and voided and while they wore underwear or a swimsuit, their body weight was measured using a digital balance accurate to 0.005 kg (E1200; Mettler, Amsterdam, Netherlands). Body mass index was calculated as weight (kg) divided by height (m) squared. Body composition was determined by using a hydrodensitometry and deuterium dilution (2H₂O) technique (21) and was calculated by using the combined equation of Siri (22).

Whole-body density was determined by underwater weighing, and residual lung volume was simultaneously measured by using the helium dilution technique (Volugraph 2000; Mijnhardt, Bunnik, Netherlands). The dilution of the deuterium isotope is a measure of total body water (23). Subjects were asked to collect a urine sample in the evening just before drinking a weighed amount of deuterium-enriched water. After ingestion of the deuterium solution, no further fluid or food consumption was permitted. A second urine sample (second voiding) was collected 10 h after ingestion of the deuterium solution. Deuterium concentrations in these urine samples were measured by using an isotope ratio mass spectrometer (Micromass Optima, Manchester, United Kingdom).

Subjects underwent two 36-h sessions in a respiration chamber for indirect calorimetry, EE, and substrate oxidation measurements. The 2 respiration chamber sessions were conducted 4 wk apart so that the subjects would be in the same phase of their menstrual cycle. At one session, 40% of fat was provided as the DG-rich oil, whereas in the other session, 40% of fat was provided as the TG control oil. The order of DG and TG was randomized. For 3 d before each respiration chamber session, the subjects were required to consume an energy-maintenance diet provided by the experimenters. The energy content of the diet was calculated for each subject by multiplying her estimated basal metabolic rate (BMR) by a physical activity level of 1.75. BMR was estimated by using the Harris-Benedict equation (24).

Respiration chamber protocol

The respiration chamber was a 14-m³ room furnished with a bed, chair, computer, television, radio and cassette player, telephone, intercom, sink, and toilet. Subjects entered the chamber at 0730 on day 1 of each experimental period. The experimental period then started at 0800 on day 1 and finished at 2000 on day 2. Full details of the procedures, diets, and data-collection procedures are given below.

Energy intake during respiration chamber experiments

During the 36-h respiration experiments, the subjects were fed in such a way that they would be in energy balance [estimated as...
BMR × 1.6; mean (±SD) EI on days 1 (24 h) and 2 (12 h) were 10.2 ± 0.7 and 6.2 ± 0.5 MJ, respectively (25). On day 1, all meals served in the respiration chamber consisted of 55% of energy from carbohydrate, 15% from protein, and 30% from fat. EI was divided over the meals as 20% for breakfast, 30% for lunch, 40% for dinner, and 10% for the evening snack. With breakfast, lunch, and dinner, 40% of the fat (ie, 12% of EI) was taken as the DG-rich or TG oil mixed with yogurt. Mean fat intake from the DG-rich oil was 33.0 ± 2.3 g (26.4 ± 1.9 g DG) on day 1. During the stay in the respiration chamber, breakfast was served at 0800, lunch at 1230, dinner at 1700, and the evening snack at 2130. All meals had to be finished within 0.5 h after being served. Subjects had ad libitum access to water for coffee or tea. Coffee and tea consumption was according to the subjects’ usual habits. Subjects were requested to go to sleep at 2300.

On day 2, EI was divided over the meals as 20% for breakfast and 30% for lunch, but no dinner was served. At 1700 the subjects were given a snack that contained 10% of daily EI and consisted of 70% of energy from carbohydrate, 15% from protein, and 15% from fat. The fat contained in the snack was comprised entirely of DG-rich or TG oil. Breakfast and lunch were served at 0800 and 1230, respectively. Breakfast and lunch each consisted of 55% of energy from carbohydrate, 15% from protein, and 30% from fat. Mean fat intake from DG-rich oil on day 2 was 22.2 ± 1.4 g (17.8 ± 1.1 g DG). All meals had to be finished within 0.5 h after being served. Subjects had ad libitum access to water for coffee or tea.

Physical activity

In the respiration chamber, the subjects were required to follow a low-intensity activity protocol consisting of bench-stepping exercises. These exercises were performed twice a day, at 1200 and at 1630, for 30 min with 5-min intervals of exercise alternated with 5 min of rest. Apart from the exercise protocol, the subjects were not restricted in their activities, except that sleeping and strenuous physical activity were not allowed. Physical activity was monitored by using a radar system that operated on the Doppler principle.

Energy expenditure and substrate oxidation

Oxygen consumption and carbon dioxide production were measured in the respiration chamber. The temperature was set at 21°C during the daytime and at 18°C during the night. The room was ventilated with fresh air at a rate of 70–80 L/min. The ventilation rate was measured with a dry gas meter (type 4; Schumberger, Dordrecht, Netherlands). The concentrations of oxygen and carbon dioxide were measured by using a paramagnetic oxygen analyzer (Magnos 6G from Hartmann and Braun, Frankfurt, Germany, and OA184A from Servomex, Crowborough, United Kingdom) and an infrared carbon dioxide analyzer (Uras 3G; Hartmann and Braun). Six samples of outgoing chamber air for each subject and one sample each of fresh air, zero gas (nitrogen), and calibration gas were measured during respective 15-min periods. The collection of gas samples to be measured was controlled by a computer that also stored and processed the data (26).

Twenty-four-hour EE consisted of sleeping metabolic rate (SMR), diet-induced thermogenesis (DIT), and activity-induced EE (AEE). Twenty-four-hour EE was calculated twice during the 36-h stay in the respiration chamber by using overlapping data: from 0800 on day 1 to 0800 on day 2 (EE 1) and from 2000 on day 1 to 2000 on day 2 (EE 2). SMR was defined as the lowest mean EE over 3 consecutive hours between 0000 and 0700. DIT was calculated by plotting EE against radar output, both of which were averaged over 30-min periods. The intercept of the regression line at the lowest radar output represented the EE in the inactive state (RMR), consisting of SMR and DIT (25). DIT was calculated as RMR minus SMR. AEE was determined by subtracting RMR from 24-h EE. AEE and DIT were calculated twice per respiration chamber period (AEE 1 and AEE 2, and DIT 1 and DIT 2) by using the same overlapping time periods that were used for EE 1 and EE 2.

RQ was calculated as carbon dioxide production divided by oxygen consumption. Twenty-four-hour RQ was calculated twice during each 36-h stay in the respiration chamber (RQ 1 and RQ 2) by using the same time periods as those used for EE 1 and EE 2. Mean RQ was calculated over 36 h.

Carbohydrate and fat oxidation were calculated by using oxygen consumption and carbon dioxide production as follows (27):

\[
\text{Carbohydrate oxidation (g/d) } = 4.17 \times \dot{V} \text{CO}_2 - 2.965 \times \dot{V} \text{O}_2 - 0.390 \times P
\]

\[
\text{Fat oxidation (g/d) } = 1.718 \times \dot{V} \text{O}_2 - 1.718 \times \dot{V} \text{CO}_2 - 0.315 \times P
\]

where \( \dot{V} \text{O}_2 \) is oxygen consumption (L/d), \( \dot{V} \text{CO}_2 \) is carbon dioxide production (L/d), and \( P \) is protein oxidation (g/d) calculated from protein intake. Carbohydrate and fat oxidation were calculated twice by using the same time periods as those used for EE 1 and EE 2.

Blood analysis

At the screening, a baseline 10-mL blood sample was obtained from the subjects after they had fasted overnight. During respiration chamber sessions, postprandial blood samples were taken at 1130 and 1600 each day. An air lock was constructed for blood sampling during the respiration chamber sessions. After each subject opened the air lock on the inside, she wrapped a plastic bag tied to the air lock tightly around her upper arm so that no air from inside the respiration chamber could escape. After that, the air lock was opened on the outside and each subject stretched her arm out so that a blood sample could be taken. The blood samples were mixed with EDTA to prevent clotting. Plasma was obtained by centrifugation (type 1302; Heltich Zentrifugen, Tuttlingen, Germany) at 4°C and 3000 rpm for 10 min and stored at –80°C until analyzed. Glucose was analyzed by a hexokinase method (Roche Diagnostics, Basel, Switzerland); TG, by the method of McGowan et al (28) (GPO Trinder 337; Sigma, St Louis); glycerol, by a glycerokinase-lipase method (Boehringer, Mannheim, Germany); FFAs, by an ACS-ACOD method (Wako Chemicals, Neuss, Germany); BHB, by the method of Moore et al (29) with a semiautomated centrifugal spectrophotometer (Cobas Fara, Roche Diagnostics); and insulin, by an enzyme-linked immunosorbent assay (10-1113-01; Mercodia, Uppsala, Sweden). Mean plasma glucose, TG, glycerol, FFAs, BHB, and insulin concentrations were calculated as the mean of the 4 blood samples taken during the 36-h stay in the respiration chamber.

Appetite

Appetite variables (hunger, fullness, appetite, satiety, thirst, estimate of prospective food intake, and desire to eat) were measured with anchored 100-mm line scales. The scales were anchored
FIGURE 1. Mean 24-h energy expenditure, consisting of sleeping metabolic rate (■), diet-induced thermogenesis (□), and activity-based energy expenditure (■), of 12 women during treatment with diacylglycerol (DG)-rich oil or triacylglycerol (TG) control oil. There were no significant differences between the treatments.

FIGURE 2. Mean (± SD) fat oxidation in 12 women during days 1 and 2 of treatment with diacylglycerol-rich oil (■) or triacylglycerol (TG) control oil (□). * Significantly different from TG (paired t test); * P < 0.05, ** P = 0.004.

with phrases such as “not hungry at all” at the left anchor and “extremely hungry” at the right anchor.

Appetite variables were measured 10 times on day 1: before and after breakfast, during the morning, before and after lunch, during the afternoon, before and after dinner, during the evening, and before sleeping. Appetite variables were measured 9 times on day 2: before and after breakfast, during the morning, before and after lunch, during the afternoon, before and after the snack, and before leaving the respiration chamber.

Twenty-four-hour (0800 on day 1 to 0800 on day 2) appetite scores were calculated as the area under the curve by interpolating between the last measurement on day 1 and the first measurement on day 2. The area under the curve on day 2 (0800 to 2000) was also calculated.

Statistical analysis

All data are presented as means ± SDs. Differences in blood variables between the DG and TG treatments were tested by using a one- or two-tailed, two-factor repeated-measures analysis of variance (treatment × time of day), depending on our hypothesis. Post hoc analysis was done with a paired t test. The same analysis could not be executed for the other variables, ie, energy balance, 24-h EE, SMR, DIT, AEE, fat and carbohydrate oxidation, RQ, and appetite. For EE and substrate oxidation, this analysis could not be executed because of the overlapping time between days 1 and 2. For the appetite variables, this analysis could not be executed because of differences in time span. Therefore, differences between values for those variables were analyzed by using a one- or two-tailed paired t test, depending on our hypothesis.

Because of problems with blood sampling, the data analysis for the blood variables was based on only 10 subjects. Because only 5 subjects completely rated their appetite on day 2 of the 36-h stay in the respiration chamber, appetite scores on day 2 were analyzed with a two-tailed Wilcoxon signed-ranks test.

All statistical analyses were conducted with the use of STATVIEW SE GRAPHICS software (version 4.5; Abacus Concepts Inc, Berkeley, CA), and the criterion for significance was set at P < 0.05.

RESULTS

Energy balance

On the basis of estimated EE, 24-h EI was intended to achieve energy balance for each subject. Actual energy balance was determined by subtracting measured EE from EI. This was done only for day 1 in the respiration chamber, because on day 2 the subjects received only 60% of their calculated 24-h EI. Energy balance with the DG treatment was 0.7 ± 0.7 MJ and was not significantly different from that with the TG treatment (0.8 ± 0.5 MJ), but both were significantly different from zero (both P < 0.01), indicating a slightly positive energy balance.

Energy expenditure

Mean 24-h EE and its components are shown in Figure 1. EE during the DG treatment did not differ significantly from that during the TG treatment. The values for SMR, DIT, and AEE during the DG treatment also did not differ significantly from those during the TG treatment.

Substrate oxidation

Fat oxidation (Figure 2) on day 1 was significantly higher (P = 0.004) with the DG treatment (60.7 ± 15.8 g/d) than with the TG treatment (55.8 ± 14.4 g/d). On day 2, fat oxidation was also significantly higher (P < 0.05) with the DG treatment (64.6 ± 16.1 g/d) than with the TG treatment (60.6 ± 13.7 g/d). Carbohydrate
oxidation did not differ significantly between the DG and TG treatments (150.8 ± 23.8 and 164.3 ± 20.0 g/d, respectively) on the first day in the respiration chamber. On day 2, a trend toward lower carbohydrate oxidation was observed with the DG treatment than with the TG treatment (158.0 ± 24.1 and 171.2 ± 19.7 g/d, respectively; P < 0.1).

Twenty-four-hour RQs for days 1 and 2, as well as 36-h mean RQs, are shown in Table 3. All RQs were significantly lower during the DG treatment than during the TG treatment, indicating higher fat oxidation with DG treatment than with TG treatment.

**Blood variables**

Plasma concentrations of glucose, TG, glycerol, FFAs, BHB, and insulin at baseline (fasted) and on days 1 and 2 (mean values from 2 samples) of each 36-h stay in the respiration chamber are shown in Table 4. There were no significant differences between the DG and TG treatments in glucose, TG, glycerol, FFA, and insulin concentrations on days 1 and 2. However, there was a significant (P < 0.05) treatment × time of day effect for BHB on day 2. Post hoc analysis showed that BHB concentrations at 1130 on day 2 of the DG treatment (350.2 ± 233.1 μmol/L) were significantly higher (P < 0.05) than those at the corresponding time on the DG treatment (208.8 ± 70.8 μmol/L), indicating greater hepatic fat oxidation with the DG treatment than with the TG treatment. However, no other significant differences in BHB concentrations were observed between the 2 treatments.

**Appetite**

There were no significant differences between the DG and TG treatments in 24-h appetite profiles on day 1 (Table 5). However, with the DG treatment, the areas under the curve over 12 h on day 2 (Table 6) for feelings of hunger, appetite, estimate of prospective food intake, and desire to eat were significantly lower than those for the TG treatment. No significant differences between the 2 treatments were seen for feelings of fullness, satiety, or thirst.

**DISCUSSION**

Compared with consumption of TG oil, consumption of DG-rich oil (80% DG) for 36 h was associated with greater fat oxidation and lower appetite, but there were no significant differences between the 2 treatments in total EE or blood variables. The results of the present study support the hypothesis that DG increases β-oxidation, ie, that it enhances fat oxidation and lowers the RQ. Evidence of higher BHB concentrations during the DG treatment than during the TG treatment points to higher hepatic fat oxidation with DG treatment. However, DG consumption did not affect postprandial TG concentrations or total 24-h EE. The data suggest that carbohydrate oxidation was lower with the DG treatment than with the TG treatment, but these data did not differ significantly between the 2 treatments. Although appetite profiles over day 1 did not differ significantly between the DG and TG treatments, feelings of hunger, appetite, estimated prospective food intake, and desire to eat on day 2 were significantly lower with the DG treatment than with the TG treatment.

Several studies indicated that in comparison with TG oil, dietary DG oils have beneficial effects on lipid metabolism. Consumption of DGs has been shown to produce less postprandial elevation in TG concentrations in humans (6, 7) and lower serum TG concentrations in humans (10) and rats (8, 9). Increased concentrations of ketone bodies in the serum of human subjects (7) and in the urine of rats (11) have been taken as an indication of higher fat oxidation. Long-term DG consumption has been reported to reduce body fat accumulation in rats (11) and visceral fat in rats (12) and humans (13, 14).

Our observations of higher fat oxidation and a lower RQ after consumption of the DG oil than after consumption of the TG oil are in line with the results of previous studies in animals that indicated alterations in fatty acid oxidation and synthesis in the liver (9). In that study, the activities of enzymes involved in β-oxidation and of enzymes necessary for fatty acid synthesis in the liver were higher and lower, respectively, in rats fed DG than in those fed TG. This may have caused the decreased liver TG concentrations observed in that study. Indications of increased (hepatic) fat oxidation were also observed by Wanatabe et al (11). They reported that in comparison with TG, a single dose of DG produced higher oxygen consumption in rats for up to 80 min after administration.

**TABLE 4**

Plasma concentrations of free fatty acids (FFA), triacylglycerol (TG), glycerol, β-hydroxybutyrate (BHB), glucose, and insulin at baseline (fasted) and on days 1 and 2 (mean values from 2 samples) of treatment with diacylglycerol (DG)-rich oil or triacylglycerol (TG) control oil.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 1</th>
<th>Day 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>FFA (μmol/L)</td>
<td>279 ± 112</td>
<td>203 ± 136</td>
<td>181 ± 126</td>
<td>190 ± 142</td>
<td>135 ± 89</td>
</tr>
<tr>
<td>TG (μmol/L)</td>
<td>1125 ± 621</td>
<td>1288 ± 648</td>
<td>1374 ± 533</td>
<td>1254 ± 369</td>
<td>1320 ± 463</td>
</tr>
<tr>
<td>Glyceral (μmol/L)</td>
<td>85 ± 33</td>
<td>56 ± 21</td>
<td>57 ± 26</td>
<td>64 ± 33</td>
<td>53 ± 21</td>
</tr>
<tr>
<td>BHB (μmol/L)</td>
<td>233 ± 85</td>
<td>251 ± 129</td>
<td>274 ± 188</td>
<td>226 ± 136</td>
<td>209 ± 76</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.1 ± 0.5</td>
<td>5.1 ± 0.4</td>
<td>5.2 ± 0.5</td>
<td>5.1 ± 0.4</td>
<td>5.2 ± 0.6</td>
</tr>
<tr>
<td>Insulin (mU/L)</td>
<td>9 ± 4</td>
<td>14 ± 9</td>
<td>17 ± 11</td>
<td>18 ± 17</td>
<td>16 ± 10</td>
</tr>
</tbody>
</table>

1x ± SD; n = 10. During the DG and TG treatments, blood samples were taken at 1130 and 1600 on days 1 and 2. There were no significant differences between the treatments. However, BHB concentrations at 1130 on day 2 of the DG treatment (350.2 ± 233.1 μmol/L) were significantly higher (P < 0.05, post hoc analysis) than those at 1130 on day 2 of the TG treatment (208.8 ± 70.8 μmol/L).
The main end products of lipase action on 1,3-DG are free glycerol and FFAs (8, 30). These end products of DG digestion may be less readily resynthesized to chylomicron TG because such synthesis requires a glycerol-3-phosphate intermediate. FFAs may therefore be more likely to be directly transported toward the liver, where they are used for β-oxidation. This may explain why replacement of TG by DG can lower serum TG in the fastest state (8–10) and in the postprandial state (6, 7) and produce a lower TG content in chylomicrons (6).

In the present study, replacement of dietary TG with a DG-rich oil increased fat oxidation by 4.9 g/d on day 1 and by 4 g/d on day 2. Although this effect seems small on a daily basis, a sustained change in fat balance of 4 g fat/d generates an effect of ≈1460 g fat/y. In our opinion, this is quite substantial. The difference in fat oxidation of 4 g/d could make a difference in the long term between successful and unsuccessful weight control, and therefore the replacement of TG with DG could contribute to the prevention of increasing body weight and related comorbidities.

In conclusion, the results of the present study support the proposed beneficial effects of consumption of DG rather than TG on body composition and lipid metabolism by providing direct evidence of higher fat oxidation and lower appetite scores in humans with DG treatment than with TG treatment. In the present study, replacement of modest amounts of TG with DG decreased RQs and increased fat oxidation. The higher BHB concentrations with DG treatment than with TG treatment. In the present study, replacement of dietary TG with a DG-rich oil increased fat oxidation by 4.9 g/d on day 1 and by 4 g/d on day 2. Although this effect seems small on a daily basis, a sustained change in fat balance of 4 g fat/d generates an effect of ≈1460 g fat/y. In our opinion, this is quite substantial. The difference in fat oxidation of 4 g/d could make a difference in the long term between successful and unsuccessful weight control, and therefore the replacement of TG with DG could contribute to the prevention of increasing body weight and related comorbidities.

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