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Increasing dietary palmitic acid decreases fat oxidation and daily energy expenditure[1,2,3]

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Abstract

Background—Oleic acid (OA) is oxidized more rapidly than is palmitic acid (PA).

Objective—We hypothesized that changing the dietary intakes of PA and OA would affect fatty acid oxidation and energy expenditure.

Design—A double-masked trial was conducted in 43 healthy young adults, who, after a 28-d, baseline, solid-food diet (41% of energy as fat, 8.4% as PA, and 13.1% as OA), were randomly assigned to one of two 28-d formula diets: high PA (40% of energy as fat, 16.8% as PA, and 16.4% as OA; $n = 21$) or high OA (40% of energy as fat, 1.7% as PA, and 31.4% as OA; $n = 22$). Differences in the change from baseline were evaluated by analysis of covariance.

Results—In the fed state, the respiratory quotient was lower ($P = 0.01$) with the high OA (0.86 ± 0.01) than with the high-PA (0.89 ± 0.01) diet, and the rate of fat oxidation was higher ($P = 0.03$) with the high-OA (0.0008 ± 0.0001) than with the high-PA (0.0005 ± 0.0001 mg · kg fat-free mass^{-1} · min^{-1}) diet. Resting energy expenditure in the fed and fasting states was not significantly different between groups. Change in daily energy expenditure in the high-OA group (9 ± 60 kcal/d) was significantly different from that in the high-PA group (−214 ±69 kcal/d; $P = 0.02$ or 0.04 when expressed per fat-free mass).

Conclusions—Increases in dietary PA decrease fat oxidation and daily energy expenditure, whereas decreases in PA and increases in OA had the opposite effect. Increases in dietary PA may increase the risk of obesity and insulin resistance.

Keywords

Palmitic acid; oleic acid; fat oxidation; energy expenditure; fatty acids

INTRODUCTION

The term “Mediterranean diet” typically refers to the dietary pattern of people living in Greece and southern Italy in the early 1960s; such persons had a long life expectancy and a low risk of coronary heart disease (1). The Mediterranean diet was low in saturated fatty acids (FAs;

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7–8% of energy) and moderate in fat (25–35% of energy), and the fat source consisted primarily of olive oil. Oleic acid (18:1; OA) constitutes ≈72% of the FAs in olive oil (1,2).

As opposed to oxidation, storage of FA in tissues other than adipose tissue may interfere with normal cellular function and lead to an increased risk of the metabolic syndrome (3–6). Studies using labeled FAs have shown that OA and other unsaturated FAs are more readily oxidized than are saturated FAs (SFAs) (7–11). These tracer data, per se, do not necessarily imply that changing the pattern of dietary FAs would affect total FA oxidation, but some studies have shown that humans or animals ingesting diets enriched with polyunsaturated FAs (PUFAs) and monounsaturated FAs (MUFAs) exhibit higher total FA oxidation, energy expenditure, or both than when ingesting diets containing more SFAs and less PUFAs or MUFAs (12–16). However, the effects of dietary FA composition on energy expenditure in rodents may be mediated by changes in brown adipose tissue metabolism (14,16) and, thus, are not relevant to humans. Thus, we conducted a dietary trial in humans to determine the effects on FA oxidation and energy expenditure of 2 different dietary FA patterns: one typical of the North American diet (equal contributions of OA and PA to the total dietary FAs) and one more typical of the Mediterranean diet (much higher OA and much lower PA).

SUBJECTS AND METHODS

Study design

Forty-three healthy, nonobese, young adults aged 21–34 y participated in a randomized, double-masked, controlled trial. The study was conducted at the General Clinical Research Center (GCRC) of The Ohio State University Medical Center, Columbus, OH (n = 3), and at the GCRC of the University of Texas Medical Branch (UTMB), Galveston, TX (n = 40). The protocol was approved by each institution's review committee for human subjects and the General Advisory Committee of the GCRC at the respective institutions. The same solid-food diet and formulas were used at both sites, and procedures for measuring energy expenditure and body composition were also similar. The inclusion criteria were as follows: age 18–35 y; healthy and no need for medication; body mass index (BMI; in kg/m^2) >18, <30, and below the 95th percentile (17); and no current use of drugs that could affect lipid metabolism, including nicotine. Subjects with a BMI >25 were included to extend the clinical relevance of the study to subjects at risk for the metabolic syndrome. Exclusion criteria were as follows: vegetarian diet, habitual fat intake < 30% of energy, pregnancy, fasting glucose, HDL-cholesterol concentration below the 5th percentile for age and sex, and total cholesterol, LDL-cholesterol, and triacylglycerol concentrations above the 95th percentile (18). As part of the screening history and physical examination, the subjects were asked whether any of their family members manifested or died from obesity, type 2 diabetes, or noncongenital heart disease.

Female subjects were initiated into the study regardless of their menstrual cycle, but each was studied at the same phase of the cycle for both the run-in and baseline diets and the experimental diet. There appeared to be only minor effects of the menstrual cycle on FA turnover (19) and thermic effect of feeding but none on resting energy expenditure (REE), FA oxidation, or the average REE after the meal (20). Moreover, one-half of our 22 female subjects were taking oral contraceptives and not ovulating.

Each subject was studied twice for 28 d, first during the consumption of a solid-food, run-in diet (baseline preformula value) and then during consumption of 1 of the 2 experimental liquid-formula diets (postformula value). Randomization to 1 of the 2 formula treatments was stratified for sex. All food and drinks, except for water, during both phases of the study were provided by the GCRC. Energy intake was adjusted during both diet phases to maintain body weight. All subjects were required to eat breakfast at the GCRC every day of the 8-wk study, but many of the subjects chose to eat one or more additional meals in our dining room each

day, especially during the week. Subjects who did not keep appointments or who had unusual changes in body weight in relation to energy intake, especially during the formula diet phase of the study (because the run-in diet provided a good approximation of energy needs), were dropped from the study (21). On this basis, one female subject in the high-PA group was excluded from the original data set; thus, although there were originally 44 subjects in the study (n = 22 in each formula group; 11 women and 11 men in each group), the results are reported for 43 subjects (n = 21 in the high-PA group; n = 22 ion the high-OA group).

The main purpose of the baseline, solid-food diet was to establish which subjects would comply with the protocol and to establish baseline data on all subjects on the same diet. We analyzed the content of the 9 separate meals composing the run-in diet (Covance Laboratories, Madison, WI). On the basis of the average of these 9 analyses, the composition of this solid-food diet was as follows: 14.6% of energy as protein, 45.1% of energy as carbohydrate, and 40.8% of energy as fat. The FA composition consisted of 13.1% of energy as OA (32.4 g/100 g total fat) and 8.4% of energy as PA (20.8 g/100 g fat). For comparison, a post hoc analysis of the dietary histories obtained from most of the participants for the period preceding the run-in diet indicated the following composition of the habitual diet: 18.0% of energy as protein, 49.6% of energy as carbohydrate, and 33.0% of energy as fat (11.0% of energy as SFA and 11.0% of energy as MUFA).

The macronutrient and FA compositions of the 2 experimental formula diets (Ross Products Division Inc, Abbott Laboratories Inc, Columbus, OH) are shown in Table 1. Compared with the baseline diet, the high-PA diet contained 100% more PA and 25% more OA as percentages of energy (n = 21); the high-OA diet was identical to the high-PA diet, except that, compared with the baseline diet, it contained 80% less PA and 140% more OA as percentages of energy (n = 22). The formulas were patterned after formulas used for nutritional support and contained adequate vitamins and minerals on a per kilocalorie basis. The fat blend in the high-PA formula was 91% palm oil, 6% high-OA sunflower oil, and 3% soy lecithin. The fat blend of the high-OA formula was 0% palm oil, 97% high-OA sunflower oil, and 3% soy lecithin. The 2 oils used in these formulas are naturally occurring and are not interesterified. Although fat absorption was not measured in this study, and fat intake did not prove to be a significant covariate in our statistical analysis, fat absorption is typically >90% for these oils. The positional distribution of FAs on the triacylglycerol molecule was typical for these oils. These oils contain naturally occurring phytochemicals, especially palm oil, but we have no basis for thinking that any of these compounds could affect the processes under study. These oils typically contain >95% triacylglycerol. Safflower oil contains ≈3% 1,3-diacylglycerol, but diacylglycerol is not detectable in palm oil.

On the 28th day of each diet period, after an evening meal at 1800 (≈33% of the daily energy intake), indirect calorimetry was performed overnight in both the fed and fasting states (22). Except for bedside bathroom privileges, the subjects remained in bed from 1700 to 0720. Oxygen consumption and carbon dioxide production (Vmax SPECTRA 29; Sensor Medics Corp, Yorba Linda, CA) were measured for 20 min each time at 60-min intervals after the meal for 7 h (fed state) and then at 120-min intervals until 13 h after the meal (fasting state) (22). Urinary urea nitrogen was measured during the 14-h interval, and protein oxidation was estimated for both the fed (1720– 0120) and the fasting (0120–0720) periods (22). REE and substrate utilization were calculated according to standard procedures by using urinary urea nitrogen measurements as estimates of protein oxidation (22–24) and the Weir equation (25). Urinary urea nitrogen was measured at the UTMB clinical laboratory according to the urease quinolinium dye method (Vitros 250 Chemistry System; Ortho-Clinical Diagnostics, Rochester, NY). Respiratory quotient (RQ), rates of fat and carbohydrate oxidation, and REE were estimated for both the fed and fasting states from the average value for each period (22). The flow sensor was manually calibrated with a syringe before each daily measurement.

This procedure was repeated if there seemed to be instability in the measurements during the overnight studies. The gas sensors were calibrated before each 20-min measurement.

Body composition was measured just before the solid-food diet began and at the end of each 28-d diet phase by dual-energy X-ray absorptiometry (Delphi QDR 4500A Bone Densitometer; Hologic, Bedford, MA), and body energy (BE) was estimated by using Atwater conversion values for fat mass (FM; 9.3 kcal/g) and body protein (4.1 kcal/g protein), assuming 0.2 g protein/g fat-free mass (FFM).

$$\begin{aligned} \text{BE (kcal)} = (9.3\ \text{kcal/g} \times \text{FM; in g}) + [4.1\ \text{kcal/g protein} \\ \times\ (1\ \text{g protein/5 g FFM}) \times \text{FFM; in g}] \end{aligned} \quad (1)$$

In addition, daily energy expenditure (DEE) was determined from the average energy intake (EI) and the change in BE (ΔBE) estimated from the dual-energy X-ray absorptiometry measurements:

$$\text{DEE} = \text{EI} - \Delta\text{BE} \quad (2)$$

where ΔBE is the BE at the end of the formula diet period (postformula value) minus the BE at the end of the run-in diet period (preformula value).

In 39 subjects (n = 21 in the high-OA group and n = 18 in the high-PA group), physical activity was estimated for 7d during the third week of each diet phase by using a uniaxial accelerometer worn on the wrist (model 71164; Computer Science and Applications, Manufacturing Technology, Fort Walton Beach, FL). Melanson and Freedson (26) previously showed that this position for the accelerometer provided the best index of energy expenditure in the field setting.

Statistical analysis

Results are expressed as means ± SEMs. The term statistical significance was applied to differences with a 2-tailed P value ≤0.05. The main approach to the statistical analysis was an analysis of covariance; this consisted of examining the change in the respective outcome variable during the experimental diet with the baseline value included as the covariate. All analyses were carried out by using either SAS (SAS System for WINDOWS, version 8.1; SAS Institute Inc, Cary, NC) (27) or SPSS (SPSS Base 10.0; SPSS Inc, Chicago, IL). Our central hypothesis was that the high-OA diet would be associated with a greater rate of fat oxidation and energy expenditure than would the high-PA diet, when corrected for the preformula value obtained from subjects consuming the same, run-in, solid-food diet.

RESULTS

Body composition

BMI was not significantly different between the high-PA and high-OA groups, either at the end of the run-in diet (23.7 ± 0.7 and 24.1 ± 0.6, respectively) or after the formula diet (23.7 ± 0.7 and 23.9 ± 0.6, respectively). At the end of the formula diet, BMI was >25.0 in 8 subjects in the high-PA group and in 6 subjects in the high-OA group (or >25.2 in 6 and 5, respectively). The groups did not manifest statistically significant differences in body composition before the formula diets (Table 2). However, there were group differences in body-composition changes during the experimental formula diets. There was a trend for a larger increase in fat mass in the high-PA group (0.52 ± 0.13 kg) than in the high-OA group (0.14 ± 0.14 kg) (P = 0.06). There was no significant difference between the high-PA and high-OA groups in the change in FFM (−0.39 ± 0.18 and −0.60 ± 0.24 kg, respectively; P = 0.52). This combination of body-composition changes resulted in an increase (P = 0.05) in body mass in the high-PA

group (0.1 ± 0.2 kg) and a decrease in body mass in the high-OA group (−0.5 ± 0.2). There was no significant difference between groups in the increase in percentage body fat. There was a substantially larger increase ($P = 0.04$) in BE in the high-PA group (4542 ± 1182 kcal) than in the high-OA group (835 ± 1207 kcal).

Substrate oxidation

Mean RQ in the fed state (RQ fed) decreased in the high-OA group (from 0.87 ± 0.01 to 0.86 ± 0.01) and increased in the high-PA group (0.88 ± 0.01 to 0.89 ± 0.01) ($P = 0.01$) during the formula diet (Table 3). Similarly, we observed a corresponding increase in the rate of fat oxidation in the fed state (g/min) during the formula diet in the high-OA group and a decrease in the high-PA group ($P = 0.04$; Table 3). When fat oxidation in the fed state was expressed as $g \cdot kg\ FFM^{-1} \cdot min^{-1}$, the value increased during the formula diet in the high-OA group (from 0.0007 ± 0.0001 to 0.0008 ± 0.0001) and decreased in the high-PA group (from 0.0007 ± 0.0001 to 0.0005 ± 0.0001) ($P = 0.03$). Conversely, the rate of carbohydrate oxidation in the fed state (g/min) increased less during the formula diet in the high-OA group (from 0.1311 ± 0.0096 to 0.1430 ± 0.0071) than in the high-PA group (from 0.1508 ± 0.0096 to 0.1743 ± 0.0092) ($P = 0.0.02$). There was no significant difference between groups in the change in fasting RQ or the fasting rate of fat oxidation (g/min) (Table 3). We also estimated the weighted average daily RQ, assuming that 17 h of the day represented the fed condition; the change in average daily RQ in the high-OA group (−0.0083 ± 0.0107) was significantly different from that in the high-PA group (+ 0.0085 ± 0.0080) ($P = 0.04$). The change in the average daily rate of fat oxidation (g/min) was not statistically significant ($P = 0.14$; Table 3). However, the average daily rate of carbohydrate oxidation increased less in the high-OA group (0.0107 ± 0.0113) than in the high-PA group (0.0191 ± 0.0095) ($P < 0.05$). There were no significant differences between experimental groups in the change in the fasting rate of carbohydrate oxidation or in the fed or fasting rates of protein oxidation. The addition of a family history of obesity, diabetes, or heart disease as additional covariates did not appreciably affect the *P* value for the change in RQ or rate of fat oxidation.

Physical activity, energy intake, and energy expenditure

Physical activity (accelerations/min) was not significantly different between the high-OA and high-PA groups, either during the run-in diet (989 ± 76 and 917 ± 58, respectively) or during the formula period (927 ± 89 and 890 ± 59, respectively). Pre-and postformula energy intakes were not significantly different between the 2 groups, and pre- and postformula fasting and fed REE values and weighted daily averages were not significantly different between the 2 groups (Table 4). However, DEE remained essentially unchanged in the high-OA group (9 ± 60 kcal/d) but decreased in the high-PA group (−214 ± 69 kcal/d; $P = 0.02$; Table 4). Similarly, DEE/FFM ($kcal \cdot kg^{-1} \cdot d^{-1}$) decreased in the high-PA group (−3.4 ± 1.4) but remained essentially the same in the high-OA group (0.5 ±1.3; $P = 0.04$; Table 4). The addition of a family history of obesity, diabetes, or heart disease as additional covariates did not appreciably affect the *P* value for the change in either DEE or DEE/FFM.

DISCUSSION

Increasing the PA intake lowered fat oxidation in the fed state, whereas lowering the PA intake and markedly increasing the OA intake had the opposite effect. Although our subjects who consumed the high-PA diet underwent a large increase in PA intake, the SFA intake of the high-PA group was still considered fairly typical for many residents of North America (28). At least one previous study in humans (12) has suggested that PUFAs, compared with SFAs, tend to increase total FA oxidation. Our study, which involved a long interval of feeding, showed that increasing the ratio of MUFAs to SFAs in the diet also increased fat oxidation without an apparent, partial, compensatory reduction in fasting FA oxidation (12). We should

emphasize that our measurements of fat oxidation and REE were carried out in the resting state, overnight, at the end of each study period. Perhaps the results would have been different had these measurements been made while the subjects were carrying out the usual tasks of daily living in a room calorimeter. These differential effects of dietary FA composition on total FA oxidation may be important because fat oxidation during feeding could affect the accumulation of lipid intermediates in skeletal muscle that, in turn, affect insulin sensitivity (3,29–32). This study did not examine the mechanisms for the results we observed, but our observations are consistent with the concept that dietary FAs may affect the fundamental mechanisms for mitochondrial fatty acid oxidation (33–37).

Our study also suggests that an increase in the ratio of SFAs to MUFAs results in a change in body composition. After being corrected for energy intake, the calculated total DEE decreased in the high-PA group but increased modestly in the high-OA group. These results are partially supported by previous studies in rats, which showed that FAs have differential effects on the thermic effect of food (16). The high-OA group showed little change in DEE from the preformula to the postformula period (9 ± 60 kcal/d), whereas the high-PA group manifested a decrease in DEE of 214 ± 69 kcal/d. Although our method of statistical analysis corrects for individual differences in body composition, sex, and other genetic factors, we found similar results when DEE was expressed as a function of FFM. The absolute value for the difference between groups in the change in DEE during the formula diet period was 223 kcal/d. A lower DEE of this magnitude would equate to ≈9.0 kg fat/y if all of the surfeit energy intake was stored in adipose tissue. However, this magnitude of adipose tissue gain would be lessened by compensatory changes in food energy intake and by increased fat-free mass accretion. Some (13,14,16) but not all (38) previous studies of dietary FA composition in rodents have suggested that an increase in the fractional SFA content of the diet tends to lower energy expenditure. Because the mechanism for this effect could be decreased thermogenesis in brown adipose tissue (14,16), it is of interest that one previous study in humans (12) suggests that increased SFA intake tends to decrease the thermic effect of food.

Our subjects were free-living, and, because energy intake is used to calculate DEE, its estimation determines, in part, the validity of reported DEE. The values recorded for food intake were the prescribed values, and their validity is obviously dependent on subject compliance with the protocol. We can propose no plausible reason for why only one dietary group (high PA) systematically would choose to eat additional food during the formula diet, which was prohibited by the protocol. However, ingestion of unmeasured, additional food energy would lead to a relative gain in BE for the recorded energy intake; thus, calculated DEE would be artifactually low. Thus, it seems unlikely that the difference in DEE between groups would be affected by errors in estimation of food intake, but there still may be inherent uncertainty in the magnitude of the difference between groups.

The experimental diets did not differentially affect REE in either the fed or fasting states, and yet, DEE decreased in the high-PA group. Although there is no apparent reason why the diets would affect physical activity, physical activity was also not different between groups. We do acknowledge that REE was assessed only on the last day of each diet period, whereas the estimation of DEE was based on the entire 28 d of observation in both diet groups. Therefore, it is possible that the REE value did not totally reflect REE during the entire study period. Nevertheless, because DEE, but not REE and physical activity, was apparently affected differentially by the diets, one could surmise that dietary PA and OA may have had differential effects on the energy cost of physical activity. There is emerging evidence that dietary and endogenous FAs, as well as the genes regulating their metabolism, affect mitochondrial pathways for FA oxidation and energy utilization (34,39–42). Because mitochondrial FA oxidation and energy generation may be especially important during states in which the demand for energy production is greater (such as physical activity), perhaps changes in mitochondrial

function could explain why the diets affected DEE (body composition) but not REE. DEE also might be altered by the differential oxidation of dietary FAs via the peroxisomal β-oxidation pathway (43,44). Finally, the energetic efficiency of muscle contraction can be affected by changes in muscle fiber type or in the expression of specific muscle proteins (45).

In our study, for practical reasons relating to our subjects' need to work and go to school, REE was measured in the early evening and overnight. Although not reported here, REE measured just before the evening meal was sometimes higher than the values measured just after the meal, which suggests that physical activity increased energy expenditure but that this effect was dissipated while the subjects rested in bed during indirect calorimetry. DEE was expressed on a daily basis but was calculated from the average daily energy intake and the changes in body composition over the 28-d study period. So, the marked difference in the results derived from the REE measurement and the DEE estimate suggests that the diets could have differentially affected energy expenditure during the daytime, when the subjects were also physically active to varying degrees. The oxidation of OA, but not of PA, seems to be relatively increased by prior exercise, which in our free-living subjects was apt to occur during the daytime hours (46). Recently, Stavinoha et al (47) provided evidence that the responsiveness of genes targeted by peroxisome-proliferator activated receptor α is enhanced during the dark cycle of rats, when the animals presumably feed or at least search for food in the wild. If these data can be extrapolated to humans, who tend to eat during the day, this mechanism may be relevant to our results, ie, there could be an interaction between the thermic effect of food and exercise that could not be detected with the design of this study.

In conclusion, increases in dietary PA lowered FA oxidation and increased BE in healthy, nonobese, young adults. Thus, calculated DEE decreased in the high-PA group. In contrast, the change to a Mediterranean-type fat pattern resulted in increased fat oxidation in the fed state and prevented the decrease in DEE that was observed with the high-PA diet. These findings may have relevance to the prevention of obesity, the metabolic syndrome, or type 2 diabetes. The results may have been the consequence of fundamental alterations in gene or protein expression.

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CLK designed the study, recruited the subjects, supervised the study and data acquisition, analyzed the data, and prepared the manuscript. JYB analyzed the data, drafted the results section of the manuscript, and critically reviewed the manuscript. FU provided medical coverage in CLK's absence and assisted in the preparation of the manuscript. None of the authors had any conflict of interest to declare.

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TABLE 1

Composition of the experimental diets[1]

	High-PA diet	High-OA diet
Caloric distribution (% of energy)		
Protein	14	14
Carbohydrate	46	46
Fat	40	40
Fatty acid profile (g/100 g)		
Palmitic	42.1	4.2
Oleic	41.0	78.4
Linoleic	11.4	13.0
Stearic	4.1	4.1
α-Linolenic	0.3	0.3
Myristic	0.9	0.0
Palmitoleic	0.2	0.0
Eicosapentaenoic	0.0	0.0
Docosahexaenoic	0.0	0.0
Arachidonic	0.0	0.0
Fatty acid class (%)		
Saturated	47.1	8.3
Monounsaturated	41.2	78.4
Polyunsaturated	11.7	13.3
Fractional energy (%)		
Total saturated	18.8	3.3
12:0	0.0	0.0
14:0	0.4	0.0
16:0	16.8	1.7
18:0	1.6	1.6
Total monounsaturated	16.5	31.4
18:1n–9	16.4	31.4
Total polyunsaturated	4.8	5.3
18:3n–3	0.1	0.1
18:2n–6	4.6	5.2

[1] PA, palmitic acid; OA, oleic acid.

TABLE 2

Body composition of the high–oleic acid (OA) and the high–palmitic acid (PA) diet groups[1]

	Preformula phase		Postformula phase	
	High-PA diet (*n* = 22)	**High-PA diet (*n* = 21)**	**High-OA diet (*n* = 22)**	**High-PA diet (*n* = 21)**
BMI	24.1 ± 0.6	23.7 ± 0.7	23.9 ± 0.6	23.7 ± 0.7
DXA body mass	71.1 ± 2.5	70.0 ± 2.7	70.7 ± 2.5	70.1 ± 2.7[2]
Body fat (%)	25.1 ± 1.7	25.1 ± 1.6	25.5 ± 1.7	25.7 ± 1.7
FM (kg)	17.7 ± 1.3	17.5 ± 1.3	17.9 ± 1.3	18.1 ± 1.4
FFM (kg)	53.4 ± 2.4	52.4 ± 2.3	52.8 ± 2.4	52.0 ± 2.3
BE (kcal)	209 000 ± 12 000	206 000 ± 13 000	210 000 ± 12 000	211 000 ± 13 000
Change from baseline				
Body fat (%)	—	—	0.41 ± 0.22	0.66 ± 0.18
FM (kg)	—	—	0.14 ± 0.14	0.52 ± 0.13[3]
FFM (kg)	—	—	−0.60 ± 0.24	−0.39 ± 0.18
BE (kcal)	—	—	835 ± 1207	4542 ± 1182[4]

[1] All values are $\bar{x}$ ± SEM. BE, total body energy estimated from fat mass (FM) and fat-free mass (FFM) (*see* text for equation); DXA, dual-energy X-ray absorptiometry.

[2–4] Significantly different from the preformula high-OA diet (analysis of covariance):

[2] $P = 0.05$,

[3] $P = 0.06$,

[4] $P = 0.04$.

TABLE 3

Respiratory quotient (RQ) and fatty acid oxidation in the high–palmitic acid (PA) and high–oleic acid (OA) diet groups[1]

	Preformula phase		Postformula phase	
	High-OA diet (*n* = 22)	**High-PA diet (*n* = 21)**	**High-OA diet (*n* = 22)**	**High-PA diet (*n* = 21)**
Average RQ				
Fed state[2]	0.87 ± 0.01	0.88 ± 0.01	0.86 ± 0.01	0.89 ± 0.01[3]
Fasting state[4]	0.87 ± 0.01	0.88 ± 0.01	0.87 ± 0.01	0.88 ± 0.01
Fatty acid oxidation (g/min)[5]				
Fed state	0.040 ± 0.006	0.038 ± 0.006	0.043 ± 0.005	0.028 ± 0.005[6]
Fasting state	0.034 ± 0.005	0.034 ± 0.004	0.035 ± 0.005	0.031 ± 0.006
Average daily fatty acid oxidation (g/min)	0.038 ± 0.006	0.033 ± 0.004	0.040 ± 0.005	0.029 ± 0.005

[1] All values are $\bar{x}$ ± SEM.

[2] Estimated from individual measurements obtained 1, 2, 3, 4, 5, 6, and 7 h after the evening meal.

[3,6] Significantly different from the preformula high-OA diet (analysis of covariance):

[3] $P = 0.01$,

[6] $P = 0.04$.

[4] Estimated from observations made 9, 11, and 13 h after the evening meal.

[5] Estimated from the weighted average of the fat oxidation rate during the fed (17 h) and fasting (7 h) periods of the day.

TABLE 4

Energy intake (EI) and balance and resting (REE) and daily (DEE) energy expenditure in the high–palmitic acid (PA) and high–oleic acid (OA) diet groups[1]

	Preformula phase		Postformula phase	
	High-OA diet (*n* = 22)	**High-PA diet (*n* = 21)**	**High-OA diet (*n* = 22)**	**High-PA diet (*n* = 21)**
Average energy intake (kcal/d)	2704 ± 90	2624 ± 100	2721 ± 100	2624 ± 108
REE (kcal/min)				
Fed state	1.12 ± 0.04	1.12 ± 0.06	1.17 ± 0.05	1.18 ± 0.05
Fasting state	0.95 ± 0.04	0.94 ± 0.04	0.96 ± 0.05	0.97 ± 0.04
Average REE (kcal/min)[2]	1.07 ± 0.04	1.07 ± 0.05	1.10 ± 0.05	1.12 ± 0.05
DEE[3]				
(kcal/d)	2682 ± 96	2675 ± 112	2691 ± 112	2461 ± 103
(kcal · kg FFM^{-1} · d^{-1})	51.3 ± 1.9	51.4 ± 1.2	51.8 ± 1.9	47.9 ± 1.6
Change in DEE from baseline				
(kcal/d)	—	—	9 ± 60	−214 ± 69[4]
(kcal · kg FFM^{-1} · d^{-1})	—	—	0.5 ± 1.3	−3.4 ± 1.4[5]

[1] All values are $\bar{x}$ ± SEM.

[2] Weighted average of the values during the fed (17 h) and fasting (7 h) periods of the day.

[3] Estimated from the equation DEE = EI − ΔBE, where ΔBE is the change in total body energy.

[4,5] Significantly different from the preformula high-OA diet (analysis of covariance):

[4] $P = 0.02$,

[5] $P = 0.04$.