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The acute effects of olive oil *v.* cream on postprandial thermogenesis and substrate oxidation in postmenopausal women

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The influence of the source of dietary fat on postprandial thermogenesis and substrate oxidation rates, was examined in twelve postmenopausal women aged 57–73 years, with BMI 21·9–38·3 kg/m^2. A single blind, randomised, paired comparison of two high-fat, isoenergetic, mixed test meals was conducted. The major source of fat was either cream (CREAM) or extra virgin olive oil (EVOO). RMR, diet-induced thermogenesis (DIT) and substrate oxidation rates over 5 h were measured by indirect calorimetry. There were no differences in body weight, RMR, fasting carbohydrate or fat oxidation rates between the two occasions. DIT (EVOO 97 (SD 46) *v.* CREAM 76 (SD 69) kJ/5 h and EVOO 5·2 (SD 2·5) *v.* CREAM 4·1 (SD 3·7)% energy) did not differ between the two test meals. The postprandial increase in carbohydrate oxidation rates, relative to their respective fasting values (ΔCOX), was significantly lower following the EVOO meal (EVOO 10·6 (SD 8·3) *v.* CREAM 17·5 (SD 10) g/5 h; paired *t* test, $P=0·023$), while postprandial fat oxidation rates (ΔFOX) were significantly higher (EVOO 0·0 (SD 4·4) *v.* CREAM -3·6 (SD 4·0) g/5 h; $P=0·028$). In the eight obese subjects, however, DIT was significantly higher following the EVOO meal (EVOO 5·1 (SD 2·0) *v.* CREAM 2·5 (SD 2·9) %; $P=0·01$). This was accompanied by a significantly lower ΔCOX (EVOO 10·9 (SD 9·9) *v.* CREAM 17·3 (SD 10·5) g/5 h; $P=0·03$) and significantly higher ΔFOX (EVOO 0·11 (SD 4·4) *v.* CREAM −4·1 (SD 4·5) g/5 h, $P=0·034$). The present study showed that olive oil significantly promoted postprandial fat oxidation and stimulated DIT in abdominally obese postmenopausal women.

Thermogenesis: Fat oxidation: Olive oil: Monounsaturated fatty acid: Obesity: Postmenopausal

The epidemic of obesity, and its co-morbidities of dyslipdaemia, insulin resistance and CVD, is a global problem. All these conditions have nutritional antecedents that are intimately related to the intake of saturated fat. The prevalence of obesity is high in Australia, and is accentuated in the older person (National Health & Medical Research Council, 1997). Potential metabolic contributors to this age-related increase in fat mass include the observations that older men and women have reduced basal metabolism (Piers *et al.* 1998), low leptin adjusted for fat mass (Soares *et al.* 2000) and a low basal rate of fat oxidation (Calles-Escandon *et al.* 1995). Older women also do not readily increase their fat oxidation to match an increase in fat intake (Melanson *et al.* 1997), and unlike older men, do not show an association between leptin and fat oxidation (Soares *et al.* 2000). Moreover, the deficiency of oestrogens at menopause is associated with visceral obesity and a higher incidence of CVD.

Energy balance is determined by macronutrient balance. For most adults in westernised societies, macronutrient balance is a composite of the balances of alcohol, carbohydrate, protein and fat. There is evidence to indicate that, except for fat, these nutrients regulate their respective balances through appropriate changes in their oxidation (Flatt, 1995). Since absorbed fat has only two routes of disposal, storage or oxidation, it is the capacity to oxidise fat that modulates fat balance. Animal studies have demonstrated different rates of weight gain in response to the consumption of different types of dietary fat (Storlien *et al.* 1998), raising the possibility that fat oxidation may vary with the type of fat. There is now considerable evidence that the type of fat influences its metabolic fate in animals

Abbreviations: CREAM, cream-rich meal; DIT, diet-induced thermogenesis; EVOO, extra virgin olive oil-rich meal.

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and human subjects (Jones *et al.* 1985, 1992; Jones & Schoeller, 1988; Clandinin *et al.* 1995; Bell *et al.* 1997; DeLany *et al.* 2000). We have recently tested the influence of the source of fat in isoenergetic breakfast meals in non-obese and mildly obese males (Piers *et al.* 2002). In that study high-fat test meals included extra virgin olive oil (EVOO) as a source of monounsaturated fat or dairy fat (CREAM) as a source of saturated fatty acids. The results indicated a significantly greater fat oxidation following the EVOO meal, with a significantly lower carbohydrate oxidation over the same time period. The present study was carried out to confirm these effects in postmenopausal women. It was based on the hypothesis that a meal rich in olive oil would promote a greater postprandial oxidation of fat, relative to an isoenergetic meal high in cream.

Subjects

Twelve postmenopausal women, aged 57–72 years and BMI 21·9–38·3 kg/m^2 who were residents of Perth, Western Australia, were recruited by advertisement in the local media. Subjects were screened by a telephone questionnaire and selected individuals underwent a clinical examination including an electrocardiogram. Inclusion criteria included: (1) absence of clinical signs or symptoms of chronic disease; (2) weight stability (±2 kg for preceding 12 months); (3) not on medication affecting metabolic rate or body composition; (4) resting diastolic blood pressure $<$90 mmHg; (5) at least 5 years postmenopausal; (6) not on hormone replacement therapy. For details of the physical characteristics of the subjects, see Table 1. All subjects gave written informed consent to participate in the study. The Human Research Ethics Committee of Curtin University of Technology (reference 214/99) approved the protocol. All measurements were made in the clinical rooms of the Bentley Campus of Curtin University, Perth.

Methods

Study design

The study was designed as a single-blind randomised comparison of the paired responses to two high-fat isoenergetic meals. The major source of fat was either cream or extra virgin olive oil. The interval between trials was 1–4 weeks, and subjects were instructed to maintain their habitual intake and activity patterns during this period.

Table 1. Physical characteristics of the twelve postmenopausal women

(Mean values and standard deviations)

	Entire group (*n* 12)		Obese (*n* 8)	
	Mean	SD	Mean	SD
Age (years)	64	4·5	65	4·8
Weight (kg)	79·3	11·6	86	4·3
BMI (kg/m^2)	31·4	4·8	34·4	2·3
Fat mass (kg)	33·2	6·5	37·6	2·9
Fat-free mass (kg)	46·1	6·0	49·0	3·5
Waist circumference (m)	0·940	0·109	0·998	0·074

Test meals

The CREAM test meal (total weight 293 g, volume 275 ml) comprised 65 g natural Swiss muesli (Uncle Toby's, Wahgunyah, Victoria, Australia), 40 g thickened cream (Peters & Brownes Group, Balcatta, Perth, Western Australia, Australia) and 188 g skimmed milk (Peters & Brownes Group). The EVOO test meal (total weight 276 g, volume 265 ml) consisted of 66 g and 195 g of the same brand of muesli and skimmed milk respectively, with 15 g extra virgin olive oil ('Olive Grove', Meadow Lea Foods Ltd, Mascot, New South Wales, Australia). The cream was blended with the skimmed milk before being served with the muesli. The olive oil was drizzled on the muesli, warmed under the grill for 2 min, then cooled to room temperature before being served with skimmed milk. The macronutrient content of the two breakfast meals was determined from Australian Food Composition Tables (English & Lewis, 1991) and manufacturer's product information (Table 2). Subjects were blinded to the fat being consumed. All subjects completed a palatability questionnaire that enquired about amount, taste, oiliness and overall acceptability of each meal. Each answer was scored on a 150 mm visual analogue scale anchored by the most negative to the most positive response for each question.

Anthropometry and body composition measurements

Standing height was measured using a stadiometer fixed to the wall and recorded to the nearest 1 mm. Body weight was measured after an overnight fast on each occasion, immediately after voiding, with subjects wearing light indoor clothing and no shoes, on a digital balance and recorded to the nearest 100 g. Mid-arm and waist circumferences were measured as described by Callaway *et al.* (1988). Body composition was estimated from subcutaneous skinfold thickness measurements at the biceps,

Table 2. Nutrient composition of test meals based on cream (CREAM) and extra virgin olive oil (EVOO)

(Mean values and standard deviations)

	CREAM meal		EVOO meal	
	Mean	SD	Mean	SD
Energy content (kJ)	1860	22	1863	24
Protein				
g	15·5	0·6	15·2	0·3
% total energy	14	0·4	14	0·3
Total fat				
g	20	0·7	20	1·0
% total energy	40	1·7	41	1·6
P:S ratio	2·0	0·02	3·0*	0·02
M:S ratio	3·0	0·02	7·0*	0·04
Carbohydrate				
g	54	2·1	55	1·3
% total energy	46	1·4	47	1·3
Sugar (g)	25	0·9	24	0·5
Starch (g)	30	1·4	31	0·9
Fibre (g)	8	0·4	8	0·2

P, polyunsaturated fatty acid; S, saturated fatty acid; M, monounsaturated fatty acid.

Mean values were significantly different from those of the CREAM meal (paired *t* test): *$P<0·005$.

triceps, subscapular and suprailiac sites as described by Durnin & Womersley (1974). All measurements were made in triplicate and the average was used in the equations of Durnin and Womersley for the sum of four skinfold thicknesses. In one subject, estimates of body composition were based on the sum of biceps and triceps skinfold thicknesses only, as measurements at the other two sites were beyond the limits of the callipers.

Measurement protocol

Subjects were requested to abstain from any strenuous exercise for 36 h before the measurement. Subjects arrived at the laboratory after a minimum of 8 h sleep, and kept physical activity to a minimum. On arrival subjects were asked to empty their bladder after which they were weighed. They lay supine for a mandatory 30 min rest period, while the calorimeter (V_{max}-29 metabolic monitor; Sensor Medics, Yorba Linda, CA, USA) was calibrated. At the end of this rest period, the canopy of the calorimeter was placed over the head of the subject and they were asked to remain awake and as motionless as far as possible in a supine position. Two 15 min RMR measurements were made, with a 10 min interval between measurements. The initial 5 min of each measurement period was discarded from the analysis. Our previous studies indicate that this protocol yields an RMR not significantly different from BMR measured immediately on waking following an overnight stay in the laboratory (Soares *et al.* 1989).

After the RMR measurement, the plastic canopy was removed and subjects made a fasting collection of urine. They were given a breakfast meal (Table 2), which they consumed within 10 min. A palatability questionnaire, based on a 150 mm visual analogue scale, was completed in this period. In between measurements, subjects were allowed to sit up in bed and listen to music or read. Some elected to sit at a table close to the bed, where they did craftwork, needlework or solved crosswords. Thirty-five minutes into each postprandial hour, subjects returned to the supine position and rested for 10 min. The canopy was then placed over the head and measurements made for the last 15 min of the hour. The first 5 min was not included in subsequent data analysis. At the end of the energy expenditure measurements (5 h after the breakfast meal), subjects were asked to void again. The two urine collections were made in separate containers. The volume and duration of collection was noted, and a portion was frozen. Total urinary N excretion was estimated by the Kjeldahl technique. All subjects were offered a light lunch before they returned home.

Resting metabolic rate

RMR was measured by indirect calorimetry using the V_{max}-29 metabolic monitor (Sensor Medics): an open-circuit ventilated canopy measurement system. The measurement was conducted under standardised conditions, as in our previous studies (Soares *et al.* 1989; Piers *et al.* 1992) with subjects lying: (1) at complete physical rest; (2) in a thermally neutral environment; (3) 12–14 h after their last meal and a minimum of 8 h of sleep; (4) awake and emotionally undisturbed; (5) without disease and fever. The within-subject CV in RMR, separated from measurement error, was 1·0 % in the present study.

The V_{max}-29 (Sensor Medics) was calibrated on the morning of each experimental day using a two-point calibration based on two separate mixtures of known gas content. Flow calibrations were achieved using a calibrated 3 litre syringe as directed in the manufacturer's instructions. Flow rate on each measurement day was set at 40 l/min. On each experimental day, the instrument was re-calibrated for flow and the analysers for drift 2 h after ingestion of the test meal. Performance of the instrument was also checked at regular intervals during the study period by monitoring the CO_2 produced:O_2 consumed during 30 min ethanol burns. The mean ratio for twenty ethanol burns was 0·668 (SD 0·016) with CV 2·3 %.

Diet-induced thermogenesis

Diet-induced thermogenesis (DIT) was measured as described previously (Piers *et al.* 1992, 2002) and expressed in absolute values (kJ/5 h) and as % energy in the breakfast meal.

Substrate oxidation rates

Whole-body substrate oxidation rates were calculated at rest (fasting), and for every hour up to 5 h postprandial, using measures of O_2 consumption, CO_2 production and urinary N excretion in the postprandial phase. The equations of Ferrannini (1988) were used to calculate energy expenditure and substrate oxidation rates.

Statistical analysis

All results are presented as mean values and standard deviations, unless otherwise stated. Pearson's correlation coefficients for all anthropometric, body composition and metabolic variables were separately calculated for each meal type. Changes between fasting and fed states were calculated by subtracting the fasting value × duration of measurement from the total postprandial value over 5 h. Since the intervals of measurement in the postprandial period were equal, this summary statistic was analogous to determining the incremental area under the curve (Mathews *et al.* 1990). Paired *t* tests were used to compare the change in metabolic variables of interest. A repeated-measures ANOVA with order of meals as between-subject factor was also used. Statistical significance was accepted at the 5 % level. Data were analysed using the SPSS for Windows (version 11; SPSS Inc., Chicago, IL, USA) statistical software package.

Results

There was no significant difference in body weight, fat mass or fat-free mass on the two occasions. The nutrient composition of the test meals is shown in Table 2. The EVOO meal had significantly higher monounsaturated: saturated fatty acids ratio as well as polyunsaturated:saturated fatty acids ratio (Table 2). There was no difference

in the results of the sensory evaluation of the test meals for the scores for 'amount' (CREAM 94 (SD 18) *v*. EVOO 91 (SD 28) mm), 'taste' (CREAM 104 (SD 34) *v*. EVOO 99 (SD 34) mm), 'oiliness' (CREAM 57 (SD 40) *v*. EVOO 60 (SD 48) mm) and 'overall acceptability' (CREAM 105 (SD 40) *v*. EVOO 100 (SD 42) mm) of each meal.

Effect of breakfast meals

There were no differences in resting metabolic variables (Table 3). DIT, in absolute terms (kJ/5 h) or as % energy in meal, was not different between test meals (Table 3). There was a significant rise in RER following both meals. However, the change in RER was significantly ($P=0{\cdot}018$) lower following the EVOO meal (Table 3, Fig. 1). Postprandial protein oxidation rates were significantly lower following both meals, but the incremental change in protein oxidation was not different between test meals (Table 3). Carbohydrate oxidation was significantly ($P=0{\cdot}023$) lower following the EVOO meal as compared with the CREAM meal (Table 3). Postprandial fat oxidation was significantly suppressed following the CREAM meal (Table 3), but not after the EVOO meal. Hence change in fat oxidation was significantly ($P=0{\cdot}028$) greater following the EVOO meal. An ANOVA for repeated measures, with order of meals as between-subject factor, provided the same statistical outcomes for each variable tested (results not shown).

Table 3. The influence of the source of fat on postprandial thermogenesis and substrate oxidation measured over 5 h in postmenopausal women*

(Mean values and standard deviations for twelve subjects)

	CREAM meal		EVOO meal		Statistical significance of effect: *P* value for paired difference†
	Mean	SD	Mean	SD	
Fasting					
RMR (kJ/h)	210	29	208	30	0·52
RER	0·85	0·046	0·86	0·038	0·33
Protein oxidation (g/h)	3·1	0·90	2·9	0·69	0·70
Fat oxidation (g/h)	1·80	1·03	1·60	0·80	0·36
Carbohydrate oxidation (g/h)	5·4	2·19	6·0	1·96	0·29
Meal-induced changes					
Diet-induced thermogenesis					
kJ/5 h‡	76·0	69	97	46	0·22
% Energy intake§	4·1	3·68	5·2	2·45	0·23
Change in RER‡	0·055	0·039	0·025	0·036	0·018
Change in protein oxidation (g/5 h)‡	−3·2	5·0	−4·0	3·74	0·59
Change in carbohydrate oxidation (g/5 h)‡	17·5	10	10·6	8·3	0·023
Change in fat oxidation (g/5 h)‡	−3·6	4·0	0·0	4·4	0·028

* For details of subjects, diets and procedures, see Tables 1 and 2 and p. 246.
† Similar statistical effects were obtained when analysed by repeated-measures ANOVA with order of meals as between-subject factor.
‡ Calculated as postprandial values over 5 h − (fasting value per h × 5).
§ Calculated as (postprandial values over 5 h − (fasting value per h × 5))/energy intake) × 100.
CREAM, cream; EVOO, extra virgin olive oil.

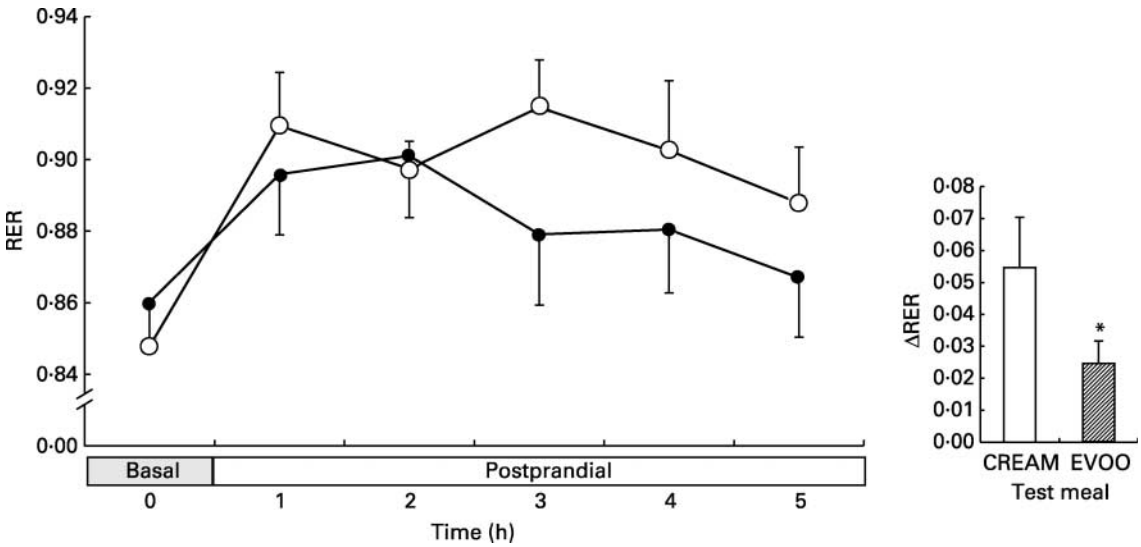

Fig. 1. Basal (fasting) and postprandial RER in twelve postmenopausal women, following meals high in cream (CREAM, ○) or extra-virgin olive oil (EVOO, ●). For details of subjects, diets and procedures, see Tables 1 and 2 and p. 246. Values are means with their standard errors shown by vertical bars. Mean value was significantly different from that of the CREAM meal (paired *t* test): *$P<0{\cdot}018$.

Correlation coefficients for body composition variables and indices of thermogenesis as well as substrate oxidation were computed for each test meal separately. They did not reveal any significant results. The only relationship of note was the inverse trend between BMI and DIT (r −0·56, P=0·06) following the CREAM meal, which was not evident after the EVOO meal (r −0·13, P=0·69). To examine whether EVOO had a different effect in the obese, a subanalysis was conducted. DIT was significantly increased in the obese following EVOO (Table 4, Fig. 2). There was a significantly lower incremental change in carbohydrate oxidation and reciprocally, a greater incremental change in fat oxidation following EVOO (Table 4).

Discussion

Stable isotope studies in human subjects indicate that differences in fatty acid oxidation stem from differences in chain length, degree of unsaturation and the position and stereoisomeric configuration of double bonds (DeLany *et al.* 2000). Medium-chain fatty acids such as laurate (2 : 0) were more readily oxidized than long-chain

Table 4. Changes in diet-induced thermogenesis and substrate oxidation in obese postmenopausal women following mixed test meals with different sources of fat*

(Mean values and standard deviations for eight subjects)

	CREAM meal		EVOO meal		Statistical significance of effect: P value for paired difference†
	Mean	SD	Mean	SD	
Fasting					
RMR (kJ/h)	222	26·4	219	28·2	0·43
RER	0·84	0·044	0·85	0·035	0·50
Protein oxidation (g/h)	3·2	0·98	3·1	0·75	0·90
Fat oxidation (g/h)	2·00	1·08	1·70	0·75	0·44
Carbohydrate oxidation (g/h)	5·5	2·19	6·1	2·08	0·46
Meal-induced changes					
Diet-induced thermogenesis					
kJ/5 h‡	46·0	53	95	37	0·009
% Energy intake§	2·5	2·86	5·1	2·00	0·01
Change in RER‡	0·055	0·042	0·023	0·039	0·016
Change in protein oxidation (g/5 h)‡	−3·4	5·78	−4·6	2·68	0·59
Change in carbohydrate oxidation (g/5 h)‡	17·3	10·48	10·9	9·87	0·030
Change in fat oxidation (g/5 h)‡	−4·1	4·54	0·11	4·45	0·034

CREAM, cream; EVOO, extra virgin olive oil.
* For details of subjects, diets and procedures, see Tables 1 and 2 p. 246.
† Similar statistical effects were obtained when analysed by repeated measures ANOVA with order of meals as between-subject factor.
‡ Calculated as postprandial values over 5 h − (fasting value per h × 5).
§ Calculated as ((postprandial values over 5 h − (fasting value per h × 5))/energy intake) × 100.

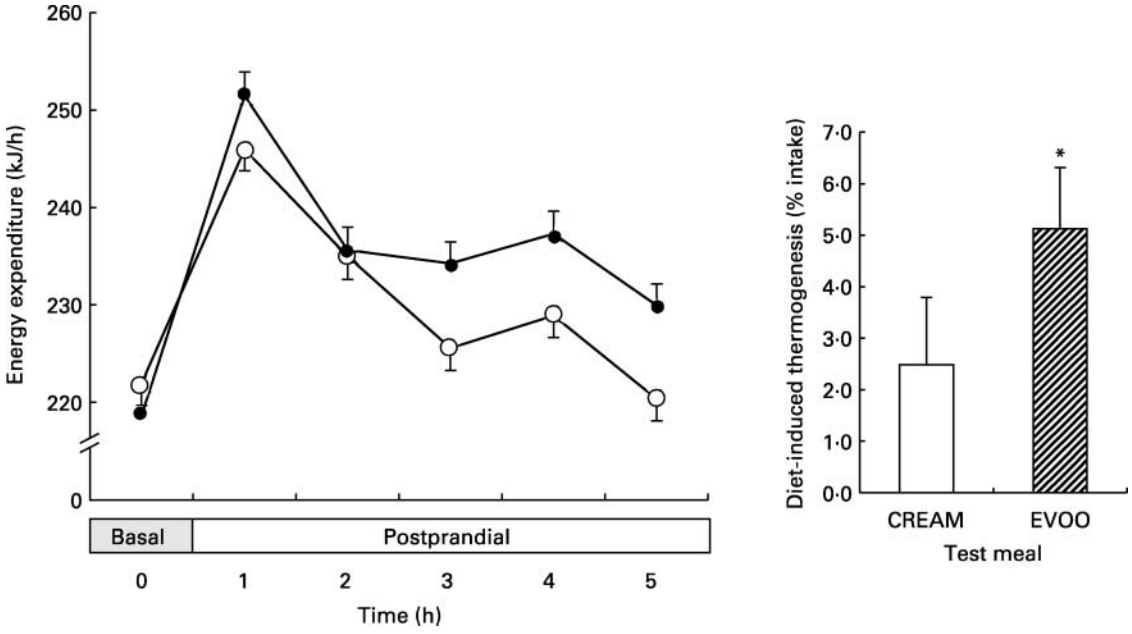

Fig. 2. Basal (fasting) and postprandial energy expenditure in eight obese postmenopausal women, following meals high in cream (CREAM, ○) or extra virgin olive oil (EVOO, ●). For details of subjects, diets and procedures, see Tables 1 and 2 and p. 246. Values are means with their standard errors shown by vertical bars. Mean value was significantly different from that of the CREAM meal (paired t test): *P<0·01.

saturated fatty acids, and unsaturated fatty acids were more readily oxidized than saturated fatty acids (DeLany *et al.* 2000). Jones *et al.* (1985) demonstrated that when chain length was similar, oleic acid was oxidised more readily than both stearic and linoleic (oxidation ratio oleic : linoleic : stearic 14·0 : 4·5 : 1·0). Since the source of fat determines the eventual mix of fatty acids in the diet, we used a whole-food approach to alter the fatty acid milieu. The nutrient composition of the test meals used showed that they were similar in their energetic value, macronutrient composition, sugar, starch and fibre content. EVOO has a high monounsaturated fatty acid content, and relative to CREAM, is a better source of polyunsaturated fatty acids. Hence, the exclusive use of EVOO resulted in a significant increase in dietary monounsaturated:saturated fatty acid ratio and a smaller but significant increase in the polyunsaturated:saturated fatty acid ratio. The effects on postprandial thermogenesis and substrate oxidation in the present study have therefore been ascribed to EVOO, rather than a specific fatty acid.

Olive oil, fat oxidation and thermogenesis

The results showed that the postprandial increase in RER was less following EVOO, suggesting a greater oxidation of fat relative to carbohydrate (Fig. 1). Calculation of substrate oxidation rates showed that the increase in carbohydrate oxidation was attenuated following the EVOO meal. Correspondingly, fat oxidation was significantly suppressed after the CREAM meal but showed no change following EVOO (Table 3). The incremental change in fat oxidation was significantly greater after EVOO (Table 3). The time course of these changes indicates that maximum suppression occurred 3–5 h after meal ingestion (Fig. 1), a period that coincides with the time for fat digestion and absorption. Similar observations in our earlier study (Piers *et al.* 2002), suggest that the prevailing mix of fatty acids has significant effects on substrate oxidation in the late postprandial state.

DIT is the result of energy expended to digest, transport, metabolise and store food. It averages about 10 % total daily energy expenditure and varies with the metabolic fate of the ingested substrate (Segal & Gutin, 1983). The significant effects of EVOO on whole-body substrate oxidation were not accompanied by statistical differences in DIT. Our subjects ranged in BMI from lean to grade 2 obese. There is evidence to suggest that obese individuals, and those predisposed to obesity, may have a defective thermogenic response to meal ingestion when compared with lean individuals (Raben *et al.* 1994; Napoli & Horton, 1996; Matsumoto *et al.* 2001). De Jonge & Bray (1997) concluded that DIT was lower in obesity, an outcome demonstrated in twenty-two of twenty-nine studies examined. In the present study we found a trend for BMI to be inversely related to DIT (r −0·56, P=0·06) following CREAM. This would support the notion that obesity is associated with a lower DIT. However, a similar correlation was not seen after EVOO. This raised the possibility that EVOO either stimulated thermogenesis in obese subjects or suppressed it in lean subjects. Our small sample size did not allow a statistically meaningful comparison to be made between lean and obese. Instead, we re-examined the responses only in the obese. The analysis indicated that dietary thermogenesis was greater by almost 60 % following the EVOO test meal (Fig. 2). This enhanced thermogenesis was accompanied by significant changes in both carbohydrate and fat oxidation. The obese women in the present study also had high waist circumferences (>0·88 m, Table 1). Previously, we had demonstrated that the DIT response to EVOO was significantly greater in men with a high, compared with a low, waist circumference (Piers *et al.* 2002). Overall, the results suggest that EVOO may be particularly beneficial to obese individuals with visceral adiposity.

Potential mechanisms

Differences in DIT, and in substrate utilisation, could potentially arise from differences in gastric events. Accordingly, a faster gastric emptying would result in an earlier rise to peak as well as a quicker descent to baseline. The DIT responses to the two test meals did not have this pattern when either the total results (not shown) or the results from obese subjects were viewed (Fig. 2). Differences in RER and DIT instead appeared at 3–5 h postprandial, a time point beyond gastric events (Figs 1 and 2). Robertson *et al.* (2002) used more direct techniques. They studied the effects of different types of fat on gastric emptying, based on the excretion in breath of [^{13}C]octanoic acid in the meal. They concluded that there were no differences in gastric emptying between saturated and monounsaturated mixed meals.

Changes in fatty acid intake will alter membrane phospholipid composition, and hence influence the metabolic fate of ingested macronutrients. However, changes in membrane composition are more likely with chronic dietary manipulation rather than with acute changes in fatty acid supply, and hence are unlikely to explain the current observations.

The role of the sympathetic nervous system may be important to the results documented here (Fagius & Berne, 1994). There is evidence for a reduced sympathetic nervous system activity in the aetiology of obesity in animals and man (Bray, 1990; Matsumoto *et al.* 2001). While all macronutrients stimulate the sympathetic nervous system (Fagius & Berne, 1994), the type of dietary fat has important influences as well (Young & Walgren, 1994). Takeuchi *et al.* (1995) and Matsuo *et al.* (1995) have demonstrated a lower sympathetic activity and low DIT, but a higher carcass fat content in rats fed beef tallow (saturated fat) as compared with safflower oil (unsaturated fat). Importantly, sympathectomy abolished the differences in body fat accumulation and DIT between the two dietary fat groups.

Femoral and gluteal fat cells have a lower lipolytic response to catecholamines than abdominal adipocytes. The latter have an increased β-adrenoceptor density and sensitivity and reduced α-adrenoceptor affinity and number (Bouchard *et al.* 1993). Abdominally obese individuals could hence be more responsive to stimulation by the sympathetic nervous system. This would explain our current observations of a higher DIT and greater fat utilisation

following EVOO in abdominally obese postmenopausal women. If it is surmised that similar mechanisms operate during chronic feeding of olive-oil-based diets, then the results of Walker *et al.* (1996) are important. These authors have shown a greater mobilisation of upper body fat (abdominal) than lower body (gluteo-femoral) fat, following a high-olive-oil weight-reducing diet in subjects with type 2 diabetes. Additional support is provided by our recent results from an *ad libitum* 4-week dietary intervention study. A predominantly high-olive-oil diet resulted in lower % body fat and lower waist : hip ratios, compared with a saturated fat diet at equivalent intakes of total fat (Piers *et al.* 2003). Overall, the sympathetic nervous system could play an important role in mediating the effects of a high-EVOO meal.

The PPAR provide a molecular mechanism for the modulation of lipid homeostasis by dietary fatty acids, and are a plausible pathway for our observations (Kliewer *et al.* 1997; Fruchart *et al.* 1999; Clarke *et al.* 2002). PPARα activation, in particular, is rapid and occurs within the postprandial phase. PPARα serves to suppress genes encoding proteins of lipid synthesis, while inducing genes encoding proteins of fatty acid oxidation and thermogenesis (Clarke *et al.* 2002). The monounsaturated and polyunsaturated fatty acid (*n*-6) content of the EVOO meal was higher than the CREAM meal (Table 2), and both types of fatty acids are more effective in stimulating PPARα than are saturated fatty acids (Clarke *et al.* 2002).

In conclusion, our present study provides additional evidence that the isoenergetic substitution of cream with olive oil results in an alteration in postprandial substrate oxidation rates. This is manifested as a lesser postprandial increase in carbohydrate oxidation, and a lack of suppression of postprandial fat oxidation following EVOO. The observation that EVOO stimulated DIT in the abdominally obese would auger well for olive-oil-based dietary interventions targeting the management of obesity and the metabolic syndrome.

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