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PAPER

The influence of the type of dietary fat on postprandial fat oxidation rates: monounsaturated (olive oil) *vs* saturated fat (cream)

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OBJECTIVE: To compare postprandial whole-body fat oxidation rates in humans, following high-fat (43% of total energy) mixed breakfast meals, of fixed energy and macronutrient composition, rich in either monounsaturated fat (MUFA) from extra virgin olive oil or saturated fat (SFA) from cream.
DESIGN: Paired comparison of resting metabolic rate (RMR), thermic effect of a meal and substrate oxidation rates following consumption of isocaloric breakfast meals, differing only in the type of fat, administered in random order 1 – 2 weeks apart.
SUBJECTS: Fourteen male volunteers, body mass index (BMI) in the range 20 – 32 kg/m^2, aged 24 – 49 y and resident in Melbourne, Australia, were recruited by advertisement in the local media or by personal contact.
MEASUREMENTS: Body size and composition was determined by anthropometry and dual energy X-ray absorptiometry (DEXA). Indirect calorimetry was used to measure RMR, thermic effect of a meal, post-meal total energy expenditure and substrate oxidation rate. Blood pressure and pulse rates were measured with an automated oscillometric system. Fasting and 2 h postprandial glucose and insulin concentrations and the fasting lipid profile were also determined.
RESULTS: In the 5 h following the MUFA breakfast, there was a significantly greater postprandial fat oxidation rate (3.08 ± 4.58 g/5 h, $P = 0.017$), and lower postprandial carbohydrate oxidation rate ($P = 0.025$), than after the SFA breakfast. Thermic effect of a meal was significantly higher (55 kJ/5 h, $P = 0.034$) after the MUFA breakfast, in subjects with a high waist circumference (HWC $\geq$ 99 cm) than those with a low waist circumference (LWC $<$ 99 cm). This difference was not detected following the SFA breakfast ($P = 0.910$).
CONCLUSION: If postprandial fat oxidation rates are higher after high MUFA, rather than SFA meals, then a simple change to the type of dietary fat consumed might have beneficial effects in curbing weight gain in men consuming a relatively high-fat diet. This may be particularly evident in men with a large waist circumference.
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Keywords: waist circumference; resting metabolic rate; thermic effect of a meal; fat oxidation rate; obesity; human

Introduction

It has been widely argued that high fat diets predispose to obesity.[1] This argument is supported by evidence that fat balance, unlike protein and carbohydrate, is not held under strict metabolic control. Fat oxidation rates adjust very slowly to fat intake. Fat oxidation rates seem largely regulated through changes in the body's carbohydrate economy and not by a change in fat intake.[2] As a consequence, a cumulative positive balance in fat intake leads to an accumulation of adipose tissue mass; this in turn increases plasma free fatty acid concentrations and fat oxidation rates, while decreasing insulin sensitivity. Both fat intake and body glycogen stores therefore determine how large body fat stores must become before fat oxidation matches fat consumption.[2]

Until recently, it was widely believed that all dietary fats are used on an equal basis for oxidation *in vivo*. However, studies of animal obesity have now clearly demonstrated that the consumption of different types of fat evokes

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different rates of weight gain.[3] Despite this animal data, the possibility that the type of dietary fat consumed by humans influences acute fat oxidation rates, and possibly energy intake and body composition has not been extensively studied. It has recently been shown, through the use of stable isotopes, that differences in fatty acid structure including differences in chain length, degree of unsaturation, and the position and stereoisomeric configuration of double bonds may affect the rate of fatty acid oxidation.[4] Medium chain fatty acids such as laurate (C12:0) appear more readily oxidised than long-chain saturated fatty acids (SFA), whereas unsaturated fatty acids are more readily oxidisable than SFA of similar chain length.[4] Studies using indirect calorimetry have also indicated that polyunsaturated fats (PUFA) are better oxidised than saturated fat in both normal weight[5] and obese men.[6] Fewer studies, however, examine the fate of important monounsaturated fatty acids (MUFA), such as oleic acid (C18:1). Jones *et al*,[7] using stable isotope-labelled fatty acids, demonstrated that monounsaturated oleic acid is oxidised more readily than saturated fat (stearic acid C18:0) or PUFA (linoleic acid C18:2) of similar chain length (oxidation ratio stearic:linoleic:oleic = 1.0:4.5:14.0).

These data would indicate that the predominance of particular fatty acids in a given dietary fat would also influence its *in vivo* partitioning between oxidation and storage. To test this hypothesis we studied normal male subjects given an isocaloric breakfast meal, high in extra virgin olive oil as a source of MUFA, or high in dairy fat (cream) as a source of saturated fatty acids (SFA). The macronutrient composition was otherwise identical in both breakfast meals. We chose to evaluate these effects using commonly consumed foods in a meal that varied only in the source of fat. We also chose these fats because they traditionally form part of the daily intake of several population groups. In addition, a reduction of saturated fat and an increase in monounsaturated fat is now advocated for control of type 2 diabetes and cardiovascular disease. If our hypothesis is substantiated it will also suggest a role for MUFA in the management of obesity.

Methods

Subjects

Fourteen male volunteers, aged between 24 and 49 y and resident in Melbourne, Australia, were recruited by advertisement in the local media or by personal approach. Inclusion criteria included: (i) absence of clinical signs or symptoms of chronic disease; (ii) weight stability (± 2 kg for preceding 12 months); (iii) not on medication affecting metabolic rate or body composition; (iv) body mass index (BMI) within the range 20–32 kg/m^2; (v) resting diastolic blood pressure < 90 mmHg. All subjects gave written informed consent to participate in the study. Monash University Ethics Committee approved the experimental protocol, and all measurements were made either in clinical rooms at the Toorak campus of Deakin University or at Monash Medical Centre, Clayton.

Study design

The study provided a paired comparison of resting metabolic rate (RMR), thermic effect of a meal and substrate oxidation rates following breakfast meals of identical energy and macronutrient content but differing fat type, in the same subject, on two separate occasions. The breakfast meals were administered in random order 1–2 weeks apart.

Anthropometry

Standing height was measured using a stadiometer fixed to the wall and recorded to the nearest 0.1 cm. Body weight was measured after an overnight fast, immediately after voiding, with subjects wearing light indoor clothing and no shoes, on a beam balance and recorded to the nearest 100 g. Mid arm, waist and hip circumferences were measured as described by Callaway *et al*.[8]

Body composition

Dual energy X-ray absorptiometry (DEXA) whole-body scans were performed using methodology described previously.[9] Lunar software (Version 1.3, USA) was used to calculate total and regional body composition. The extended research mode of the analysis program was used to determine whole body bone mineral mass (BMM), lean tissue mass (LTM) and fat mass (FM), in grams. The software also permitted determination of the composition of the limbs and trunk using bony landmarks to demarcate each region of interest. This enabled the determination of BMM, LTM and FM in the trunk and each of the four limbs. Fat-free mass was calculated by subtracting FM from body weight.

Resting metabolic rate

Resting metabolic rate (RMR) was measured by indirect calorimetry using a Deltatrac II metabolic monitor (Datex, Finland), an open-circuit ventilated canopy measurement system. The measurement was conducted under standardised conditions, as in our previous studies:[9–11] subjects were lying (a) at complete physical rest; (b) in a thermally neutral environment; (c) 12–14 h after their last meal and a minimum of 8 h of sleep; (d) awake and emotionally undisturbed; and (e) without disease and fever.

The Deltatrac was calibrated using a calibration gas mixture of oxygen (95%) and carbon dioxide (5%; Quickcal, Datex, Finland) each morning prior to the RMR measurements. Air-flow rates through the canopy (46.5 l/min) were checked by means of ethanol burning tests as described by the manufacturer (Datex, Finland), conducted once each month, during the months of data collection. Performance of the Deltatrac monitor was also checked by monitoring the

ratio of carbon dioxide produced to oxygen consumed, during the ethanol burns. The mean (±s.d.) ratio for the last 15 min of the tests was 0.66 (±0.02), which was within the manufacturer's recommended range of 0.64–0.69. RMR measured under similar conditions on two separate occasions, 1–2 weeks apart, were not significantly different ($P > 0.325$). The mean difference (±s.d.) in RMR on the two occasions was 4.1 ± 14.9 kJ/h.

Thermic effect of a meal

Thermic effect of a meal was measured as described previously.[10,11] Thermic effect of a meal was expressed both in absolute terms (kJ/5 h) and as percentage of the energy in the breakfast meal.

Substrate oxidation rates

Whole-body substrate oxidation rate were calculated using measures of oxygen consumption, carbon dioxide production, total urinary nitrogen excretion and the equations of Ben-Porat *et al*[12] and the assumption that nitrogen excretion was steady over the duration of urine collection.[13,14] No correction was made for a possible change in urea pool size.

Breakfast meals

The breakfast high in saturated fat (SFA-breakfast) comprised 92 g natural Swiss muesli (Uncle Toby's, Wahgunyah, Victoria, Australia), 57 g of cream (Bulla, Regal Cream Products, Colac, Victoria, Australia) and 275 g skim milk (Australian Milk Marketing, Abbotsford, Victoria, Australia). The breakfast high in monounsaturated fat (MUFA breakfast) consisted of 96 g muesli baked with 20 g extra virgin olive oil (Bertolli, Preston, Victoria, Australia) and served with 285 g skim milk. Subjects were blinded to the type of fat being consumed. The macronutrient content of the two breakfast meals, determined from Australian Food Composition Tables NUTTAB95[15] are given in Table 1.

Table 1 Macronutrient content of breakfast meals

	SFA[a] *breakfast*	*MUFA*[b] *breakfast*
Protein (percentage energy)	15	15
Total fat (percentage energy)	43	43
Carbohydrate (percentage energy)	42	42
Fibre (g)	11.5	12.0
P:M:S[c] ratio	0.3:0.6:1.0	1.1:3.8:1.0
P,M,S[c] (g)	4.1, 8.8, 16.1	5.6, 18.6, 4.9
Energy content (MJ)	2.5	2.5

[a]Saturated fatty acid.
[b]Monounsaturated fatty acid.
[c]P = polyunsaturated fat; M = monounsaturated fat; S = saturated fat.

Blood samples

A fasting and a 2 h postprandial blood sample (10 ml) were collected from all subjects on both measurement occasions. The samples were centrifuged at 2000 *g* for 15 min and the plasma was separated and stored at −70°C. Plasma glucose, insulin concentrations and lipid profile were measured in the fasting sample while glucose and insulin concentrations were measured in the postprandial samples as described previously.[16] Homeostasis model assessment (HOMA = fast-fasting serum insulin (μU/ml)×fasting plasma glucose (mmol/l)/22.5) was used to calculate insulin sensitivity (insulin resistance >2.5).[17] A mean value was computed from the results obtained on the two measurement occasions for all fasting blood samples.

Blood pressure and pulse rate

Systolic and diastolic blood pressure and pulse rate were measured during measurement of RMR, using a Dinamap portable adult vital signs monitor (model 100, Critikon, USA).

Measurement protocol

Subjects were requested to abstain from any strenuous exercise for 36 h prior to the measurement. On the evening prior to the measurement, they were asked to collect all urine passed after first emptying their bladder at 19:00 h. The next morning (07:00–08:00 h) subjects were transported to the laboratory after a minimum of 8 h sleep, again keeping physical activity to a minimum. On arrival, subjects were weighed and then required to lie down for a mandatory 30 min rest period, while the Deltatrac was calibrated and a fasting blood sample was collected. At the end of this rest period, 12 h after the last meal, the canopy of the Deltatrac was placed over the head of the subject. They were then asked to remain awake and as motionless as possible in a supine position while a 35 min RMR measurement (as above) was completed. Our previous studies indicate that this protocol yields an RMR not significantly different from a BMR measured immediately on waking, following an overnight stay in the laboratory.[18]

After the RMR measurement, the plastic canopy was removed and subjects were given a breakfast meal (SFA breakfast or MUFA breakfast, Table 1) which they were asked to consume within 10 min. Directly afterwards, the canopy was replaced and subjects were once again asked to lie quietly as before, while oxygen consumption and carbon dioxide production were measured for 30 min every hour for 5 h following the breakfast. The canopy was removed during each of the intervening 30 min periods. Subjects were required to remain supine but were allowed to read or listen to music during this break from measurement. We made the assumption that energy expenditure and substrate oxidation rate measured over the 30 min period was representative of the whole hour. We have previously demonstrated the accuracy and

precision of this measurement.[10] Two hours after the breakfast, a second blood sample was collected. At the end of the energy expenditure measurements (5 h after the meal), subjects were asked to void again and the exact time was noted in order to determine the total duration for the urine collection (usually between 19–20 h). The weight of urine voided was then determined. Collected urine was then thoroughly mixed and an aliquot was frozen for the measurement of total urinary nitrogen excretion as described by Tudball *et al*.[19] After collection of the urine sample the remaining anthropometric measurements were performed followed by the DEXA scan. The subjects were then offered a light lunch before being permitted to return home.

Statistics

Data were analysed using the SPSS for Windows (Version 10, SPSS Inc., USA) statistical software package. All data are presented as mean ± s.d., unless otherwise stated. Pearson's correlation coefficients were calculated for all anthropometric, body composition and metabolic variables of interest. Paired *t*-tests were used to compare metabolic variables measured on the two occasions. Analysis of covariance (ANCOVA) was used to compare RMR (adjusting for differences in FFM) and postprandial for oxidation rates (adjusting for differences in basal fat oxidation rates) between the LWC and HWC groups.

Results

Characteristics of the 14 male subjects studied are presented in Table 2. Subjects were healthy men, ranging in age from 24 to 49 y and varying between normal adiposity to mild obesity (BMI 21.1–32.0 kg/m^2). Consistent with BMI, body fat percentage (BF%) ranged from 19.3 to 34.7%. RMR adjusted for differences in fat-free mass (FFM) was not significantly different ($P > 0.761$) between the LWC and HWC groups (mean ± s.e.; LWC, 310.2 ± 4.6 *vs* HWC 308.1 ± 4.6 kJ/h). Despite some obesity the men evidenced no hypertension (systolic and diastolic blood pressure < 140 and 85 mmHg, respectively) and were normoglycaemic (fasting glucose < 6.7 mmol/l).

Table 2 Subject characteristics[a] as a single group and as two sub-groups of low waist circumference (LWC < 99 cm) and high waist circumference (HWC ≥ 99 cm)

	All subjects (n = *14*)	*LWC* (n = *7*)	*HWC* (n = *7*)	*LWC vs HWC* P-*value*
Age (y)	38 ± 9	33 ± 5	43 ± 9	0.026
Weight (kg)	89.0 ± 13.2	81.4 ± 11.7	96.5 ± 10.4	0.025
Body mass index (kg/m^2)	27.8 ± 3.2	25.5 ± 2.9	30.0 ± 1.5	0.003
Waist (cm)	96.8 ± 9.6	88.9 ± 5.9	104.6 ± 4.9	< 0.0005
Waist:hip ratio	0.93 ± 0.06	0.89 ± 0.04	0.98 ± 0.04	0.001
Body fat (%)	29.5 ± 4.8	27.1 ± 5.9	31.9 ± 1.5	0.076
Fat mass (kg)	26.3 ± 6.5	22.1 ± 6.1	30.5 ± 3.4	0.008
Trunk fat mass (kg)	14.6 ± 3.6	12.2 ± 3.3	17.0 ± 2.1	0.006
Fat-free mass (kg)	62.7 ± 8.5	59.3 ± 8.6	66.0 ± 7.5	0.144
Resting metabolic rate (kJ/h)	309.2 ± 37.3	295.9 ± 37.6	322.4 ± 34.4	0.194
Systolic blood pressure (mmHg)	121 ± 11	117 ± 12	125 ± 9	0.145
Diastolic blood pressure (mmHg)	70 ± 9	66 ± 7	74 ± 8	0.092

[a]Values are mean ± s.d.

Table 3 Indirect calorimetry data[a] from 14 healthy men given isocaloric breakfast meals differing only in the type, but not amount, of dietary fat

	SFA[b] *breakfast*	*MUFA*[c] *breakfast*	*Difference*	P-*value*
Resting metabolic rate (kJ/h)	311 ± 40	307 ± 36	4 ± 15	0.325
Thermic effect of a meal (kJ/5 h)	148 ± 50	142 ± 50	6 ± 60	0.731
Thermic effect of a meal (percentage energy)	5.9 ± 2.0	5.7 ± 2.0	0.2 ± 2.4	0.753
Post meal total energy expenditure (kJ/5 h)	1704 ± 204	1678 ± 204	26 ± 50	0.074
Total urinary nitrogen (g/day)	14.4 ± 3.1	13.9 ± 2.6	0.48 ± 2.60	0.498
Substrate oxidation rates				
Protein (g/h)	3.7 ± 0.8	3.6 ± 0.7	0.13 ± 0.68	0.505
Fat — fasting (g/h)	3.6 ± 1.7	3.6 ± 1.4	0.04 ± 1.58	0.934
Fat — postprandial (g/5 h)	12.5 ± 4.9	15.6 ± 5.6	3.08 ± 4.58	0.017
Carbohydrate — fasting (g/h)	6.3 ± 3.2	6.3 ± 3.3	0.00 ± 4.25	1.000
Carbohydrate — postprandial (g/5 h)	55.1 ± 12.3	46.5 ± 11.6	8.63 ± 12.77	0.025

[a]Values are mean ± s.d.
[b]Saturated fatty acid.
[c]Monounsaturated fatty acid.

Data from indirect calorimetry are presented in Table 3, while fasting and postprandial respiratory quotients (RQ) are presented in Figure 1. There were no significant differences in energy expenditure (RMR, thermic effect of a meal or post-meal total energy expenditure measured on the two occasions; $P > 0.05$). Nor were there significant differences in total urinary nitrogen excretion ($P = 0.498$); fasting fat or carbohydrate oxidation rates ($P > 0.05$). There was, however, a significantly greater postprandial fat oxidation rate ($P = 0.017$), and a significantly lower postprandial carbohydrate oxidation rate ($P = 0.025$), in the 5 h following the MUFA breakfast, than after the SFA breakfast.

There were no significant differences ($P > 0.05$) in fasting or 2 h postprandial plasma lipids, glucose or insulin on the two occasions (Table 4). Nor was there a significant difference in the insulin resistance as calculated by HOMA. The mean HOMA-derived insulin resistance, however, was found to be significantly and positively related to BF% ($r = 0.618$, $P = 0.018$), total FM ($r = 0.558$, $P = 0.038$) and trunk FM ($r = 0.608$, $P = 0.021$). Relationships with waist circumference and the waist-hip-ratio (WHR), while also positive, failed to reach significance ($P = 0.061$ and $P = 0.086$, respectively).

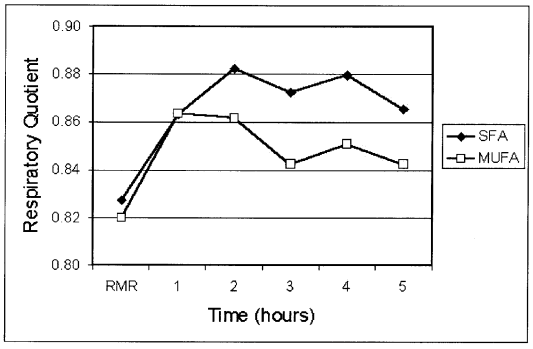

Figure 1 Respiratory quotient obtained during the RMR measurements and in the hours following the saturated fatty acid (SFA, filled shape) and monounsaturated fatty acid (MUFA, open shape) breakfast.

Table 4 Plasma, lipid, glucose and insulin concentrations[a] in 14 healthy men given isocaloric breakfast meals differing in the type, but not amount, of dietary fat

	SFA[b] *breakfast*	*MUFA*[c] *breakfast*
Total cholesterol (mmol/l)	5.1 ± 0.76	5.1 ± 0.92
Triacylglycerol (mmol/l)	2.1 ± 1.7	1.8 ± 1.5
LDL:HDL ratio	3.25 ± 0.75	3.50 ± 0.85
Glucose — fasting (mmol/l)	5.37 ± 0.52	5.27 ± 0.40
Glucose — 2 h postprandial (mmol/l)	5.30 ± 0.61	5.34 ± 0.55
Insulin — fasting (μU/ml)	8.5 ± 3.9	8.2 ± 2.8
Insulin — 2 h postprandial (μU/ml)	22.2 ± 13.7	21.0 ± 12.7
HOMA insulin resistance	2.06 ± 1.07	1.91 ± 0.63

[a]Values are mean ± s.d. and not significantly different ($P > 0.05$) on the two occasions.
[b]Saturated fatty acid.
[c]Monounsaturated fatty acid.

Postprandial fat oxidation rate following both breakfast meals was significantly related to waist circumference (SFA breakfast, $r = 0.556$, $P = 0.039$; MUFA breakfast, $r = 0.539$, $P = 0.047$). However, while postprandial fat oxidation rate after the MUFA breakfast was significantly related to thermic effect of a meal either expressed in terms of a percentage ($r = 0.656$, $P = 0.011$) or in absolute terms ($r = 0.655$, $P = 0.011$), no similar relationship was evident following the SFA breakfast ($r = -0.082$, $P = 0.779$ for TEM expressed as a percentage and $r = -0.087$, $P = 0.769$ for thermic effect of a meal in absolute terms).

The 14 subjects were arbitrarily divided into a LWC group (waist circumference < 99 cm, $n = 7$) and a high HWC group (waist circumference ≥ 99 cm, $n = 7$). LWC subjects were significantly younger ($P = 0.026$), weighed less, ($P = 0.025$), and had a lower BMI ($P = 0.003$), WHR ($P = 0.001$), total FM ($P = 0.008$) and trunk FM ($P = 0.006$) than HWC subjects (Table 2). There were no significant differences in BF% ($P = 0.058$), or in systolic or diastolic blood pressure ($P = 0.145$ and $P = 0.092$, respectively).

After the SFA breakfast, postprandial fat oxidation rate tended to be lower in LWC (10.0 ± 1.9 g/5 h) than HWC subjects (14.9 ± 5.8 g/5 h), although this difference failed to reach significance ($P = 0.052$). After the MUFA breakfast, postprandial fat oxidation rate was found to be significantly ($P = 0.014$) lower in LWC (12.1 ± 3.6 g/5 h) than in HWC subjects (19.0 ± 5.3 g/5 h). However, after adjustment for the significant difference ($P = 0.005$) in basal fat oxidation rates between the two groups (LWC, 2.6 ± 0.9 *vs* HWC, 4.6 ± 1.2 g/h) using an analysis of covariance, the difference in postprandial fat oxidation rate was no longer evident ($P = 0.224$). Thermic effect of a meal either expressed in absolute terms (LWC, 115 ± 26 *vs* HWC, 170 ± 55 kJ/5 h), or as a percentage of the energy content of the breakfast meal (LWC, 4.5 ± 1.1 *vs* HWC, 6.8 ± 2.2%), was significantly lower ($P = 0.034$) in LWC than HWC subjects after the MUFA breakfast, but not after the SFA breakfast in absolute terms (LWC, 150 ± 41 *vs* HWC, 146 ± 61 kJ/5 h, $P = 0.910$) or as a percentage of the energy in the meal (LWC, 6.0 ± 1.7% *vs* 5.8 ± 2.5%, $P = 0.910$). There were no differences ($P > 0.05$), on either measurement occasion, in total urinary nitrogen excretion (SFA breakfast LWC, 14.8 ± 3.1 *vs* HWC, 14.0 ± 3.3 g/day; MUFA breakfast LWC, 12.9 ± 2.3 *vs* 14.9 ± 2.6 g/day), postprandial carbohydrate oxidation rate (SFA breakfast LWC, 56.7 ± 10.9 *vs* HWC, 53.6 ± 14.2 g/5 h; MUFA breakfast LWC, 50.9 ± 11.5 *vs* 42.1 ± 10.8 g/5 h) or mean HOMA insulin resistance (LWC, 1.76 ± 0.72 *vs* HWC, 2.21 ± 0.87), calculated from fasting insulin and glucose concentrations measured on the two occasions in each group of subjects.

Discussion

Fat balance is of critical importance in the maintenance of body fat stores.[2] Thus, factors influencing fat oxidation, fat storage and fat intake may be expected to affect total body

fat stores and body weight. One poorly recognised factor affecting fat oxidation may be the type or source of dietary fat. In the studies reported here, we have demonstrated for the first time, using indirect calorimetry in a group of healthy non-obese to mildly obese men that mean (±s.d.) postprandial fat oxidation rate was significantly higher ($P=0.017$), by approximately 3.08 ± 4.58 g/5 h, following a 2.5 MJ meal with a high MUFA content than an energetically equivalent meal of identical macronutrient composition but high SFA content (Table 3). Conversely, the postprandial carbohydrate oxidation rate was significantly lower following the high MUFA meal than the high SFA meal; although the thermic effect of a meal (expressed in absolute terms or as a percentage of the energy in the meal), post-meal total energy expenditure, and protein oxidation rates did not differ between the two meals. If a similar difference in postprandial fat oxidation rate were to persist in subsequent meals, in an individual whose total energy intake was 10 MJ/day, an extra 12 g of fat might be oxidised in 24 h, thereby disposing of an extra 450 kJ/day.

These results are consistent with data from previous studies indicating that the type of dietary fat may be an independent factor influencing fat oxidation rates. Studies using both indirect calorimetry and individual labelled fatty acids, have shown that fat oxidation increases as the ratio of polyunsaturated:saturated (P:S) fat in the diet increases.[5,6,20] Moreover, the early studies of Jones *et al* have established that in the 9 h following a test meal, ^{13}C labelled stearic acid (C18:0) was oxidised more slowly than either linoleic acid (C18:2) or oleic acid (C18:1; stearic:linoleic:oleic = 1.0:4.5:14.0).[7]

A plausible molecular mechanism to explain the results of our current study may be offered by the peroxisome proliferator-activated receptors (PPARs). These are a subgroup of the nuclear receptor gene family and appear to serve as physiological sensors of lipid levels. They may provide a molecular mechanism whereby dietary fatty acids (FA) modulate lipid homeostasis.[21] All PPARs (α, δ (or β) and γ) are to different extents activated by fatty acid (FA) and FA derivatives. FA activation of PPARα in particular, causes many pleiotropic effects resulting in stimulation of lipid oxidation, and alteration in lipoprotein metabolism.[22] Importantly, both MUFA and PUFA appear to be more effective in stimulating PPARα than SFA.[21]

Our data also revealed a significant relationship ($P<0.05$) between waist circumference and postprandial fat oxidation rate. Accordingly, subjects were arbitrarily divided into two groups. The HWC group (waist circumference ≥ 99 cm), exhibited greater postprandial fat oxidation rate following the MUFA breakfast than subjects of LWC (waist circumference < 99 cm; $P=0.014$). Although a similar trend was observed following the SFA breakfast, this difference fell short of significance ($P=0.052$). It is relevant that both *in vitro*[23,24] and *in vivo* studies[25,26] suggest that abdominal fat is particularly resistant to the insulin-mediated suppression of lipolysis. In our study it might therefore be thought that higher central fat depots in the men of HWC were responsible for a greater postprandial impairment of free FA suppression,[27] thus explaining the greater postprandial fat oxidation rate. However, adjustment for differences in basal fat oxidation rates nullified these differences in postprandial fat oxidation rate. Hence, the differences in postprandial fat oxidation rate appear to be related more to differences in body size, and therefore energy requirements, than to any intrinsic differences in lipid metabolism.

There are several ways of measuring and expressing the thermic effect of a meal.[10,11] Each of these methods has its advantages and disadvantages; however the consensus of opinion favours the use of a standard test meal; to quote Garrow, 'a log of a given size gives off the same amount of heat in a small or a large oven'.[28] Hence, we opted for a standard test meal rather than one related to RMR, body weight, FFM, or ideal body weight.[10,11] The high-MUFA breakfast induced a significantly greater thermogenic effect only in the HWC subjects. This observation may be related to an interesting observation made in our earlier study.[29] In that randomised cross-over study, overweight subjects with type 2 diabetes were placed for 12 weeks on a weight-reducing diet with a normal fat content (35% of total energy), half of which was from olive oil (modified fat (MF) diet), and on a high-carbohydrate low-fat diet (fat content = 23% of total energy). While similar amounts of body weight and body fat were lost on both diets, on the MF diet there was evidence of a greater mobilisation of upper body fat (abdominal) than lower body (gluteo-femoral) fat. This response may be due to the fact that femoral and gluteal fat cells exhibit a lower lipolytic response to catecholamines than abdominal adipocytes that have an increased β-adrenoceptor density and sensitivity and reduced α-adrenoceptor affinity and number.[30] The results of our current and previous studies, taken together, suggest a possible role for the sympathetic nervous system (SNS) in mediating the effects of a high-MUFA diet. This idea is supported by animal studies.[31,32]

Ljung *et al*[33] studied 50 non-diabetic, middle-aged men to investigate possible differences between generally and abdominally obese men, in activity and regulation of the hypothalamic–pituitary–adrenal (HPA) axis and the SNS. Their results suggested a mild dysregulation of the HPA axis, occurring with elevated WHR, independent of the BMI. The results also indicated a central activation of the SNS, such as in the early phases of hypertension, correlating with insulin resistance. In the current study both systolic and diastolic blood pressure and HOMA-insulin resistance tended to be higher in the HWC as compared to the LWC subjects, but these differences did not reach statistical significance ($P>0.05$).

In conclusion, our study demonstrates for the first time, using indirect calorimetry, significantly greater postprandial fat oxidation rates ($P<0.05$) in healthy normal weight to mildly obese men fed a high-fat (43% of energy) MUFA breakfast meal as compared to a SFA breakfast meal. In

addition, the MUFA breakfast was associated with a significantly ($P < 0.05$) greater thermic effect of a meal in the HWC subjects by about 55 kJ/5 h, increasing potentially to 220 kJ/day for an individual with a total energy intake of 10 MJ/day. The impact of these differences, in substrate oxidation rates and thermogenesis, on body weight and composition in the long term, potentially amounting to over 650 kJ/day on a high MUFA diet, remain to be determined. If a greater postprandial fat oxidation rate and thermogenesis were sustained over time, then simply changing the source of dietary fat might encourage some body fat loss or a least help to curb fat gain, even on a relatively high-fat diet.

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