The effect of palm oil, lard, and puff-pastry margarine on postprandial lipid and hormone responses in normal-weight and obese young women

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Only a few studies have been published on the postprandial effects of different fatty acids in obese subjects. Therefore, the present study investigated the effects of three test meals containing palm oil (PO), lard (LD), or puff-pastry margarine (PPM), all normal dietary ingredients, on postprandial lipid and hormone responses in normal-weight and obese young women. The study was performed as a randomized, crossover design. The fats differed in the content of palmitic acid, stearic acid, and *trans* monounsaturated fatty acids allowing a dietary comparison of different 'solid' fatty acids. The obese women had significantly higher fasting concentrations and postprandial responses of plasma total triacylglycerol (TAG), chylomicron-TAG, and insulin compared with the normal-weight women but there was no significant difference in the postprandial responses between the three test meals. The obese women had fasting concentrations of leptin four times greater than the normal-weight women. There were no postprandial changes in the concentrations of leptin. The fasting concentrations of HDL-cholesterol were significantly lower in the obese women than in the normal-weight women, whereas there was no significant difference between the two groups in the concentrations of total cholesterol or LDL-cholesterol. These results provide evidence that obese women have exaggerated lipid and hormone responses compared with normal-weight women but the different contents of saturated and *trans* monounsaturated fatty acids provided by PO, LD, and PPM have no effect in either group.

Obesity: Postprandial response: *Trans* fatty acids: Saturated fatty acids

The high prevalence of obesity in the Western world and its well-documented association with the cardiovascular risk factors diabetes mellitus, dyslipidaemia, and hypertension represents a major problem for the general health status of industrialized countries. Diet therapy, with an emphasis on weight control is considered the major intervention to control the metabolic imbalance and attempt to reduce the risk of accelerated atherosclerosis.

The dietary treatment of obese persons has focused on diets relatively high in carbohydrate and low in total fat. However, recent studies have reported that high-carbohydrate diets may cause aggravation of dyslipidaemia in patients with non-insulin-dependent diabetes mellitus (Garg *et al*. 1988, 1992; Coulston *et al*. 1989). Instead of reduction in total fat, several official nutritional recommendations include reduction of the intake of saturated fatty acids (SFA) which are known to increase the risk of CHD. However, not all SFA are equally cholesterolaemic. The serum cholesterol raising effect of SFA in the diet appears to be limited to lauric acid (12 : 0), myristic acid (14 : 0), and palmitic acid (16 : 0) while stearic acid (18 : 0) seems to be neutral (Hegsted *et al*. 1965; Grande *et al*. 1970; Bonanome & Grundy, 1988; Denke & Grundy, 1992; Storm *et al*. 1997).

The responses of plasma lipoproteins during absorption of dietary fats have gathered increasing interest due to the fact that man, by eating regular meals, spends most of the time in the postprandial phase. Plasma total triacylglycerols (TAG) increase shortly after the first meal of the day and return to baseline concentrations several hours after the last meal. Intestine-derived chylomicrons (CM) as well as liver-derived VLDL, both of which contribute to the postprandial responses (Cohn *et al*. 1988*a*; Potts *et al*. 1991), compete for hydrolysis by lipoprotein lipase (*EC* 3.1.1.34) as well as the hepatic receptor-mediated removal of remnants from plasma (Brunzell *et al*. 1973; Schneeman *et al*. 1993). Previous studies have shown that the intensity of postprandial lipaemia, especially the presence of remnant particles, plays an

Abbreviations: AUC, area under the curve; CH, cholesterol; CM, chylomicron; LD, lard; MUFA, monounsaturated fatty acid; PO, palm oil; PPM, puff-pastry margarine; PUFA, polyunsaturated fatty acid; SFA, saturated fatty acid; TAG, triacylglycerol.

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important role in the pathogenesis and progression of atherosclerosis (Groot *et al*. 1991; Patsch *et al*. 1992; Karpe *et al*. 1994). Delayed clearance of the TAG circulating in the blood is therefore associated with increased atherogenic risk.

The influence of *trans* fatty acids in the diet has been discussed during recent years. Experiments with human subjects (Mensink *et al*. 1992; Nestel *et al*. 1992; Lichtenstein *et al*. 1993; Wood *et al*. 1993; Judd *et al*. 1994) have pointed to an unfavourable effect of *trans* fatty acids on blood lipids. An epidemiological study performed by Willett *et al*. (1993) showed a relation between intake of *trans* fatty acids from partially hydrogenated vegetable oils and risk of CHD.

Several studies suggest that low-carbohydrate, high-fat diets where monounsaturated fatty acids (MUFA) constitute the majority of fat have beneficial effects for patients with non-insulin-dependent diabetes mellitus compared with high-carbohydrate diets. The beneficial effects include improved glycaemic control (Garg *et al*. 1988; Rasmussen *et al*. 1993) and a subdued 24 h ambulatory blood pressure (Rasmussen *et al*. 1993). Christiansen *et al*. (1997) studied the effects of diets enriched in SFA or *trans* MUFA on the postprandial insulinaemia and fasting serum levels of lipids and lipoproteins in obese patients with non-insulin-dependent diabetes mellitus. They found that in the presence of unchanged glycaemia, both dietary SFA and *trans* fatty acids induced an increase in postprandial insulinaemia compared with a diet with *cis* MUFA. However, it was impossible to differentiate precisely between a long-term effect of the different diets on postprandial insulinaemia and an acute effect of the diets following the test meal or even a combination of these effects (Christiansen *et al*. 1997). In this context it is interesting that recent studies with mouse islets have revealed a higher insulinotropic potency of the *trans* fatty acids compared with the *cis* forms (Alstrup *et al*. 1999). Information about the acute metabolic effect on lipid and hormone responses in obese subjects after consumption of different types of fat is sparse. Rasmussen *et al.* (1996) studied the acute metabolic effect on blood glucose and insulin responses in subjects with non-insulin-dependent diabetes mellitus after ingestion of SFA and *cis* MUFA and they found that SFA increased the release of insulin compared with MUFA. Ingestion of *trans* MUFA was not studied.

In the present investigation we compared the acute postprandial effects of three normal edible fats used for spreads and cooking, differing in contents of 16 : 0, 18 : 0, and *trans* MUFA but containing comparable amounts of *cis* MUFA and polyunsaturated fatty acids (PUFA), on plasma total TAG, CM-TAG, insulin, and leptin responses in normal-weight and obese young women. This is important as *trans* MUFA, present in food products that contain partially hydrogenated oils and ruminant fats, are often substituted for SFA due to their similar physical properties. The comparison between normal-weight and obese women is interesting since the higher TAG levels normally observed in obese subjects might be a result of delayed clearing. Previously, some studies have found that insulin has a stimulatory effect on serum leptin after 6 h at high physiological concentrations (Malmström *et al*. 1996), that leptin increases between 5 and 10 h after short-term overfeeding (Kolaczynski *et al*. 1996*b*; Joannic *et al*. 1998; Saad *et al*. 1998), and that changing meal time by 6·5 h will shift rhythm by 5–7 h (Schoeller *et al*. 1997). Taking into account that the increase in leptin levels is related to the insulin fluctuations in response to meals (Laughlin & Yen, 1997) we wanted to see whether any increment in leptin occurred up to 8 h after the test meals and whether the responses differed between normal-weight and obese subjects. The fatty acid compositions of the postprandial CM lipids were studied to determine if discrimination in the absorption or a specific clearing was taking place.

Experimental methods

Subjects

Seven obese women and eight normal-weight women participated in the study. The BMI was used to group the women. The normal-weight group had BMI 19·2–23·7 kg/m^2 and the obese group had BMI 28·8–47·5 kg/m^2. The normal-weight and obese women were well matched for age (mean 27 (SD 2) years *v.* 29 (SD 3) years respectively).

Two women of normal weight used oral contraceptives. Three women from the obese group were smokers. None of the women was taking any medications and none had any history of recent intercurrent diseases. There was no evidence of lipid or thyroid dysfunction or kidney and liver disease. The haemostatic variables were in the normal range.

The women in the obese group were outpatients of the medical clinic at Bispebjerg Hospital in Copenhagen, Denmark and the women in the control group were students and personnel at The Technical University of Denmark.

Participants were fully informed of the experimental nature of the study and the protocol was approved by the Municipal Ethical Committee of Copenhagen and Frederiksberg, Denmark to be in accordance with the Helsinki-II declaration.

Study design

The study was performed as a randomized, crossover design where all women consumed three test meals with palm oil (PO), lard (LD), or puff-pastry margarine (PPM). The test meals were given with a washout period of at least 1 week. After an overnight fast the participants arrived in the morning at the medical clinic where a cannula was inserted into a vein from which blood samples were collected during the day. Following the collection of a fasting blood sample the test meal was served and ingested within 15 min. Blood samples were taken at hourly intervals including at 8 h.

The concentrations of plasma total TAG and CM-TAG were determined before the meal and monitored over 8 h. Insulin concentrations were determined in the fasting state and monitored until starting values were obtained again (4 h after consumption of the three test meals). The concentrations of leptin were determined before the meal and after 4 h and 8 h. Fasting blood samples were also analysed to determine concentrations of total cholesterol (CH), LDL-CH, and HDL-CH.

Table 1. Major fatty acids (g/100 g total fatty acids) in palm oil (PO), lard (LD), and puff-pastry margarine (PPM)

Fatty acid	PO	LD	PPM
14 : 0	1·1	1·6	0·9
16 : 0	43·5	25·5	34·5
16 : 1*n*-7	0·2	2·2	0·1
18 : 0	4·6	16·8	7·4
18 : 1 *trans**	0·2	0·3	7·0
18 : 1 *cis*†	38·9	39·2	31·5
18 : 2*n*-6	10·3	10·0	14·5
18 : 3*n*-3	0·7	1·2	2·1
ΣSFA	49·2	43·9	42·8
ΣMUFA	39·3	41·7	38·6
ΣPUFA	11·0	11·2	16·6

SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.
* The sum of *trans* isomers.
† The sum of *cis* isomers.

Immediately after the 4 h blood sample was taken an almost fat-free lunch was served as described later. Water was allowed *ad libitum* during the 8 h study period.

Test meals

Three normal edible fats with different fatty acid compositions were tested in the study. The fats were selected to allow a comparison of the effect of the two SFA, palmitic and stearic acid, and of *trans* MUFA. The fatty acid compositions of the three fats are given in Table 1. PO (Aarhus Oliefabrik A/S, Aarhus, Denmark) is used as an ingredient in industrially produced edible fats such as margarines. LD (Raffinol, Copenhagen, Denmark) is used for deep-fat frying, and PPM (Danisco, Grindsted, Denmark) is used for baking of Danish pastries. PO differed from LD and PPM in its high content of palmitic acid and LD differed from the other two fats in its content of stearic acid. However, the total content of 'solid' fatty acids (16 : 0, 18 : 0, and *trans* MUFA) was of the same magnitude. The total amount of the non-atherogenic fatty acids (*cis* MUFA and PUFA) was approximately the same.

The test meal consisted of mashed potato made from potato flakes (Pfanni, Pfanni-Werke, Munich, Germany), skimmed milk (MD Foods, Aarhus, Denmark (0·1 g fat/100 g)), salt, water, and one of the three fats. The meal was served with three pieces of raw cucumber (20 g) and a glass of artificially-sweetened blackcurrant juice (Cadiso Food A/S, Frederikssund, Denmark). It was necessary to use 1 g potato flakes per 1 g fat to camouflage the taste and mouth feel of the fat. The water content of PPM was taken into account in the preparations.

The participants were given 29 g fat/m^2 body surface area which gave an energy content of 1·7 MJ/m^2 (406 kcal/m^2) of which 30 % came from carbohydrate, 5 % from protein, and 65 % from fat. The content of fat accounted for less than 0·5 % energy in the other ingredients. The average fat intake was 51 (SD 4) g per meal for the normal-weight women and 64 (SD 6) g per meal for the obese women (range 46–58 g and 56–73 g respectively).

The nearly fat-free lunch consisted of various fruits and a piece of light crisp bread with salad and tomato. The energy content of this meal was 435 kJ of which 83 % came from carbohydrate, 10 % from protein, and 7 % from fat (<1 g fat). All subjects received the same amount of food for lunch.

Isolation of chylomicrons

Blood samples were taken into 10 ml vacutainer tubes with EDTA to give a final concentration of 1 ml EDTA/l. Plasma was separated by centrifugation (1500 ***g***) for 15 min at room temperature. The TAG-rich CM were isolated by layering a NaCl solution of density 1·004 g/ml on top of plasma followed by centrifugation (27 000 ***g***, 1 h, 15°) in a Sorvall centrifuge RC5C (Du Pont, Stevenage, Herts., UK) SS34 rotor. The CM were obtained by removing the top layer from the tube.

Table 2. Fasting and postprandial responses of plasma triacylglycerol (TAG), chylomicron triacylglycerol (CM-TAG), insulin, and leptin in normal-weight and obese women consuming test meals with palm oil (PO), lard (LD), or puff-pastry margarine (PPM)*

(Mean values and pooled standard deviations for eight normal-weight and seven obese women)

	Normal-weight				Obese				
	PO	LD	PPM	Pooled SD	PO	LD	PPM	Pooled SD	Group effect†
Fasting TAG (mmol/l)	0·85	0·81	0·95	0·32	1·37	1·23	1·66	0·58	$P<0·001$
Fasting CM-TAG (mmol/l)	0·03	0·03	0·04	0·01	0·08	0·08	0·12	0·06	$P<0·001$
TAG incremental AUC (mmol · h/l)	2·43	2·33	1·99	1·33	3·38	3·72	4·23	1·93	$P<0·01$
CM-TAG incremental AUC (mmol · h/l)	1·80	1·32	1·52	0·71	2·11	2·32	2·65	1·09	$P<0·01$
TAG_{MAX} (mmol/l)	0·51	0·63	0·58	0·35	0·77	0·99	1·05	0·58	$P<0·02$
CM-TAG_{MAX} (mmol/l)	0·34	0·42	0·34	0·21	0·54	0·67	0·73	0·36	$P<0·01$
TAG T_{MAX} (h)	1·75	1·75	1·88	0·66	2·14	1·67	1·57	0·95	NS
CM-TAG T_{MAX} (h)	1·88	2·00	2·00	0·58	1·86	1·67	1·57	0·92	NS
Fasting insulin (pmol/l)	41	27	19	26	112	105	128	66	$P<0·001$
Insulin incremental AUC (pmol · h/l)	168	209	213	115	749	500	512	501	$P<0·001$
Fasting leptin (ng/ml)	13	10	11	7	41	42	45	15	$P<0·001$

TAG incremental AUC, incremental area under the postprandial TAG response curve; CM-TAG incremental AUC, incremental area under the postprandial chylomicron TAG response curve; TAG_{MAX}, maximum postprandial TAG concentration; CM-TAG_{MAX}, maximum postprandial CM-TAG concentration; TAG T_{MAX}, time to the first maximum postprandial TAG concentration; CM-TAG T_{MAX}, time to the first maximum postprandial CM-TAG concentration; insulin incremental AUC, incremental area under the postprandial insulin response curve.
* For details of test meals and procedures see pp. 470–471.
† Group effect, differences between normal-weight and obese women tested in the split plot design as described on p. 473.

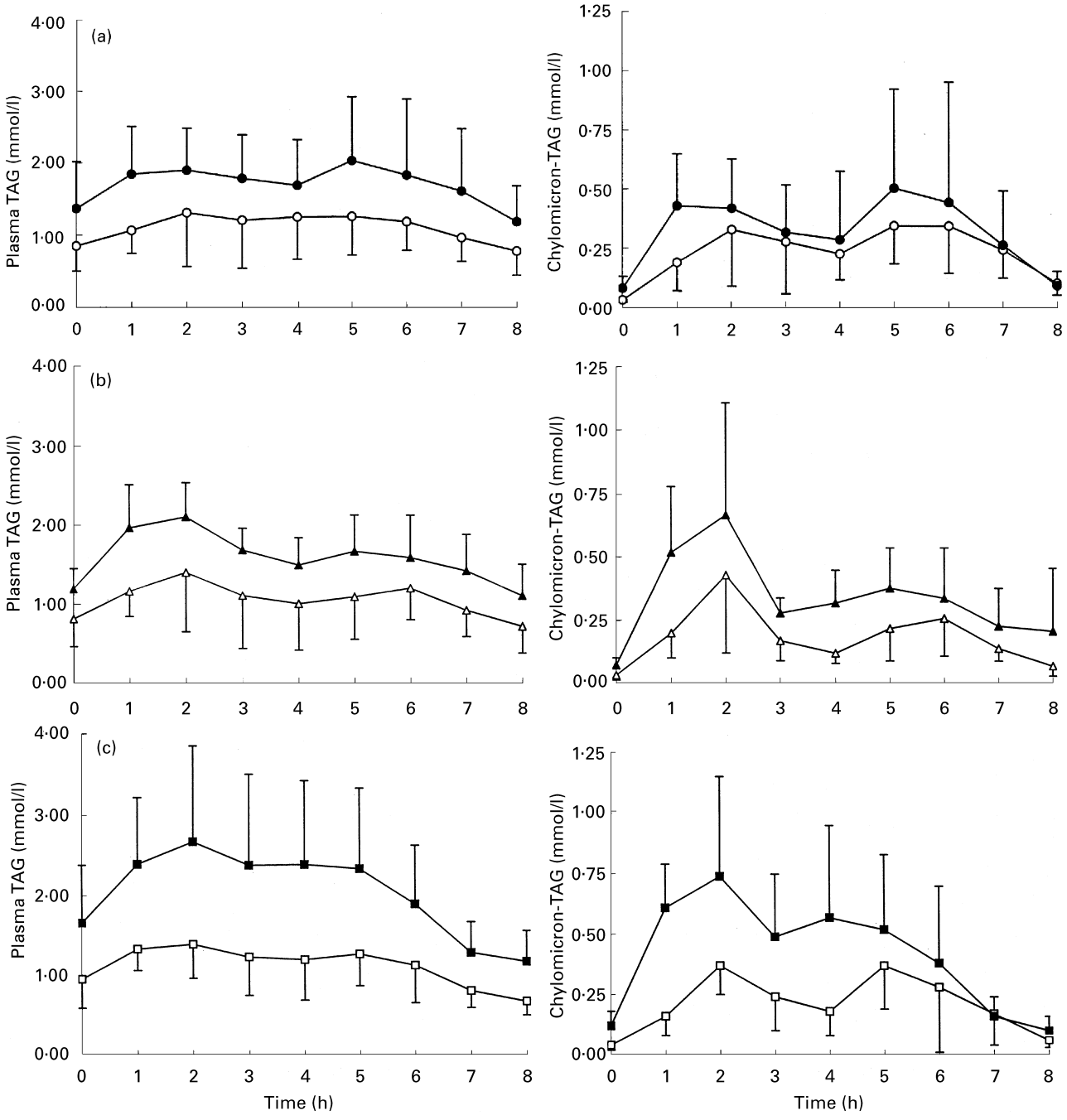

Fig. 1. Plasma triacylglycerol (TAG) and chylomicron-TAG responses (0–8 h) to test meals containing (a) palm oil (○, ●), (b) lard (△, ▲), and (c) puff-pastry margarine (□, ■) in normal-weight (○, △, □) and obese (●, ▲, ■) women. Values are means with standard deviations represented by vertical bars. For details of test meals and procedures see pp. 470–471.

Analytical methods

Lipids were extracted from the CM fractions with chloroform–methanol (1 : 1, v/v), dried with Na_2SO_4, filtered and subsequently saponified with 0·5 M-NaOH in methanol and methylated with BF_3 in methanol (140 g/l) in the presence of hydroquinone (0·2 g/l methanol). The fatty acid methyl esters were extracted with heptane and analysed by GLC on a Hewlett Packard 5880A instrument (Palo Alto, CA, USA) equipped with an autosampler and a flame-ionization detector. An SP2380 capillary column (30 m, i.d. 0·32 mm, 0·2 μm film; Supelco, Bellefonte, PA, USA) was used. The inlet pressure of the carrier gas (He) was 76 kPa. The temperature was maintained at 250° for the injector (split mode) and 260° for the detector. The initial oven temperature was 140°. The temperature was raised to 160° at 2°/min, then held constant for 2 min and finally raised 3°/min to 200°, where it was unchanged for 5 min.

Peaks were identified using fatty acid methyl ester standards (Nu Chek Prep, Inc., Elysian, MN, USA) and

quantified using heptadecanoic acid (17 : 0) as internal standard. The major fatty acids in the isolated CM are presented as a percentage of the total fatty acid mass measured. The inter-assay CV were <1 % for major components. Previous analyses have shown that TAG is the predominant class of fatty acid-containing lipid in CM (> 95 %) (results not shown). As an approximation we have assumed a TAG content of 100 % in CM.

Plasma total TAG and total CH analyses were carried out on a Boehringer Hitachi 717 EC analyser (Roche Diagnostic A/S, Hvidovre, Denmark) at 37°. Plasma total TAG content was measured by a standard coupled enzymic method using lipase (*EC* 3.1.1.3)–glycerol kinase (*EC* 2.7.1.30). Total CH was measured using a standard coupled enzymic procedure using cholesterol esterase (*EC* 3.1.1.13)–cholesterol oxidase (*EC* 1.1.3.17), all supplied as test kits (Boehringer Mannheim, c/o Ercopharm, Kvistgaard, Denmark). HDL-CH was determined using phosphotungstic acid–$MgCl_2$ precipitation followed by analysis of CH as described earlier. LDL-CH was calculated from the measurements of total CH, HDL-CH, and TAG, as described by Friedewald *et al.* (1972). Plasma insulin was determined by an ELISA (Andersen *et al*. 1993). Plasma leptin was measured by a radioimmunoassay (Maffei *et al*. 1995). The inter-assay CV were 1·5 %, 1·5 %, 2·6 %, 7·5 % and 4·5 % for plasma total TAG, total CH, HDL-CH, plasma insulin, and plasma leptin respectively.

Statistical methods

The postprandial responses of plasma total TAG, CM-TAG, and insulin were analysed as areas under the curves (AUC) as recommended by Matthews *et al*. (1990). The responses were calculated separately for each subject as the difference between the absolute area and the rectangular area determined by the fasting concentrations (incremental AUC), thus representing changes occurring after the meal.

To compare the three test meals (fasting and incremental AUC values) a split-plot structured analysis (variables: group, person, meal, period, and meal × group interaction) was carried out using the GLM procedure of the SAS System (SAS Institute Inc., Cary, NC, USA). The level of significance was set at $P < 0{\cdot}05$.

Repeated measures ANOVA was performed within groups to test differences in plasma total TAG, CM-TAG, insulin, and leptin levels over time.

Results

Plasma total triacylglycerol and chylomicron-triacylglycerol

Table 2 presents the fasting concentrations and postprandial responses of TAG in plasma and CM in normal-weight and obese women. The fasting concentrations of plasma total TAG were significantly higher in the obese women than in the normal-weight women ($P < 0{\cdot}001$). The small fractions of CM present in the fasting state were also isolated and the corresponding concentrations of CM-TAG were significantly higher in the obese subjects than in the normal-weight subjects ($P < 0{\cdot}001$). The concentrations of plasma total TAG increased significantly during the postprandial period (Fig. 1). Some individuals showed a monophasic or a triphasic response curve but the majority showed a biphasic response curve with an initial peak 1–2 h and a second peak 4–7 h after the meal. The concentrations returned to baseline values at 8 h. There was a parallel and significant rise in CM-TAG corresponding to the rise in plasma total TAG (Fig. 1).

The incremental areas under the postprandial plasma total TAG response curves (TAG incremental AUC) and the postprandial CM-TAG response curves (CM-TAG incremental AUC) are presented in Table 2. They were both significantly higher in the obese women compared with the normal-weight women ($P < 0{\cdot}01$, $P < 0{\cdot}01$ respectively). For TAG incremental AUC the difference was more pronounced

Table 3. Major fatty acids in palm oil (PO) and chylomicrons isolated every second hour in the postprandial period (g/100 g total fatty acids)§ (Mean values and standard deviations for eight normal-weight and seven obese women)

			Normal-weight					Obese				
Time (h) … Fatty acid		PO	0	2	4	6	8	0	2	4	6	8
16 : 0	Mean	43·5	25·2	36·6	35·7	36·0	33·0	22·5	34·5	32·6	34·5	30·5
	SD		2·1	2·6	2·0	1·2	4·7	2·8	1·7	3·0	2·1	1·9
16 : 1*n*-7	Mean	0·2	2·0	0·6	0·6	0·5	0·8	2·3	1·1	1·2	0·9	1·4
	SD		0·6	0·2	0·4	0·2	0·3	1·0	0·7	0·6	0·3	0·5
18 : 0	Mean	4·6	11·3	6·3	6·5	6·1	7·2	7·9*	5·8	6·4	5·5	6·4
	SD		2·7	0·9	0·8	0·5	1·3	1·3	0·2	0·8	0·3	0·5
18 : 1 *trans*†	Mean	0·2	0·8	0·3	0·3	0·3	0·5	1·6	0·5	0·5	0·4	0·5
	SD		0·2	0·1	0·1	0·1	0·2	2·1	0·3	0·4	0·2	0·1
18 : 1 *cis*‡	Mean	38·9	26·7	36·8	37·1	37·4	32·4	32·5**	38·0	35·9	38·0	34·9
	SD		1·9	1·8	1·2	1·4	4·9	3·5	1·6	1·8	1·0	2·0
18 : 2*n*-6	Mean	10·3	16·7	13·2	11·6	13·5	15·8	17·3	13·6	14·9	13·7	15·7
	SD		2·8	1·5	0·9	0·8	5·4	3·1	1·1	2·0	1·0	1·3
18 : 3*n*-3	Mean	0·7	0·5	0·5	0·4	0·3	0·5	1·2*	0·5	0·4	0·4	0·4
	SD		0·6	0·3	0·2	0·1	0·3	0·6	0·1	0·1	0·1	0·2

Mean values were significantly different from those of the control group: * $P < 0{\cdot}05$, ** $P < 0{\cdot}01$.
† The sum of *trans* isomers.
‡ The sum of *cis* isomers.
§ For details of test meals and procedures see pp. 470–471.

Table 4. Major fatty acids in lard (LD) and chylomicrons isolated every second hour in the postprandial period (g/100 g total fatty acids)§
(Mean values and standard deviations for eight normal-weight and seven obese women)

			Normal-weight					Obese				
Time (h) …			0	2	4	6	8	0	2	4	6	8
Fatty acid		LD										
16 : 0	Mean	25·5	22·3	25·1	24·1	24·5	22·9	24·0	25·5	23·9	24·6	24·1
	SD		2·4	0·9	1·2	1·4	2·5	2·5	1·1	3·2	1·1	0·7
16 : 1*n*-7	Mean	2·2	2·1	2·1	2·0	2·0	1·6	2·5	2·1	1·9	2·1	2·0
	SD		0·9	0·2	0·5	0·1	0·3	0·7	0·9	0·4	0·2	0·3
18 : 0	Mean	16·8	11·0	14·9	14·5	14·0	13·6	7·7	12·6	12·7	12·9	12·3
	SD		5·3	0·8	1·2	0·5	2·0	1·5	1·7	2·8	0·8	1·0
18 : 1 *trans*†	Mean	0·3	1·0	0·5	0·6	0·5	0·6	0·6**	0·4	0·5	0·5	0·5
	SD		0·2	0·1	0·1	0·1	0·1	0·2	0·1	0·1	0·1	0·1
18 : 1 *cis*‡	Mean	39·2	26·7	36·3	34·5	37·1	31·6	33·2*	37·7	38·4	36·7	35·2
	SD		4·9	1·9	2·3	1·1	3·7	2·8	0·8	1·1	1·9	1·9
18 : 2*n*-6	Mean	10·0	18·7	12·0	12·9	12·7	13·1	17·1	12·5	13·3	13·0	14·0
	SD		3·4	1·3	1·4	1·2	1·0	3·5	0·9	1·5	1·2	2·0
18 : 3*n*-3	Mean	1·2	0·5	0·9	0·9	0·9	0·6	0·8	0·9	0·8	0·8	0·8
	SD		0·7	0·1	0·3	0·2	0·4	0·3	0·1	0·2	0·1	0·1

Mean values were significantly different from those of the control group: * $P < 0·05$, ** $P < 0·01$.
† The sum of *trans* isomers.
‡ The sum of *cis* isomers.
§ For details of test meals and procedures see pp. 470–471.

after the test meal with PPM whereas for CM-TAG incremental AUC the difference was more distinct for LD. There was no significant difference in the incremental AUC following the three test meals for either of the two groups.

The first TAG peak in plasma and in CM (TAG_{MAX}, CM-TAG_{MAX} respectively) was significantly higher in the obese women compared with the normal-weight women ($P < 0·02$, $P < 0·01$ respectively) (Table 2). Neither TAG_{MAX} nor CM-TAG_{MAX} were significantly different when comparing test meals. The time to the first maximum postprandial plasma total TAG concentration (TAG T_{MAX}) and CM-TAG concentration (CM-TAG T_{MAX}) was not significantly different between groups or test meals.

Fatty acid composition of chylomicrons

The fatty acid compositions of the different fats used in the present study together with the compositions of their respective CM isolated every second hour are shown in Tables 3, 4 and 5. The fatty acid compositions in the fasting state were similar for all 3 d of treatment. The overall fatty acid composition was not very different between the two

Table 5. Major fatty acids in puff-pastry margarine (PPM) and chylomicrons isolated every second hour in the postprandial period (g/100 g total fatty acids)§
(Mean values and standard deviations for eight normal-weight and seven obese women)

			Normal-weight					Obese				
Time (h) …			0	2	4	6	8	0	2	4	6	8
Fatty acid		PPM										
16 : 0	Mean	34·5	23·7	31·0	29·8	30·1	27·8	23·0	29·7	29·9	29·3	26·9
	SD		1·6	0·7	1·0	0·9	1·2	4·1	1·8	1·4	2·3	1·7
16 : 1*n*-7	Mean	0·1	2·4	0·6	0·7	0·6	0·9	3·1	1·2	1·2	1·2	1·9
	SD		1·1	0·2	0·2	0·2	0·2	1·7	0·9	0·9	0·8	0·9
18 : 0	Mean	7·4	8·8	8·1	8·5	8·2	6·7	7·0	7·6	7·7	7·6	7·5
	SD		1·3	0·4	0·3	0·5	0·5	2·3	0·9	0·6	0·7	1·4
18 : 1 *trans*†	Mean	7·0	0·8	5·3	4·9	5·1	3·7	0·7	4·5	4·5	4·3	2·9
	SD		0·3	0·3	0·4	0·6	0·8	0·4	0·6	0·6	0·8	0·8
18 : 1 *cis*‡	Mean	31·5	28·5	31·6	30·2	29·9	26·9	35·5*	33·3	33·0	32·6	31·5
	SD		4·8	0·7	1·2	2·5	3·0	3·8	1·0	0·6	1·2	2·7
18 : 2*n*-6	Mean	14·5	17·2	15·9	16·5	16·6	18·2	16·5	16·0	15·6	16·4	18·0
	SD		2·6	0·7	1·0	0·9	1·6	2·8	1·6	1·2	1·7	2·7
18 : 3*n*-3	Mean	2·1	0·8	1·4	1·3	1·2	1·0	1·4	1·5	1·3	1·2	1·0
	SD		0·8	0·2	0·3	0·2	0·1	1·0	0·3	0·2	0·2	0·4

Mean values were significantly different from those of the control group: * $P < 0·05$.
† The sum of *trans* isomers.
‡ The sum of *cis* isomers.
§ For details of test meals and procedures see pp. 470–471.

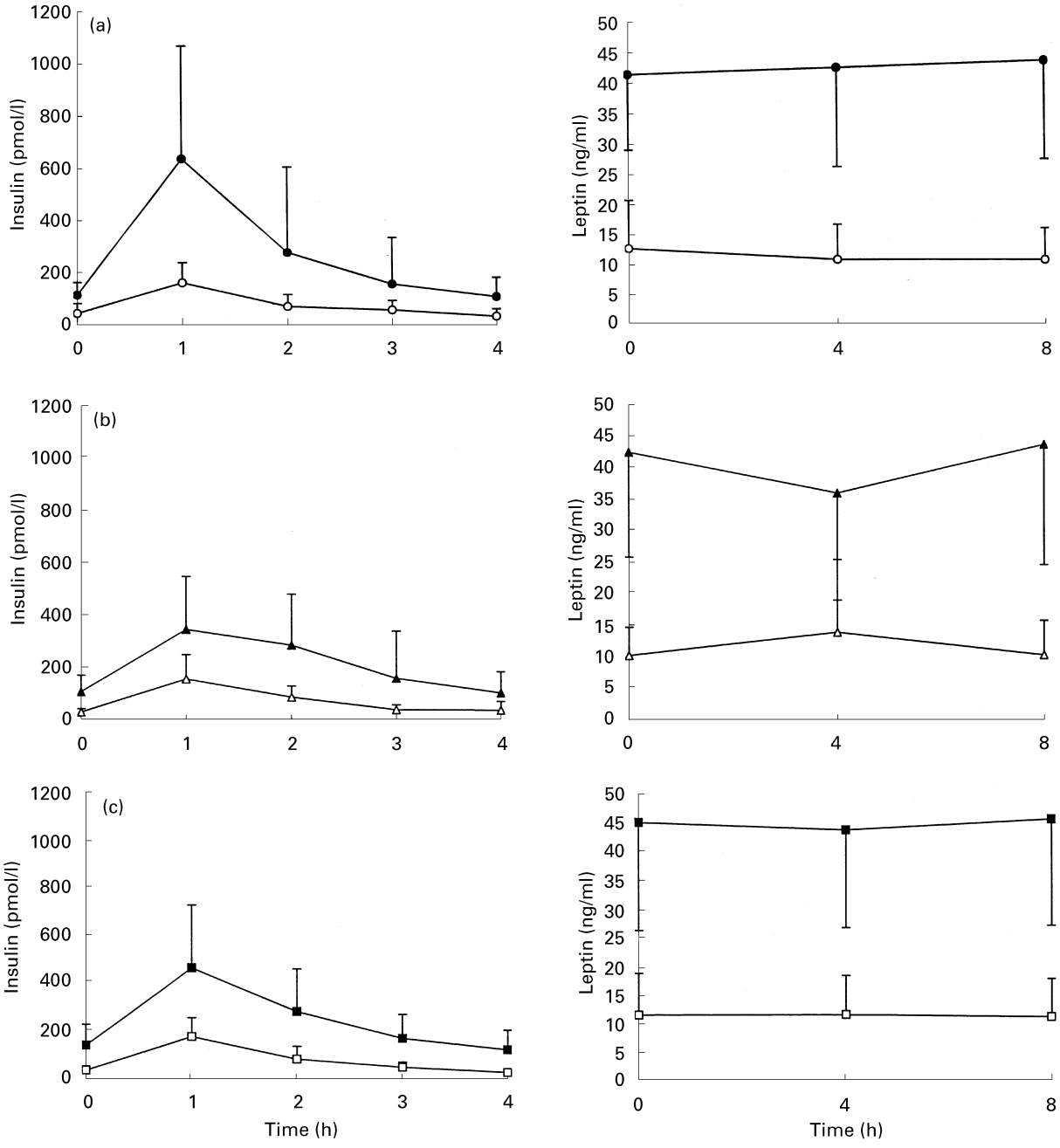

Fig. 2. Insulin responses (0–4 h) and concentrations of leptin (0, 4 and 8 h) after test meals containing (a) palm oil (○, ●), (b) lard (△, ▲), and (c) puff-pastry margarine (□, ■) in normal-weight (○, △, □) and obese (●, ▲, ■) women. Values are means with standard deviations represented by vertical bars. For details of test meals and procedures see pp. 470–471.

groups, however, the obese women had significantly higher contents of *cis* 18 : 1 isomers compared with the normal-weight women ($P < 0{\cdot}05$). The fasting values of 18 : 2*n*-6 were relatively high for both groups and not significantly different. In the postprandial period the fatty acid composition of CM lipids reflected that of dietary fat and particularly so at the maximal absorption (2 h).

Cholesterol

There was no statistically significant difference between the obese and the normal-weight women for fasting total CH (4·6 (SD 0·9) *v.* 4·3 (SD 0·2) mmol/l respectively) or LDL-CH (2·8 (SD 0·6) *v.* 2·2 (SD 0·3) mmol/l respectively). However, fasting HDL-CH was significantly lower in the obese women compared with the normal-weight women (1·2 (SD 0·1) *v.* 1·7 (SD 0·1) mmol/l respectively, $P < 0{\cdot}01$).

Insulin and leptin

Table 2 presents the fasting concentrations and postprandial responses of insulin and leptin in normal-weight and obese women. The fasting insulin concentrations were significantly higher in the obese women than in the

normal-weight women ($P < 0{\cdot}001$). The insulin concentrations increased significantly at 1 h in both groups and returned to baseline at 3–4 h (Fig. 2). The incremental areas under the insulin response curves were significantly higher in the obese women compared with the normal-weight women ($P < 0{\cdot}001$) (Table 2). However, there was no significant difference in the incremental AUC following the three test meals for either of the two groups. The fasting leptin concentrations were significantly higher in the obese group than in the normal-weight group ($P < 0{\cdot}001$) (Table 2) but there were no significant postprandial changes in the concentrations of leptin (Fig. 2).

Discussion

Information about the acute effect on lipaemia is sparse in obese subjects after consumption of different types of fat. Most previous studies have focused on postprandial insulinaemia and glycaemia (Rasmussen *et al.* 1996; Christiansen *et al.* 1997). Our results indicate that test meals containing PO, LD, or PPM produce similar postprandial responses of plasma total TAG and CM-TAG in both normal-weight and obese young women, although the differences in the fatty acid composition of the fats were distinct. However, we cannot exclude that even more marked differences in the fatty acid pattern would have resulted in different postprandial responses.

The amount of fat administered was related to the subject's body surface area; thus, generally, the obese women consumed more fat than the normal-weight women (51 (SD 4) and 64 (SD 6) g per meal respectively). The reason for this was based on an earlier study of Griffin *et al.* (1945) who found a slightly better correlation between plasma volume and body surface area compared with either weight or height. The obese women had significantly higher postprandial responses of TAG than the normal-weight women. This supports earlier investigations of obesity where body surface area also was used to determine the amount of administered fat (Lewis *et al.* 1990, 1991; Couillard *et al.* 1998). In studies of obesity where all subjects were fed on the same amount of fat the obese subjects still displayed greater elevations in TAG concentrations in the postprandial period (Coppack *et al.* 1992; Potts *et al.* 1994, 1995).

In agreement with previous studies fasting TAG concentrations were significantly higher in the obese compared with the normal-weight women (Lewis *et al.* 1990; Potts *et al.* 1995; Couillard *et al.* 1998). In some of the earlier studies the magnitude of the postprandial TAG responses was found to be closely related to the fasting TAG concentrations (Lewis *et al.* 1990, 1991; O'Meara *et al.* 1992; Potts *et al.* 1994; Couillard *et al.* 1998), but this association was not found to be significant in the present study. However, the fasting TAG concentrations in this study, as in others (Lewis *et al.* 1990; Akanji *et al.* 1992; O'Meara *et al.* 1992; Potts *et al.* 1994; Couillard *et al.* 1998) were related to the obesity of the women.

The pattern of postprandial TAG varies considerably between subjects (Cohn *et al.* 1988*b*); plasma total TAG may be found to peak either once, twice, or three times during the postprandial period. The majority of the women in the present study showed a biphasic response curve. The nearly fat-free (<1 g fat) lunch after 4 h could have influenced the response curves of TAG. This is based on the study of Ercan *et al.* (1994) who investigated whether fat ingestion with a morning meal had an effect on TAG responses to a second meal not containing fat, and observed a biphasic curve. The two incremental AUC (0–4 h and 4–8 h respectively) were similar. If fat was present in the second meal the areas were even greater. Nevertheless, no increase in the postprandial TAG concentrations in plasma or lipoprotein fractions was seen after ingestion of 5 g fat (Jeppesen *et al.* 1995). The rise in plasma total TAG corresponded to marked increases in the TAG concentrations of CM and a positive correlation between the postprandial areas was found (r 0·79; $P < 0{\cdot}05$).

The difference in the level of plasma total TAG and CM-TAG represents the contribution of TAG from other lipoproteins especially VLDL with minor contributions from LDL and HDL (Potts *et al.* 1994). VLDL is the major lipoprotein present in the fasting state and contaminations of the CM fractions with VLDL have been revealed by combining centrifugation with SDS-polyacrylamide gel electrophoresis (Potts *et al.* 1994). A contamination would explain the higher fasting concentrations of CM-TAG in the obese women and in addition the fatty acid composition of the CM lipids. Probably the higher VLDL levels in the obese women (results not shown) are due to an increased hepatic VLDL production despite higher fasting insulin levels, i.e. reflecting a higher degree of insulin resistance. It was notable that the obese women had significantly higher contents of *cis* 18 : 1 isomers than the normal-weight women. These differences between the two groups could be a result of different food intake the day before the study or a contamination of VLDL particles. The fatty acid composition of the fasting VLDL fractions (results not shown) showed low contents of 18 : 0 (approximately 5 g/100 g total fatty acids) and high contents of *cis* 18 : 1 isomers (approximately 34 g/100 g total fatty acids) in both groups. However, the fasting fatty acid compositions for normal-weight and obese women were surprisingly equal, indicating that the fats consumed by the obese women are not characterized by high amounts of the more atherogenic SFA and correspondingly lower levels of PUFA. The obesity is therefore not related to differences in oxidative degradation of ingested fatty acids.

The fatty acid pattern after the meals changed during the first 2 h; thus the composition of CM lipids reflected that of the dietary fat ingested at peak absorption (2 h). This corroborates with previous postprandial studies of compositional changes of CM lipids in human subjects (Bonanome & Grundy, 1989; Nestel *et al.* 1995) and in rats (Lambert *et al.* 1996). However, no changes appeared for either of the two groups through the postprandial period (2–6 h), indicating that no preferential clearing of TAG occurred. The fatty acid compositions gradually approached the fasting values when the CM-TAG concentrations declined after 7–8 h. The obese women had significantly lower concentrations of HDL-CH than the normal-weight women, which is in agreement with earlier findings (Lewis *et al.* 1990; Couillard *et al.* 1998). As the total CH is equal for the two groups, the lower HDL-CH was probably compensated for by a higher amount of CH in the VLDL fraction. Despres *et al.*

(1987) suggested that adipose cells, particularly enlarged cells from abdominal subcutaneous fat in obesity, could be directly responsible for the decreased plasma HDL-CH concentrations due to increased uptake of CH.

As expected, the fasting concentrations of insulin and the postprandial response areas were significantly higher in the obese group than in the normal-weight group but there was no statistically significant difference between the three test meals. Gatti *et al*. (1992) reported similar insulin responses in normal volunteers after ingestion of saturated (butter) and unsaturated fatty acids (olive and maize oil) compared with bread alone. In contrast, Rasmussen *et al*. (1996) reported elevated insulin concentrations in patients with non-insulin-dependent diabetes mellitus after ingestion of mashed potato with saturated fat (butter) compared with potato alone, whereas addition of monounsaturated oil (olive oil) apparently had no effect. The effect of butter agrees with findings of Gannon *et al.* (1993) and Collier *et al.* (1988). However, the studies mentioned used a meal without fat as reference.

Recent studies have documented an increased level of serum leptin in obese human subjects (Hamilton *et al*. 1995; Lönnqvist *et al*. 1995; Maffei *et al*. 1995; Considine *et al*. 1996*b*). These observations are consistent with the results of the present study where we found a four-times higher level of leptin in the obese group compared with the normal-weight group and strong positive correlations between serum leptin concentrations and BMI and fasting insulin concentrations (r 0·80; $P < 0·01$ and r 0·89; $P < 0·01$ respectively). As the *ob* gene is normally expressed in adipocytes of obese subjects and as the coding sequence of *ob* gene is not altered in obese subjects (Considine *et al*. 1995, 1996*a*), obese subjects appear to be resistant to their endogenous leptin. A number of studies indicate that leptin levels increase 5–10 h after meals (Kolaczynski *et al*. 1996*b*; Malmström *et al*. 1996; Laughlin *et al*. 1997; Schoeller *et al*. 1997; Saad *et al*. 1998). However, most studies have been unable to detect that the increase in insulin secondary to feeding does increase serum leptin (Kolaczynski *et al*. 1996*a*; Muscelli *et al*. 1996; Pratley *et al*. 1996; Ryan & Elahi, 1996; Vidal *et al*. 1996). The present data corroborate the opinion that metabolic regulation of leptin secretion most probably occurs at the level of transcription and is not due to acutely regulated secretory pathways. Furthermore, it is in agreement with the study of Joannic *et al*. (1998) showing no effect of changing the content of fatty acids from MUFA to PUFA in the test meals.

In summary, our results clearly demonstrate that obese women have exaggerated postprandial lipid and hormone responses compared with those of normal-weight women. The different fatty acid compositions of normal dietary fats such as PO, LD, and PPM show no influence on the measured postprandial responses. The unchanged fatty acid profiles during the postprandial period indicate that neither discrimination in the absorption nor any preferential clearing took place.

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