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Polyunsaturated fatty acid-rich diets: effect on adipose tissue metabolism in rats

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The aim of the present study was to evaluate the effect of diets rich in *n*-6 and *n*-3 fatty acids on adipose tissue metabolism. Starting at weaning, male Wistar rats were fed *ad libitum*, for 8 weeks with one of the following diets: C, rat chow; S, rat chow containing 15 % (w/w) soyabean oil; F, rat chow containing 15 % (w/w) fish oil; SF, rat chow containing 15 % (w/w) soyabean and fish oil (5:1, w/w). Casein was added to the fat diets to achieve the same 20 % (w/w) protein content as in the control chow. Food intake and body weight were measured weekly. The rats were killed by decapitation and the retroperitoneal (RET) and epididymal (EPI) white adipose tissues were removed and weighed. Tissue lipid and protein content, *in vivo* lipogenesis rate, uptake of diet-derived lipids, *in vitro* lipolytic rate, adipocyte area, lipoprotein lipase, ATP citrate lyase, and malic enzyme activities were evaluated. Carcass lipid and protein contents were also measured. Energy intake was reduced while carcass lipid content was increased in the three fat-fed groups. However, carcass protein and body weight gains were elevated only with diets F and SF. Lipolysis rate was diminished by diets F and SF, while the uptake of diet-derived lipids was elevated by the diet S in both RET and EPI tissues. These metabolic alterations may have contributed to the increase in *in vivo* lipogenesis rate in the presence of decreased ATP citrate lyase and malic enzyme activities induced by the three lipid diets. These results indicate that enrichment of the diet with polyunsaturated fatty acids causes changes in adipose tissue metabolism that favour fat deposition. Different metabolic pathways were preferentially affected by each type of fatty acid used.

***n*-6 and *n*-3 Fatty acid-rich diets: Adipose tissue metabolism: Nutrition**

Although it is well documented that the consumption of high-fat diets can induce obesity, the effect of the type of dietary fatty acid on lipid accumulation has not been elucidated. We have previously shown that feeding young rats for 8 weeks on diets containing either *n*-6 polyunsaturated fatty acid (PUFA) or long-chain saturated fatty acids, as 33 % total energy produced similar elevations in body-weight gain and carcass fat content (Gaiva Gomes da Silva *et al.* 1996). Similar results were obtained by Awad *et al.* (1990). In contrast, Shimomura *et al.* (1990) reported that a safflower oil diet produced a lower body-fat gain in young rats than a tallow diet, both at 45 % total energy. However, rats fed a maize oil diet for 9 months were heavier and fatter than those that received a lard diet (Hill *et al.* 1993).

The *n*-3 PUFA found in fish oils have received considerable interest, since they have been shown to exert beneficial health effects (Calder, 1998). Tsuboyama-Kasaoka *et al.* (1999) have demonstrated that mice receiving 60 % dietary energy as *n*-3 fatty acids during 5 months did not develop obesity. Contrarily, a fish oil diet elevated body fat and lowered body protein content, compared with a safflower oil diet (Dulloo *et al.* 1995), while no difference in body-weight gain was observed between rats fed lard or an *n*-3 fatty acid-supplemented lard diet (Rustan *et al.* 1993).

Abbreviations: C, control group; EPI, epididymal white adipose tissue; F, fish group; LPL, lipoprotein lipase; PUFA, polyunsaturated fatty acids; RET, retroperitoneal white adipose tissue; S, soyabean group; SF, soyabean and fish group.

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The white adipose tissue is the most important extrahepatic site regulating *in vivo* lipid metabolism. Tissue triacylglycerol deposition results from *in situ de novo* synthesis, fatty acid uptake from lipoproteins of dietary or hepatic origin and lipolysis rate. The impact of dietary fatty acid composition on adipose tissue lipid metabolism has been examined by some authors, with conflicting results. No effect on lipolysis and lipogenesis rates was reported by Awad *et al.* (1990) when comparing *n*-6 PUFA, *n*-3 PUFA, and saturated diets, while Fickova *et al.* (1998) found higher noradrenaline-stimulated lipolysis in rats fed *n*-3 PUFA than in those fed *n*-6 PUFA. Diets enriched with *n*-6 PUFA have been shown to decrease fatty acid synthase mRNA in liver and adipose tissue, and thus lipogenesis capacity, in rats (Tsuboyama-Kasaoka *et al.* 1999).

The purpose of the present study was therefore to examine the effect of diets rich in *n*-6 and *n*-3 fatty acids on fat accumulation and on adipose tissue lipid metabolism in rats.

Materials and methods

Animals

The Experimental Research Committee of the Federal São Paulo University approved all procedures involving animals. We used male Wistar rats supplied by the animal care facility of the Physiology Department of the São Paulo Federal University. Immediately after weaning (day 30 of life), the animals were divided into four groups of sixteen rats each, according to diet composition. All groups were maintained in a room at 23 ± 1°C with lights on from 07.00 to 19.00 hours and fed *ad libitum* the diets for 8 weeks. After the treatment, eight rats from each group were used to measure lipogenesis rate, enzyme activities, lipolysis rate and adipocyte area, and another eight rats from each group were used to measure [^{14}C]triolein uptake by adipose tissue and carcass lipid and protein content.

Preparation of the diets

The groups were offered one of the following diets: (1) control group (C), a commercial rat chow (Nuvilab CR-1, Paraná, Brazil) consisting of (w/w) 20 % protein and 3 % fat; (2) soyabean group (S), a commercial rat chow plus PUFA, prepared by adding 15 % (w/w) soyabean oil to the chow; (3) fish group (F), commercial rat chow plus PUFA, prepared by adding 15 % (w/w) fish oil (Sigma, St Louis, MO, USA) to the chow; (4) soyabean plus fish group (SF), a commercial rat chow plus PUFA, prepared by adding 15 % (w/w) soyabean oil and fish oil (ratio 5:1, w/w).

Casein was added to the lipid diets to achieve 20 % (w/w) protein. Diet C contained 17·4 kJ/g and the diets rich in fatty acids contained 20·5 kJ/g, with 33 % of the total energy from lipids. The detailed preparation of the diets has been presented previously (Guimarães *et al.* 1990).

Food was given fresh each day and 24 h food intake and body weight were recorded weekly.

Experimental procedure

Measurements of lipogenesis rate. Rats were killed by decapitation, 1 h after the intraperitoneal administration of 0·111 GBq $^{3}H_2O$. Retroperitoneal (RET) and epididymal (EPI) white adipose tissues were removed and weighed. Aliquots (500 mg) were saponified and the fatty acids extracted by the method of Stansbie *et al.* (1976). Tissue lipogenesis rate was expressed as μmol $^{3}H_2O$ incorporated into lipid/h.g tissue (Robinson & Williamson, 1978). Lipid content was measured as described by Oller do Nascimento & Williamson (1986).

Adipocytes area determination. A fragment (50 mg) of RET and EPI was fixed in 0·2 M-collidine buffer, pH 7·4, containing 2 % (w/v) OsO_4 at 37°C. After 48 h, these were washed with warmed saline (9 g NaCl/l), as described by Hirsch & Gallian (1968). The adipocyte area was measured using an image analysis software (Leica Q 500 MC; Lecia Imaging Systems Ltd, USA) and expressed as μm^2.

Enzymes activities. Portions (about 1 g) of the RET and EPI tissues were used to determine enzymes activities. Lipoprotein lipase (LPL) activity was determined as previously described by Nilsson-Ehle & Schotz (1972) and modified by Llobera *et al.* (1979), and the results expressed as nmol fatty acids released/min per mg protein. Tissue protein content was determined as in Lowry *et al.* (1951). Malic enzyme activity was measured according to Newsholme & Williams (1978) and expressed as μmol/min per 100 mg tissue. ATP-citrate lyase activity was measured as described by Corrigan & Rider (1983) and expressed as μmol/min per 100 mg tissue.

In vitro *determination of lipolytic rate*. Tissue fragments (about 100 mg) were used for the *in vitro* determination of glycerol release, an index of lipolytic rate (Arner & Engfeldt, 1987). The samples were minced into small fragments and incubated for 1 h at 37°C under continuous shaking in Ca^{2+}-free Krebs–Henseleit solution containing 3 % (w/v) bovine albumin (fraction V, essentially fatty acid-free), pH 7·4. Lipolysis was interrupted by the addition of 2 M-H_2SO_4. Tissue fragments were then removed. The medium was neutralized with NaOH and the glycerol content determined enzymically by the method of Eggstein & Kreutz (1966). For each donor rat, two sets of incubation vials were prepared in duplicate, one with and the other without 100 μM-noradrenaline (Arterenol; Sigma Chemical Co., St Louis MO, USA) in the incubation medium. The results were expressed as μmol glycerol released/h per 100 mg tissue.

Measurement of ^{14}C-labelled lipid absorption from intestine and accumulation in tissue. The rats received an intragastric load of [1-^{14}C]triolein (about 0·5 g; 0·3 11·1 Bq/rat). After 4 h, the rats were killed by decapitation. The whole intestinal tract and samples (about 1 g) of RET and EPI were removed and weighed. The intestinal tract was homogenized with water (1:1, w/v) in a Waring blender. KOH (30 % (w/v); 3 ml) was added to the intestinal tract homogenates (2–3 g) and white fat samples. The lipids were saponified and extracted by the method of Stansbie *et al.* (1976). The extracted fatty acids were dissolved in 5 ml scintillation liquid, and the radioactivity was measured to determine the ^{14}C-labelled lipid accumulated in the tissues

Table 1. Body weight (g), food intake (g/d), and energy intake (kJ/100 g body weight per d) of rats fed the control diet (C), soyabean diet (S), fish diet (F) or soyabean plus fish diet (SF) during 8 weeks of treatment§
(Mean values with their standard errors for sixteen animals)

Week of treatment		Initial		1		2		3		4		5		6		7		8	
Dietary group		Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
C	Body weight	45·77	0·81	67·8	5·1	112·6	9·2	151·5	8·8	173·0	10·0	203·5	9·3	226·3	7·1	251·4	5·8	269·8	4·7
	Food intake			11·6	0·2	12·9	0·3	15·5	0·8	21·5	0·4	22·3	0·4	24·1	1·5	27·7	7·1	29·3	2·1
	Energy intake			315·1	19·2	238·1	25·9	194·1	20·5	223·4	9·6	197·9	5·9	185·8	9·6	189·5	18·4	191·6	14·2
S	Body weight	43·75	1·52	76·6	3·9	116·1	6·8	154·3	6·6	176·2	9·4	212·0	9·7	239·8	7·8	263·3	7·1	283·9	7·4
	Food intake			9·6*	0·5	12·4	0·8	12·1*	0·6	13·1*	0·5	15·0*	0·5	16·2*	0·4	16·6*	0·2	18·7*	2·3
	Energy intake			257·5	4·2	220·5	5·0	160·2*	2·9	157·3*	4·6	147·3*	4·2	131·4*	8·8	131·8*	5·0	136·8*	4·2
F	Body weight	46·22	1·44	78·3	4·0	122·9	7·6	159·4	7·8	192·7	8·5	232·6*	7·9	264·7*†	6·3	295·7*†	5·5	317·4*†	3·8
	Food intake			9·5*	0·3*	12·1	0·4	14·3†	0·5	16·2*	0·4	16·7*	0·6	16·8*	0·5	17·9*	0·3	18·8*	0·6
	Energy intake			251·9	5·9	207·9	7·9	185·4	4·6	166·9*	9·6	148·5*	3·8	130·1*	2·9	124·3*	1·7	122·2*	1·7
SF	Body weight	42·35	1·59	74·4	5·1	115·7	8·2	153·7	6·9	196·0	5·9	234·6*	4·4	264·0*†	3·2	292·4*†	3·2	312·1*†	4·0
	Food intake			11·4†‡	1·2	14·3	1·7	15·3†	0·4	15·8*	0·3	16·3*	2·3	15·6*	1·7	15·3*	0·7	16·4*	2·1
	Energy intake			305·0	13·4	242·3	11·7	205·0†	4·2	168·6*	3·3	143·5*	1·7	121·8*	1·7	107·0*	1·3	108·7*†	1·7

Mean values were significantly different from those for group C: * $P \leq 0.05$.
Mean values were significantly different from those for group S: † $P \leq 0.05$.
Mean values were significantly different from those for group F: ‡ $P \leq 0.05$.
§ For details of diets and procedures, see p. 372.

and the amount of ^{14}C-labelled lipid that remained in the intestinal tract. The absorption of ^{14}C-labelled lipid was determined by subtracting the radioactivity remaining in the intestinal tract from the amount administered.

Statistical analysis

The results are expressed as mean values with their standard errors. Intergroup comparisons were performed by one-way ANOVA followed by the Duncan's test. The Student's *t* test was used to compare basal and noradrenaline-stimulated lipolysis rates. Significance was set at the $P \leq 0.05$ level.

Results

Body weight, food intake and carcass lipid and protein content

Body weight was increased for groups F and SF from the fifth to the eighth week of treatment, as compared with group C, and from the sixth until the eighth week compared with the S group. Food and energy intakes were lower for the lipid groups than for group C from the fourth week until the end of the experimental period (Table 1).

Carcass lipid content for the lipid groups was higher than that for the group C rats. Protein content was higher for groups F and SF than that for groups C and S (Table 2).

Retroperitoneal white adipose tissue

RET weight was increased for group S as compared with group C, and for group SF as compared with groups C, S and F. For SF group lipid content increased and protein content decreased in comparison with both groups C and S. The adipocyte area was higher for group SF than in groups C and F. The *in vivo* lipogenesis rate was increased in RET by all fatty acid diets, while the activities of malic enzyme and ATP-citrate lyase were decreased (Table 3).

The basal lipolytic rate was not affected by fat enrichment of the diet, although the noradrenaline lipolytic effect was decreased for group F compared with group C and for group SF in relation to groups C and S (Table 4).

As shown in Table 5, the percentage accumulation of diet-derived lipid in RET was significantly increased for group S (127; $P<0.05$) in comparison with C, while the increments observed for groups F (62) and SF (75) did not reach statistical significance. An effect of diet on LPL activity was seen only for group SF, with a 39 % reduction when compared with group C.

Epididymal white adipose tissue

When compared with group C, EPI weight was increased for groups F and SF. However, adipocyte area was decreased for group F only. All diets rich in fatty acids had decreased protein contents and ATP-citrate lyase activities and increased lipogenesis rates, as compared with group C (Table 6). The basal lipolytic rate was decreased for group F relative to groups C and S, and for group SF as compared with group C. Noradrenaline-stimulated lipolysis in all groups, although its effect was less pronounced for groups F

Table 2. Carcass total lipid and protein content of rats fed the control diet (C), soyabean diet (S), fish diet (F) or soyabean plus fish diet (SF) during 8 weeks of treatment‡

(Mean values with their standard errors for eight animals)

Dietary group…	C		S		F		SF	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Carcass total lipid content (g)	7·8	0·6	12·6*	1·1	12·1*	1·1	12·9*	1·0
Carcass total protein content (g)	49·8	2·0	50·8	4·0	59·6*†	1·4	63·2*†	1·4

Mean values were significantly different from those for group C: * $P<0·05$.
Mean values were significantly different from those for group S: † $P<0·05$.
‡ For details of diets and procedures, see p. 372.

and SF compared with that for groups C and S (see Table 4). As shown in Table 5, the percentage of diet-derived lipid accumulated in EPI was significantly increased for group F (108; $P<0·05$) and increased nonsignificantly for groups SF (41) and S (58). LPL activity was not affected by any of the diets rich in fatty acid.

^{14}C-labelled lipid absorption from intestine

As compared with C group, the absorption of ^{14}C-labelled lipid by the intestinal tract was increased for groups S and SF (see Table 5).

Discussion

In the present study feeding rats PUFA-rich diets resulted in a significant decrease in food intake ($P\leq0·05$; Table 1), in agreement with previous reports (Silveira *et al.* 1995; Himaya *et al.* 1997). Several studies, in animals and human subjects, have shown that high-lipid diets increase the secretion of cholecystokinin, a hormone involved in the satiety process (Maggio *et al.* 1988; French *et al.* 1995; Horn *et al.* 1996). Starting on the fourth week of the study, the energy intake, although similar among the lipid-fed groups, was lower than that of group C (Table 1). However, increased body weight was observed for groups F and SF, but not for group S (Table 1). This finding indicates that there was a higher food efficiency for the lipid-fed groups. This fact has been reported previously and attributed to the lower energetic cost of lipid deposition from dietary fat than from dietary carbohydrate (Oudart *et al.* 1997). Moreover, the presence of *n*-3 PUFA resulted in increased carcass protein content (Table 2), in accordance with earlier reports (Su & Jones, 1993).

In agreement with the present findings, high dietary fat has been shown to increase body adipose mass and to affect tissue metabolism (Frayn *et al.* 1992), even in the absence of increased energy intake (Boozer *et al.* 1995; West & York, 1998). Additionally, regional differences in the sensitivity of adipose tissue depots to dietary manipulations have also been found (Masoro, 1981; Belzung *et al.* 1993). In the present study we also observed some differences between RET and EPI responses to the fatty diets. For instance, diet S significantly increased RET weight and ^{14}C-labelled lipid accumulation, while the same variables were affected in EPI by diet F.

All PUFA-rich diets increased incorporation of $^{3}H_2O$ into lipid and decreased lipogenic enzyme activities in both tissues. Increased RET lipogenesis rate after *n*-3-and *n*-6-rich diets has been reported previously (Raclot & Groscolas, 1994; Fickova *et al.* 1998). However, a distinction in the metabolic pathway preferentially affected by each PUFA type could be observed, as the presence of fish oil was necessary to decrease lipolysis. This observation is consistent with the reported fish oil-induced reduction in plasma free fatty acids (Otto *et al.* 1992) and elevation of

Table 3. Tissue weight, lipid and protein content, adipocyte area, lipogenesis rate, malic enzyme activity and ATP-citrate lyase activity in retroperitoneal white adipose tissue of rats fed the control diet (C), soyabean diet (S), fish diet (F) or soyabean plus fish diet (SF) during 8 weeks of treatment§

(Mean values with their standard errors for eight animals)

Dietary group…	C		S		F		SF	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Tissue weight (g)	1·82	0·23	3·31*	0·49	2·72	0·26	4·46*†‡	0·6
Lipid content (mg/100 mg)	82·7	1·9	77·4	1·7	85·9†	1·9	89·1*†	2·4
Protein content (mg/100 mg)	28·8	3·5	28·3	2·6	22·8	1·2	17·1*†	1·5
Adipocyte area (μm^2)	5563	545	7319	640	6115	284	9081*‡	1265
Lipogenesis rate (μmol $^{3}H_2o$ incorporated into lipid/h per g tissue)	0·75	0·08	2·22*	0·49	2·76*	0·13	2·58*	0·23
Malic enzyme activity (μmol substrate utilized/min per g tissue)	0·78	0·09	0·41*	0·04	0·61*†	0·05	0·25*‡	0·03
ATP-citrate lyase activity (μmol substrate utilized/min per g tissue)	0·35	0·08	0·08*	0·02	0·05*	0·01	0·05*	0·02

Mean values were significantly different from those for group C: * $P<0·05$.
Mean values were significantly different from those for group S: † $P<0·05$.
Mean values were significantly different from those for group F: ‡ $P<0·05$.
§ For details of diets and procedures, see p. 372.

Table 4. Lipolysis *in vitro* in retroperitoneal (RET) and epididymal (EPI) white adipose tissues of rats fed the control diet (C), soyabean diet (S), fish diet (F) or soyabean plus fish diet (SF) during 8 weeks of treatment§

(Mean values with their standard errors for eight animals)

	In vitro lipolysis rate (μmol glycerol released/h per 100 mg tissue)							
	RET				EPI			
	Basal		Nor		Basal		Nor	
Dietary group	Mean	SE	Mean	SE	Mean	SE	Mean	SE
C	0·06	0·01	0·19*	0·04	0·07	0·01	0·26*	0·03
S	0·07	0·01	0·15*	0·02	0·05	0·01	0·25*	0·03
F	0·04	0·01	0·09*†	0·01	0·02†‡	0·04	0·12*†‡	0·02
SF	0·03‡	0·01	0·05*‡	0·01	0·04†	0·01	0·12*†‡	0·01

Mean values were significantly different from those for basal: * $P<0·05$.
Mean values were significantly different from those for group C: † $P<0·05$.
Mean values were significantly different from those for group S: ‡ $P<0·05$.
§ For details of diets and procedures, see p. 372.

Table 5. Accumulation of ^{14}C-labelled lipid (^{14}C-lipid; % absorbed dose/g tissue) and lipoprotein lipase enzyme activity (LPL; nmol fatty acid release/min per mg protein) in retroperitoneal (RET) and epididymal (EPI) white adipose tissues and the absorption of ^{14}C-labelled lipid by intestinal tract (IT; % administered dose) of rats fed the control diet (C), soyabean diet (S), fish diet (F) or soyabean plus fish diet (SF) for 8 weeks at 4 h after intragastric administration of [^{14}C] triolein§

(Mean values with their standard errors for eight animals]

Tissues…	RET				EPI				IT	
	^{14}C-lipid		LPL		^{14}C-lipid		LPL		Lipid absorption	
Dietary group	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
C	0·37	0·07	1·60	0·22	0·58	0·12	0·85	0·05	67·9	3·90
S	0·84*	0·10	1·26	0·06	0·92	0·14	0·78	0·06	82·3*	4·20
F	0·60	0·13	1·22	0·14	1·21*	0·23	0·96	0·14	66·5†	5·36
SF	0·65	0·09	0·97*	0·13	0·82	0·11	0·88	0·08	83·2*‡	3·48

Mean values were significantly different from those for group C: * $P<0·05$.
Mean values were significantly different from those for group S: † $P<0·05$.
Mean values were significantly different from those for group F: ‡ $P<0·05$.
§ For details of diets and procedures, see p. 372.

Table 6. Tissue weight, lipid and protein content, adipocyte area, lipogenesis rate, malic enzyme activity and ATP-citrate lyase activity in the epididymal white adipose tissue of rats fed the control diet (C), soyabean diet (S), fish diet (F) or soyabean plus fish diet (SF) during 8 weeks of treatment§

(Mean values with their standard errors for eight animals)

Dietary group…	C		S		F		SF	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Tissue weight (g)	2·69	0·21	3·53	0·42	3·80*	0·37	4·06*	0·26
Lipid content (mg/100 mg)	81·6	3·7	79·1	2·2	86·9	3·3	86·2	2·4
Protein content (mg/100 mg)	48·4	7·2	34·7*	0·4	32·5*	3·1	23·9*	2·1
Adipocyte area (μm^2)	7904	559	6425	644	5078*	278	7097‡	717
Lipogenesis rate (μmol ^3H_2O incorporated into lipid h per g tissue)	0·94	0·05	2·80*	0·64	2·00*	0·13	2·09*	0·25
Malic enzyme activity (μmol substrate utilized/min per g tissue)	0·44	0·06	0·23*	0·03	0·48†	0·07	0·25*‡	0·03
ATP-citrate activity (μmol substrate utilized/min per g tissue)	0·25	0·06	0·03*	0·01	0·12*†	0·01	0·06*	0·02

Mean values were significantly different from those for group C: * $P<0·05$.
Mean values were significantly different from those for group S: † $P<0·05$.
Mean values were significantly different from those for group F: ‡ $P<0·05$.
§ For details of diets and procedures, see p. 372.

insulin sensitivity (Hill *et al.* 1993). Thus, for groups F and SF, a lower lipolysis rate could explain the higher amount of ^3H_2O incorporation into lipids in both RET and EPI.

The reduction of adipose tissue lipolysis by *n*-3 PUFA has been shown previously and implicated as a mechanism contributing to the plasma triacylglycerol-lowering effect of dietary fish oil (Singer *et al.* 1990; Dagnelie *et al.* 1994). Indeed, eicosapentaenoic acid reportedly inhibited the isoprenaline-induced elevation of cAMP levels in adipocytes (Tisdale, 1993).

In group S increased lipid uptake from circulating lipoproteins in RET (127 %) and EPI (58 %) was observed (Table 5), a finding that could help explain the higher lipogenesis rate and tissue weight (Tables 3 and 6). However, LPL activity, as measured *in vitro*, was unchanged (Table 5). No effect on LPL activity of diets rich in fatty acids has been reported previously (Takeuchi *et al.* 1995). Conversely, a good correlation has been found between the activity of LPL and the uptake of chylomicron triacylglycerols by fat pads (Cryer *et al.* 1974), and increased circulating triacylglycerol levels have been shown to stimulate LPL *in vivo* (Fielding & Frayn, 1998). It is thus possible that the *in vivo* activity of LPL was actually high in groups S and SF, since intestinal lipid absorption was elevated (Table 5).

The results indicate that enrichment of the diet with PUFA causes changes in adipose tissue metabolism that favour fat deposition. Different metabolic pathways are preferentially affected by each type of fatty acid used.

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