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Short term effects of energy restriction and dietary fat sub-type on weight loss and disease risk factors

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Abstract

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Keywords

fat, restriction, effects, factors, risk, short, loss, weight, sub, dietary, energy, term, disease, type

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Short term effects of energy restriction and dietary fat sub-type on weight loss and disease risk factors

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KEYWORDS

Obesity;
Weight loss;
Low fat diets;
Polyunsaturated fatty acids;
Controlled dietary intervention

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Methods and results: One-hundred and fifty overweight men and women were randomized into a 3 month controlled trial with four low fat (30% energy) dietary arms: (1) isocaloric (LF); (2) isocaloric with 10% polyunsaturated fatty acids (LF-PUFA); (3) low calorie (LF-LC) (-2 MJ); (4) low calorie with 10% PUFA (LF-PUFA-LC). Primary outcomes were changes in body weight and body fat and secondary outcomes were changes in fasting levels of leptin, insulin, glucose, lipids and erythrocyte fatty acids. Changes in dietary intake were assessed using 3 day food records. One-hundred and twenty-two participants entered the study and 95 completed the study. All groups lost weight and body fat ($P < 0.0001$ time effect for both), but the LC groups lost more weight ($P = 0.026$ for diet effect). All groups reduced total cholesterol levels ($P < 0.0001$ time effect and $P = 0.017$ intervention effect), but the LC and PUFA groups were better at reducing triacylglycerol levels ($P = 0.056$ diet effect). HDL increased with LF-LC and LF-PUFA but not with LF-PUFA-LC (0.042 diet effect). The LF and LF-LC groups reported greater dietary fat reductions than the two PUFA groups ($P = 0.043$).

Conclusion: Energy restriction has the most potent effect on weight loss and lipids, but fat modification is also beneficial when energy restriction is more modest.

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Abbreviations: PUFA, polyunsaturated fatty acids; LF, low fat; IC, isocaloric; MUFA, monounsaturated fatty acids; SFA, saturated fatty acids; LC, low calorie; VAT, visceral adipose tissue.

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Introduction

Worldwide, more than one billion adults are overweight and more than 350 million are obese. Many will suffer from obesity related diseases, such as cardiovascular diseases and diabetes, and in turn premature death [1]. The

mechanisms behind obesity development are complex and multifactorial but diet and physical activity are central. Decreasing energy intake relative to energy expenditure is the indisputable tenet of weight loss, but success with obesity management remains practically elusive. One of the many ways forward lies with addressing metabolic links between body fat and food components, in particular dietary fat. Lifestyle intervention, including a low calorie, low fat diet has proven to promote weight loss and reduce the incidence of diabetes [2].

In addition to caloric restriction, modifying the type of dietary fat may be beneficial. With respect to cardiovascular risk, focusing on dietary polyunsaturated fat improves circulating triacylglycerol levels, a benefit additional to weight loss [3,4]. Likewise, replacing dietary carbohydrate [5] or saturated fat [6] with unsaturated fats produces favourable changes in circulating lipids. With obese insulin resistant participants, higher proportions of dietary unsaturated fats can reduce central fat distribution [7] and improvements in insulin sensitivity can be observed with modified fat diets where the total fat level is kept below 37% energy [8]. One small study of normal healthy adults, found that replacing 6 g/day of dietary fat with fish oil reduced body fat mass and increased lipid oxidation under isocaloric conditions [9].

Despite this knowledge, the level of obesity appears resistant to interventions. Mechanisms have been put forward to explain why obese people do not lose much weight, suggesting that metabolic adaptations might be worth exploring [10]. For example, feeding studies have shown that fat induced thermogenesis is reduced in obesity, and this could reflect an adaptive response to weight gain [11]. Manipulating the type of dietary fat might ameliorate this effect because polyunsaturated fatty acids (PUFA) are known to suppress the expression of genes associated with lipogenesis, but activate genes involved in fat oxidation [12]. Increasing fat oxidation might help to reduce body fat, although it might also affect further weight loss because the energy deficit required to lose a kilogram of body weight depends on fat mass [13]. Either way, whether dietary manipulation affects the energy deficit required for weight loss over time is a question worth pursuing.

In the end, these are theoretical positions that need to be translated to practice under 'free living' conditions. In this study we used a food guidance system to compare the effect on weight loss and adiposity of a standard low fat diet with a low fat diet inclusive of PUFA rich foods, either under isocaloric or low calorie (-2 MJ/day) conditions. Secondary outcomes included biomarkers of cardiovascular disease and diabetes risk, and changes in energy deficit.

Methods

Study design

The study was a 3 month randomized controlled trial conducted at the Smart Foods Centre at the University of Wollongong, Australia, with four arms:

- (1) Low fat (LF): participants received low fat isocaloric (IC) dietary advice, targeting an intake of 20% protein,

- 50% carbohydrate and 30% fat, (5% PUFA, 15% mono-unsaturated fat (MUFA) and 10% saturated fat (SFA)).

- (2) LF-PUFA: participants received low fat isocaloric dietary advice inclusive of foods high in polyunsaturated fat, targeting 20% protein, 50% carbohydrate, 30% fat (10% PUFA, 10% MUFA and 10% SFA).

- (3) LF-low calorie (LC): participants received the same advice as (1) but with a 2 MJ/day energy restriction.

- (4) LF-PUFA-LC: participants received the same advice as (2) but with a 2 MJ/day energy restriction.

Each subject attended the clinic on a monthly basis during the intervention period to receive dietary counselling support. The outcome measures, (anthropometry, fasting blood for biochemical analyses, body fat distribution and energy deficit) as well as information on diet were measured at baseline and after 3 months. Physical activity was assessed at baseline by a questionnaire [14] for calorimetry protocols and sample characterisation.

The study was approved by the Human Research and Ethics Committee of the University of Wollongong and the South Eastern Sydney and Illawarra Area Health Service, and the trial was registered with the Australian Clinical Trials Registry [ACTRN12608000453381].

Subjects and recruitment

A sample size of 20 in each of four arms was calculated to provide a power of 80% (allowing for three drop-outs per group), with reference to published data on fat modification and visceral adipose tissue changes [15] and assuming a within subject standard deviation equal to change in visceral adipose tissue (VAT) of 20 cm^3 . Even less ($n = 6$) were calculated to provide 80% power for changes in weight based on published data of a similar study [16]. Randomisation into the four intervention arms was conducted using random permuted blocks by a computerised random number generator. Participants were stratified by sex and blinded to the type of dietary intervention. They were informed the aim was to assess effectiveness of dietary interventions but not the differences between diets. It was not possible to blind the dieticians as the advice was targeted to interventions. Participants were recruited through local media advertisements. Eligible participants were men and women aged >18 years, with a BMI >25 . The exclusion criteria for the study were smoking, major illnesses such as cancer and diabetes, taking regular medication (except contraceptives), food allergies, habits inhibiting the study, illiteracy and/or inadequate conversational English.

Diet and physical activity

Individualized dietary advice was based on numbers of serves of food groups defined in the Australian Guide to Healthy Eating [17]. Dietary modelling was undertaken to ensure the advice matched targeted energy and macronutrient levels. Participants were asked not to take fish oil supplements and to walk briskly for 30 min on three

occasions per week. All participants had the same contact with a dietician during the study.

Dietary intake was assessed at baseline and at 3 months using a validated diet history interview [18] and 3 day food records. Dietary data was analysed using the Foodworks software system (Xyris Software, Highgate Hill, QLD, Australia, Version 3, 2002). Nutrient intake data was analysed in terms of energy and macronutrients and fatty acid sub-types using the AusNut and AusFood databases (2001) and the Australian Fatty Acids Rev 6 2002: RMIT, Melbourne, Australia, respectively.

Body weight and body composition

Body weight and percentage body fat was measured in an upright position in minimal clothing (no shoes) using scales with a bioelectrical impedance component (Tanita TBF-622) that compares reasonably well with dual x-ray absorptiometry [19]. An abdominal CT scan was taken at the fourth lumbar vertebra, fifth lumbar vertebra, and the level of the sacroiliac joints by a trained observer at a commercial x-ray facility (Southcoast X-Ray, Wollongong, NSW, Australia). Subcutaneous and visceral adiposity areas were assessed at each of the levels using SIENET Sky software (Siemens Corporation, NY, USA) that drives the Siemens CT scanner (Siemens AG, Munich, Germany). Relative adipose tissue densities at each level were measured at anterior and posterior positions and within the abdominal cavity.

Biochemical analyses

Fasted blood samples were drawn by trained professionals and sent to a quality assured laboratory (Southern IML Pathology, Wollongong, NSW, Australia) for analysis of glucose, lipids and insulin levels. For the lipids, total cholesterol, high density lipoprotein cholesterol (HDL-cholesterol) and triacylglycerol (TG) levels were measured. Low density lipoprotein cholesterol (LDL-cholesterol) was calculated with the Friedewald formula [20].

Plasma leptin was analysed in batches by a single scientist following storage at -80°C , using the radioimmunoassay technique with ^{125}I as the labelling isotope (Human Leptin RIA Kit; Linco Research, St Charles, MO, USA). Each sample was analysed in duplicate, allowing analysis of 112 samples per kit. Gamma radiation emitted by the ^{125}I -labeled sample preparation was measured using the Wallac 1480 WIZARD (PerkinElmer Life Sciences, Inc.) gamma counter. The standard curve based on the gamma counter output was generated using Prism v.4 (Macintosh) computer software by GraphPad Software, Inc (San Diego, CA, USA). Unknown sample concentrations were established using automated calculation of x-axis intercepts based on the standard curve equations.

Erythrocyte fatty acid composition concentrations were analysed by gas chromatography using a Shimadzu GC-17A which included a fused silica $50\text{ m} \times 0.25\text{ mm}$ capillary fully bonded highly polar column with hydrogen as a carrier gas. Identification of fatty acids was based on the retention time of authentic fatty acid methyl ester standards (Sigma Aldrich, Castle Hill, Australia). Fatty acid data was sorted prior to analysis to arrive at a total figure for SFA, MUFA and PUFA.

Measurement of energy deficit

To examine the effect of different dietary strategies, the concept of energy deficit (%) was applied to energy expenditure (EE) data from a sub-sample of participants (given that (EE) was not a primary outcome). The analysis would expose the extent to which dietary energy deficit was responsible for weight loss over time, after considering reductions in EE due to weight loss. For these purposes we defined energy deficit (%) as $[\Delta\text{EI} - \Delta\text{EE}]/\Delta\text{EI} \times 100$, where ΔEI = reduction in energy intake and ΔEE = reduction in energy expenditure. EI was assessed from diet history records and EE was assessed using the Wollongong calorimeter facility, details of which have been previously published [21]. The prescription of meals in the chamber was based on dietary modelling using Foodworks software (Xyris Software, Highgate Hill, QLD, Australia, Version 3, 2002). Height, weight and activity measures were used to predict habitual levels of energy expenditure while in the chamber using predictive equations. Participants were admitted into the calorimeter chamber fasting between 8:30 and 9:00 am and stayed there for 24 h. The participants were provided with three meals and snacks at appropriate times during the stay to provide 85% of their predicted energy requirements (assuming 85% of predicted usual activity occurs in the chamber [22]). The sub-sample was formed from participants willing and available to stay in the calorimeter, and of the 60 completers of this procedure, data was available on 48 participants due to technical issues.

Statistical analyses

Data were analysed using SPSS (version 11.5.0: SPSS Chicago, IL, 2002). Baseline differences between diet groups were assessed using a one-way ANOVA. Changes in the primary and secondary outcome measures were analysed for all four diet groups using the general linear model for repeated measures, with model assumptions checked prior to analysis. Data were adjusted for sex.

Changes in energy deficit were assessed by comparing baseline and 3 month data from two sets of two groups. In each case, the four groups were reduced to two groups. The four groups were as follows: low calorie (LC) (LF-LC and LF-PUFA-LC) vs. isocaloric (IC) (LF and LF-PUFA), and higher PUFA (PUFA) (LF-PUFA and LF-PUFA-LC) vs. lower PUFA (LF) (LF and LF-LC). Differences between the two groups were assessed using independent *t*-tests. Differences in dietary intake were similarly analyzed as LF vs. PUFA group data using independent *t*-tests. Correlations between changes in reported dietary PUFA and erythrocyte PUFA values were examined using Spearman's rank correlation.

Results

Subjects

Of 183 participants screened, 150 were enrolled and randomised into the isocaloric LF ($n = 38$), LF-PUFA ($n = 38$), and low calorie LF-LC ($n = 37$), and LF-PUFA-LC ($n = 37$)

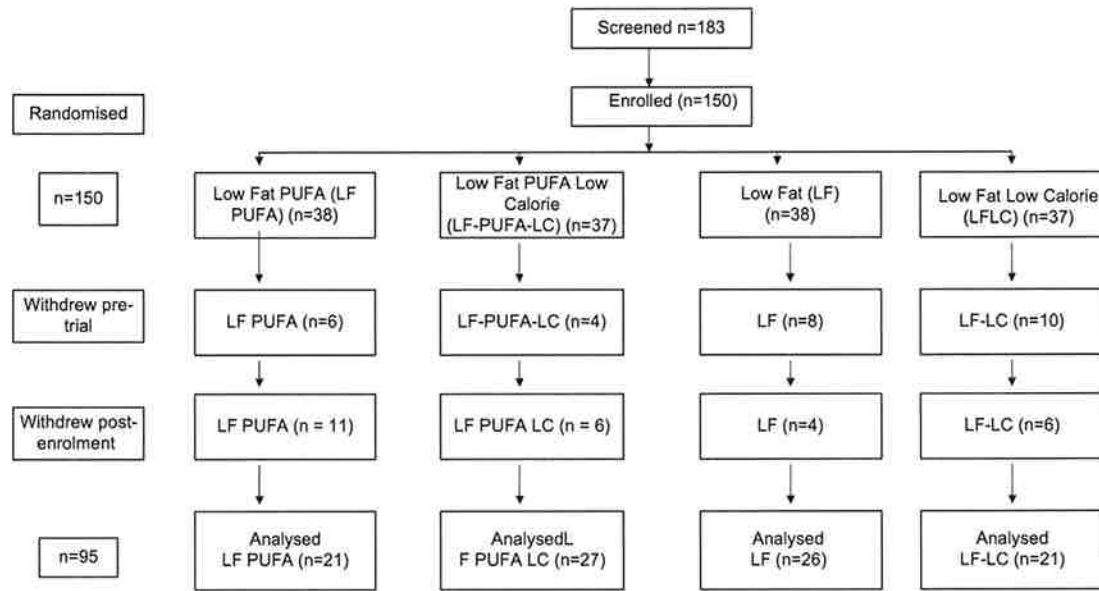


Figure 1 Flow chart of the participants during the trial.

dietary advice groups (Fig. 1). Withdrawals resulted from delays in the commencement of the diet. By the end of the study similar numbers were lost in each group, with reasons of lack of time, work and family commitments, family health issues, overseas travel, and moving out of the area. At baseline there were no significant differences between groups in demographic, anthropometric, clinical or dietary intakes, with one exception. The LF-PUFA group reported higher carbohydrate and lower total fat and PUFA intakes compared to the other groups ($P = 0.009$, $P = 0.044$ and $P = 0.007$ respectively) (Table 1). A comparison of data from completers and non-completers at baseline showed no statistically significant differences in weight ($P = 0.396$), VAT ($P = 0.622$), body fat ($P = 0.138$) or BMI ($P = 0.564$).

Change in body weight and body fat

After 3 months all groups lost weight but the LC groups lost more weight (around 2 kg more) than those receiving isocaloric advice ($P = 0.026$) (Table 2). Post hoc analysis of weight loss in each group showed significant effects in all groups (LF, LF-PUFA, LF-LC, LF-PUFA-LC: $P = 0.000$, 0.001 , 0.000 , 0.000 respectively).

All groups also lost body fat producing a significant time effect ($P < 0.0001$) and while the low calorie groups appeared to lose more fat, there was a high degree of variation and the difference between groups was not significant. All groups showed reductions in visceral adipose tissue (VAT L4) (significant time effect, $P = 0.000$), but the variation was very high. This was the case particularly for the LF-LC group ($-38.42 \pm 64.51 \text{ cm}^2$), which produced a non-significant result. There was a tendency toward a diet effect for changes in subcutaneous adipose tissue (SAT) ($P = 0.070$), with the LC groups showing a greater loss, and a significant time effect for all groups ($P = 0.001$). In keeping with the observed reductions in body fat, leptin levels dropped in all groups with a significant intervention

effect ($P = 0.001$) but there was no difference between groups. As verification of the results an intention to treat (ITT) analysis was conducted on the primary outcome measures (weight and VAT) using two different methods, a linear mixed model and a last observation carried forward (LOCF) approach using the repeated measures analysis of variance (RMANOVA) under the general linear model (GLM). The results remained substantially unchanged with the interaction term for weight and VAT (0.026 and 0.352 respectively, completers only) changing only slightly (0.023 and 0.418 LMM respectively, and 0.068 and 0.510 LOCF respectively).

Changes in lipids, insulin and glucose

Bearing in mind that weight loss was achieved throughout the study sample, all groups showed a significant reduction in total cholesterol levels ($P < 0.0001$ time effect, $P = 0.017$, intervention effect). There was a significant diet effect for increased HDL levels ($P = 0.042$) and a borderline significant diet effect for reductions in TG levels ($P = 0.056$).

Post hoc analysis revealed there were no significant changes to HDL and TG levels in the LF and LF-PUFA-LC groups (LF: HDL, $P = 0.693$; TG, $P = 0.502$. LF-PUFA-LC: HDL, $P = 0.953$; TG, $P = 0.084$), although there was a tendency to reduced TG in the LF-PUFA-LC group ($P = 0.084$). The LF-PUFA group produced reductions in TG ($P = 0.041$), and a tendency for increased HDL ($P = 0.061$). The LF-LC group produced both reduced TG ($P = 0.032$) and increased HDL ($P = 0.012$).

There were no significant effects on fasting glucose, but there was a significant time effect for insulin ($P = 0.040$) (Table 2).

Changes in energy deficit

Since there was no intervention effect on weight loss we collapsed the four diet groups into two to compare the

Table 1 Baseline characteristics of study participants.

Variable	LF (n = 30)	LF-PUFA (n = 32)	LF-LC (n = 27)	LF-PUFA-LC (n = 33)	P value
Age (years)	43.8 ± 12.1	45.0 ± 8.3	46.1 ± 10.4	42.8 ± 11.8	0.662
Weight (kg)	89.8 ± 14.5	86.4 ± 13.0	87.2 ± 15.4	88.5 ± 14.6	0.807
BMI (kg/m ²)	32.1 ± 4.1	30.5 ± 3.4	30.7 ± 3.8	30.9 ± 3.7	0.395
Visceral fat (cm ²)	118 ± 78	124 ± 76	134 ± 92	114 ± 73	0.820
Subcutaneous fat (cm ²)	344 ± 124	348 ± 74	323 ± 96	340 ± 100	0.828
Body fat (%)	40.3 ± 6.6	38.0 ± 5.8	38.6 ± 6.5	38.0 ± 6.4	0.441
Waist (cm)	95 ± 11	92 ± 10	95 ± 13	93 ± 11	0.739
Hip (cm)	114 ± 9.4	111 ± 7.6	110 ± 7.5	110 ± 8.5	0.299
W:H ratio	0.84 ± 0.09	0.83 ± 0.09	0.84 ± 0.10	0.84 ± 0.09	0.985
Umbilicus (cm)	104 ± 12	98 ± 6.5	102 ± 11	98 ± 12	0.192
Iliac crest (cm)	106 ± 11	102 ± 10	104 ± 10	102 ± 11	0.312
Fasting blood glucose (mmol/L)	5.4 ± 0.4	5.4 ± 0.5	5.6 ± 0.7	5.5 ± 0.6	0.361
Fasting insulin (mU/L)	11.3 ± 5.2	11.2 ± 3.7	11.6 ± 9.7	9.8 ± 4.7	0.715
Cholesterol (mmol/L)	5.3 ± 0.9	5.8 ± 1.4	5.5 ± 0.91	5.1 ± 1.3	0.171
LDL (mmol/L)	3.1 ± 0.9	3.3 ± 1.2	3.3 ± 0.9	3.1 ± 1.0	0.829
HDL (mmol/L)	1.5 ± 0.4	1.5 ± 0.3	1.4 ± 0.4	1.6 ± 0.4	0.362
TG (mmol/L)	1.6 ± 0.9	1.9 ± 1.7	1.7 ± 1.1	1.3 ± 0.5	0.215
Baecke PA questionnaire	7.4 ± 1.1	7.6 ± 1.2	7.1 ± 1.3	7.2 ± 1.5	0.449
Energy intake (kJ)	9380 ± 3800	9160 ± 3140	8870 ± 2310	10130 ± 3270	0.459
Carbohydrate intake (%E)	41.9 ± 7.0	47.0 ± 8.1	41.2 ± 6.5	42.9 ± 6.7	0.009*
Protein intake (%E)	20.1 ± 3.6	19.0 ± 3.4	20.2 ± 3.9	19.9 ± 3.6	0.548
Fat intake (%E)	32.9 ± 4.9	29.0 ± 5.3	30.8 ± 6.1	31.8 ± 5.7	0.044*
Saturated fat intake (%E)	11.4 ± 2.2	11.0 ± 2.8	11.1 ± 3.2	11.7 ± 2.5	0.717
Polysaturated fat intake (%E)	5.8 ± 2.3	4.3 ± 1.3	5.0 ± 1.7	4.9 ± 1.3	0.007*
Monounsaturated fat intake (%E)	12.6 ± 2.7	10.9 ± 2.8	11.8 ± 2.3	12.3 ± 3.1	0.064
P:S ratio of intake	0.53 ± 0.3	0.43 ± 0.2	0.48 ± 0.2	0.44 ± 0.2	0.200

Data is given as mean ± standard deviation.

*Significant difference between groups using the one-way ANOVA analysis.

effects on weight loss and energy deficit from caloric restriction or from higher dietary PUFA. This meant comparing LC (LF-LC and LF-PUFA-LC) vs. IC (LF and LF-PUFA) groups and comparing PUFA (LF-PUFA and LF-PUFA-LC) vs. LF (LF and LF-LC) groups. The pattern of weight loss was the same in the calorimeter sub-sample ($n = 48$), as for the full sample ($n = 95$). In the calorimeter sub-sample the two LC groups also lost significantly more weight ($P = 0.002$), compared to the two IC groups. When reductions in energy expenditure after weight loss were taken into account, the calculated energy deficit was not significantly different between the LC and IC groups or between the LF and PUFA groups (Fig. 2). This suggested that, in our hands, energy restriction and not PUFA intake had a measurable effect on weight loss.

Dietary change

All groups reported reduced energy intakes (~2.1 MJ for LF groups, 1.68–2.52 MJ for PUFA groups). Changes in macronutrient intake profiles were similar for the two LF groups and for the two PUFA groups respectively. To ascertain if these similarities were significantly different between the LF and PUFA categories, the dietary data from the four diet groups were collapsed into two groups (LF and PUFA) (Fig. 3). Significant differences between groups occurred for changes in dietary macronutrient proportions for total

fat, SFA and PUFA only. The LF groups reduced total dietary fat significantly more than the PUFA groups ($P = 0.043$) and the PUFA groups reduced dietary SFA and increased dietary PUFA significantly more than the LF groups ($P = 0.025$ and $P < 0.0001$ respectively). This was confirmed through biomarker data with a significant diet effect for changes in erythrocyte PUFA ($P = 0.045$). The LF groups produced reduced levels (-2.11 ± 5.46) and the PUFA groups increased levels (0.16 ± 4.16), commensurate with reported changes in dietary intake (see Fig. 2). The PUFA groups showed positive correlations between reported PUFA intake and erythrocyte PUFA levels ($r = 0.18$), and the LF groups showed negative correlations ($r = -0.19$). Both values were non-significant ($P > 0.05$). Bearing in mind that the LF-PUFA group reported relatively lower intakes of PUFA at baseline ($P = 0.007$), this group showed the greatest change, and there was a trend for a significance diet effect between the four groups ($P = 0.075$).

Discussion

Low-fat (LF) diets for weight loss are advocated by many scientific and governmental agencies [23], but whether low fat is a preferable approach to energy restriction is under debate [24]. We found that energy restriction produced a greater weight loss than LF dietary advice alone ($P = 0.026$). We also found that increasing the

Table 2 Changes in anthropometric and clinical variables in completers after 3 months.

Parameter	LF (n = 26)	LF-PUFA (n = 21)	LF-LC (n = 21)	LF-PUFA-LC (n = 27)	Time effect ^a	Intervention effect ^b	Diet effect ^c
Weight (kg)	-2.6 ± -2.7	-2.6 ± -3.2	-4.8 ± -4.2	-4.7 ± -3.3	0.000	0.795	0.026 (0.030)
Body fat (%)	-1.8 ± -2.4	-1.3 ± -2.2	-3.2 ± -4.0	-2.2 ± -2.6	0.000	0.185	0.164 (0.202)
WHR (cm)	0.0 ± -0.05	0.02 ± -0.04	-0.0 ± -0.03	-0.0 ± -0.04	0.414	0.459	0.388 (0.373)
VAT L4 (cm ²)	-20.2 ± -43	-11.6 ± -38	-38.4 ± -65	-18.4 ± -38	0.000	0.680	0.352 (0.441)
SAT L4 (cm ²)	-14.0 ± -53	0.09 ± -68	-27.8 ± -56	-42.1 ± -4	0.001	0.138	0.070 (0.058)
VAT:SAT	-0.07 ± -0.2	-0.06 ± -0.2	-0.11 ± -0.2	-0.04 ± -0.2	0.002	0.483	0.685 (0.675)
Anterior density (dpi/mm ²)	-9.8 ± -49	-0.00 ± -4.8	2.3 ± -9.7	3.1 ± -4.8	0.687	0.193	0.322 (0.339)
Posterior density (dpi/mm ²)	1.1 ± -5.0	-0.9 ± -5.3	0.5 ± -7.7	0.3 ± -4.8	0.680	0.237	0.738 (0.745)
Visceral density (dpi/mm ²)	2.6 ± -10.5	2.3 ± -8.4	0.6 ± -8.8	1.8 ± -11.6	0.109	0.386	0.932 (0.936)
Total cholesterol (mmol/L)	-0.25 ± -0.3	-0.50 ± -1.2	-0.27 ± -0.5	-0.30 ± -1.0	0.000	0.017	0.716 (0.711)
Triacylglycerol (mmol/L)	0.09 ± -0.7	-0.47 ± -1.0	-0.26 ± -0.5	-0.16 ± -0.4	0.008	0.091	0.056 (0.058)
Fasting Insulin (mU/L)	-1.8 ± -2.2	0.4 ± -5.5	-1.1 ± -4.1	-1.2 ± -4.3	0.040	0.241	0.326 (0.264)
Fasting glucose (mmol/L)	0.1 ± -0.4	0.06 ± -0.5	-0.04 ± -0.5	0.05 ± -0.6	0.355	0.697	0.785 (0.878)
HDL-cholesterol (mmol/L)	0.02 ± -0.2	0.05 ± -0.1	0.16 ± -0.3	0.00 ± -0.2	0.007	0.744	0.042 (0.069)
LDL-cholesterol (mmol/L)	-0.27 ± -0.4	-0.24 ± -0.6	-0.32 ± -0.4	-0.44 ± -0.7	0.000	0.081	0.634 (0.533)
Leptin (ng/mL)	-3.0 ± -13.3	-2.9 ± -5.2	-4.7 ± -2.1	-4.9 ± -6.6	0.128	0.001	0.857 (0.760)

Data given as difference in means (baseline-3 months) ± standard deviation differences. Repeated measures analysis of variance (ANOVA); significant at $P < 0.05$. Data refers to trial completers. WHR, waist-hip-ratio; VAT, visceral adipose tissue; SAT, subcutaneous adipose tissue.

^a Time effect = changes over time.

^b Intervention effect = effect of different diets within group.

^c Diet effect = effect of different diets over time between groups (interaction effect) (adjusted for sex).

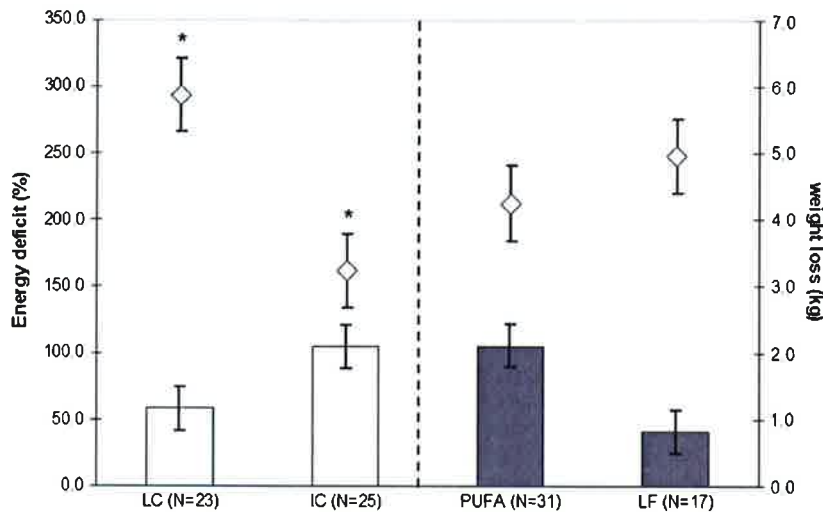


Figure 2 Differences in energy deficit required for weight loss. The energy deficit (%) indicates the extent to which the dietary energy deficit is responsible for weight loss. The histograms show the differences in energy deficit (% scale on the left side) and the diamonds illustrate the differences in weight loss (kg, scale on the right side). Two separate comparisons are made (divided by dotted line). These are: (1) between low calorie (LC) vs. isocaloric (IC) groups; and (2) between higher polyunsaturated fat (PUFA) vs. lower polyunsaturated fat (LF) groups. LC, low calorie groups (LF + LF-PUFA-LC); IC, isocaloric groups (LF + LF-PUFA); PUFA, high polyunsaturated fat groups (LF-PUFA + LF-LC-PUFA); LF, lower polyunsaturated fat groups (LF + LF-LC); energy deficit = $[\Delta EI - \Delta EE] / \Delta EI \times 100$, where: ΔEI = reduction in energy intake from baseline to 3 months; ΔEE = reduction in energy expenditure from baseline to 3 months; \diamond = weight loss (kg). *Significant difference in weight loss between groups, independent t-test, $P = 0.002$.

proportion of dietary PUFA had no distinctive effects on weight loss compared to a more general LF approach. A loss of body fat with concomitant reductions in leptin levels was observed in the study sample, but unlike a similar study using MRI scanning [15], we did not find a diet effect for changes in visceral fat. The wide variation in our data may reflect individual gene–nutrient interactions and the consequent regulation of metabolic

pathways [25]. It is also possible that the inclusion of both men and women in the study may have contributed to a wide variation in data.

With all groups losing weight, the reductions in cholesterol levels were as expected. The dyslipidemic profile of high triacylglycerol levels and low HDL was of particular interest as disease risk factors for diabetes and cardiovascular disease. Our observed PUFA effect on lowering triacylglycerol levels was consistent with the literature [26], but LC and LF advice was also effective. Other short term studies of weight loss have shown the type of diet is less of an issue than adherence to energy deficits [24,27]. Nevertheless, given that weight loss is difficult to maintain, the observed triglyceride lowering effect of dietary fat modification suggests this strategy has added benefits.

We did not find that manipulating dietary fat had any discernable effect on the energy deficit required to lose weight over time (Fig. 2). This was of interest because the energy deficit is linked to fat mass [14], which in turn is affected by level of fat oxidation. Animal model studies have found that increasing PUFA intakes results in decreased accumulation of body fat, suggesting higher dietary induced thermogenesis and fat oxidation [28,29]. Experimental human studies using indirect calorimetry have supported this, demonstrating a higher fat oxidation, dietary induced thermogenesis and resting metabolic rate after consumption of a diet with a high compared to a low P:S ratio [30,31]. Studies of labelled breath tests have shown that dietary polyunsaturated and monounsaturated fats are oxidized more rapidly than saturated fats [32]. Despite this, under free living

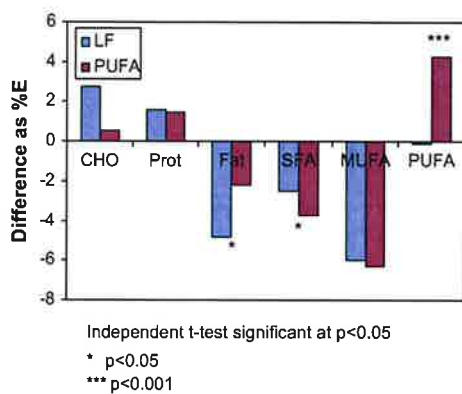


Figure 3 The difference in dietary nutrients expressed as %E from baseline to 3 months, in the low fat (LF) groups (low fat isocaloric + low fat–low calorie) compared to the PUFA groups (low fat isocaloric–PUFA + low fat–low calorie–PUFA). PUFA, polyunsaturated fatty acids; MUFA, monounsaturated fatty acids; SFA, saturated fatty acids; CHO, carbohydrate; Prot, protein. Independent t-test significant at $p < 0.05$. * $p < 0.05$, *** $p < 0.001$.

conditions, we failed to expose metabolic adaptations associated with weight loss [10] and adaptive responses to weight gain.

All studies are limited by their sample population, but there is value in their key features. We achieved a high completion rate (73%) and the weight loss results were similar in the calorimeter sub-sample as they were for the full study sample. The high PUFA groups were given additional advice on PUFA rich choices such as extra fish, walnuts, and oils, with reductions in other foods. This enabled a significant difference between groups for caloric intake and type of dietary fat, but while we were able to control for dietary carbohydrate and protein, both of which may have acted as confounders, the LF groups ended up consuming significantly less total fat within the dietary macronutrient profile (Fig. 3). This difference in dietary fat may have masked the effect of increased PUFA. Further research with higher fat and energy intakes may be required. Finally, working between foods and nutrients is one of the fundamental problems in nutrition research as it moves to the clinical setting [33]. Working with macronutrient proportions has the benefit of strong mechanistic underpinnings, but this has to be translated in a context where people freely chose to eat foods. Further research that integrates energy balance and disease risk theory with multiple dietary components that more readily reflects food choice patterns might also lead to more informative results for practice.

Conclusion

Caloric restriction produced the most potent effect on weight loss and lipids, but we confirmed a triacylglycerol lowering effect of increased dietary polyunsaturated fat.

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