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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 *Background and aims:* Decreasing energy intake relative to energy expenditure is the indisputable tenet of weight loss. In addition to caloric restriction modification of the type of dietary fat may provide further benefits. The aim of the present study was to examine the effect of energy restriction alone and with dietary fat modification on weight loss and adiposity, as well as on risk factors for obesity related disease.
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.

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

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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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% monounsaturated 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