ARTICLE

Monounsaturated fat-rich diet reduces body adiposity in women with obesity, but does not influence energy expenditure and substrate oxidation: a parallel randomized controlled clinical trial

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BACKGROUND: Obesity is an important and growing health problem whose treatment involves dietary changes. In this context, studying the role of macronutrients in weight loss is required in order to understand which strategies may be applied for weight loss. We aimed to evaluate the effects of diets rich in polyunsaturated (PUFAs) and monounsaturated fatty acids (MUFAs) on resting energy expenditure (REE), substrate oxidation, and weight loss in women with obesity.
METHODS: Randomized, controlled, single blind, parallel-group clinical trial was conducted for 60 days. Participants ($n = 32$) were divided into three groups: G1= normocaloric PUFAs-rich diet (12% of total energy expenditure (TEE), 10% of n-6 and up to 2% of n-3); G2= normocaloric MUFAs-rich diet (15–20% TEE); and G3= maintenance of the usual diet. Anthropometric and metabolic variables (REE and substrate oxidation by indirect calorimetry) were evaluated.
RESULTS: G2 decreased body weight (-1.92 ± 1.99 kg, $P = 0.02$), body mass index (BMI) (-0.69 ± 0.70 kg/m^2; $P = 0.02$), waist circumference (WC) (-1.91 ± 1.82 cm; $P = 0.02$), and body fat (-1.14 ± 1.53 kg; $P = 0.04$).
CONCLUSION: MUFAs-rich diet reduces body weight, BMI, body fat, and WC. Clinical Trials: NCT02656940.
CLINICAL TRIAL REGISTRATION: Clinical Trials: NCT02656940.

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INTRODUCTION

In the last decades, studies have searched for nutritional strategies to optimize body weight loss [1]. Some authors believe that decreasing energy is more relevant than managing the distribution of macronutrients [2, 3]. However, studies have emphasized the impact of changing the amount of protein, carbohydrate or fat in the treatment of obesity [4, 5].

Manipulation of dietary fats has been suggested as an option to control the obesity epidemic. Studies showed fatty acids may affect the balance between intake and energy expenditure (EE) through different factors related to adipogenesis, such as resting energy expenditure (REE), substrate oxidation [6], diet-induced thermogenesis (DIT) [7], and satiety [8], reducing body mass and/or fat storage in adipose tissue [6]. However, these effects have not been fully elucidated in humans and the results are still controversial.

The benefits of high-unsaturated fats diets, such as Mediterranean diet, have been explored in the last years. However, there are few studies evaluating the isolated effect of high-monounsaturated fatty acids (MUFAs) or high-polyunsaturated fatty acids (PUFAs) diets on health and weight loss [9, 10].

A systematic review showed weight loss after high-MUFAs diet, in which some studies evaluated the acute effects of high-MUFAs meals and others studies verified the effects of high-MUFAs diets in varying intervention periods [11]. On the other hand, some studies investigated the effects of PUFAs, especially n-3 PUFAs, in the treatment of obesity [12, 13]. However, most studies used n-3 PUFAs supplementation, and few studies had control diet for n-3 and n-6 PUFAs. In this context, our study aimed to evaluate the effects of high n-3 and n-6 PUFAs or high-MUFAs diets on REE, substrate oxidation, and body weight loss in obese women without comorbidities.

SUBJECTS AND METHODS

Study subjects

The study was performed at the Laboratory of Nutritional Assessment (LANUTRI) of the Institute of Nutrition Josué de Castro of the Federal University of Rio de Janeiro. Volunteers were recruited from March 2013 to

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Table 1. Prescribed diet composition for the test groups (G1 and G2).

	G1 ($n = 13$)	G2 ($n = 13$)	*P*-value[a]
Energy (kcal)	2588.6 ± 393.3	2593.1 ± 384.3	0.98
Carbohydrates (%)	51.4 ± 0.2	51.4 ± 0.3	0.60
Carbohydrates (g)	332.8 ± 50.0	333.2 ± 49.8	0.98
Proteins (%)	15.9 ± 0.2	15.8 ± 0.2	0.45
Proteins (g)	102.7 ± 16.2	102.5 ± 15.7	0.97
Lipids (%)	32.7 ± 0.1	32.8 ± 0.2	0.16
Lipids (g)	94.1 ± 14.3	94.5 ± 13.7	0.94
SFAs (%)	6.9 ± 0.1	7.1 ± 0.1	<0.01[b*]
SFAs (g)	19.8 ± 3.2	20.5 ± 3.3	0.59
MUFAs (%)	11.9 ± 0.2	17.5 ± 0.4	<0.01[*]
MUFAs (g)	34.4 ± 5.1	50.2 ± 7.0	<0.01[*]
PUFAs (%)	12.2 ± 0.2	6.8 ± 0.2	<0.01[*]
PUFAs (g)	35.1 ± 5.4	19.5 ± 2.8	<0.01[*]
n-6PUFAs (%)	10.6 ± 0.2	6.0 ± 0.2	<0.01[*]
n-6PUFAs (g)	30.4 ± 4.9	17.4 ± 2.5	<0.01[*]
n-3 PUFAs (%)	1.6 ± 0.08	0.8 ± 0.0	<0.01[*]
n-3 PUFAs (g)	4.7 ± 0.5	2.2 ± 0.3	<0.01[*]
n-6/n-3ratio	6.5 ± 0.4	7.9 ± 0.3	<0.01[*]
Total fiber (g)	33.9 ± 3.8	32.6 ± 4.9	0.45

Values were represented as mean ± SD (for all values).
G1 n-6/n-3 PUFAs-rich diet, *G2* MUFAs-rich diet, *SFAs* saturated fatty acids, *MUFAs* monounsaturated fatty acids, *PUFAs* polyunsaturated fatty acids.
[a]Differences between groups were analyzed using the Mann–Whitney U test.
[b]Despite the similarity, the groups differed.
[*]$P < 0.05$.

June 2014. Inclusion criteria were as follows: (1) adult women (aged between 20–40 years); (2) any ethnic; (3) obesity classes I and II (body mass index (BMI) between 30 and 39.9 kg/m^2); (4) pre-menopause; (5) without weight loss greater than 3 kg in 3 months; (6) and elementary school complete.

The exclusion criteria were: (1) women previously diagnosed with diabetes mellitus, cardiovascular, kidney, liver or autoimmune diseases, thyroid disorder, inflammatory bowel diseases, acquired immunodeficiency syndrome, or cancer; cholecystectomy and other surgeries (last 12 months); (2) pregnant or breastfeeding; (3) smoking; (4) using lipid-lowering, hypoglycemic, diuretics, antidepressants, or antihypertensive drugs, supplements, herbal products, and/or diets for weight loss in the last 4 weeks; (5) and those with food allergy or intolerance to vegetable oils (olive, soy, or canola oil), fish oil, fish, and/or seafood.

The research was approved by the Research Ethics Committee of the University Hospital Clementino Fraga Filho (Rio de Janeiro, RJ, Brazil) (protocol n.072/10) according to the declaration of Helsinki. All participants provided written informed consent. In addition, the present study was registered at ClinicalTrial.gov (NCT02656940).

Experimental design and characteristics of dietary intervention

A parallel, single blind, randomized, controlled clinical trial was conducted for 60 days with a convenience sample. Randomization was determined in advance, through a list of allocation groups, before the volunteer recruitment steep. Based on the previously established list, the women were continuously assigned to one of three groups: G1 ($N = 10$) - normocaloric n-3 and n-6 PUFAs-rich diet; G2 ($N = 11$) - normocaloric MUFAs-rich diet; or G3 ($N = 11$) - placebo and their usual diet.

The energy value of prescribed diets (G1 and G2) was calculated based on the REE obtained by indirect calorimetry (Vmax 29®, Viasys Healthcare, USA), using Weir equation [14] and corresponding physical activity factor [15]. The diets were previously calculated using the diet analysis program Diet Pro® 5i (Federal University of Viçosa) based to the Brazilian Table of Food Composition [16].

The G1 and G2 diets were adequate in carbohydrates (50–55% of total energy expenditure (TEE)), proteins (15–20% of TEE), and fats (30–35% of TEE) [17]. The G1 diet contained 12% of TEE of PUFAs (10% of n-6 PUFAs and up to 2% of n-3 PUFAs), and G2 17% of TEE of MUFAs [18]. Both groups were oriented to consume up to 10% saturated fatty acids (SFA) [18]. The average chemical composition of the prescribed diets is described in Table 1.

Sachets containing soy oil and extra virgin olive for G1, sachet with extra virgin olive oil for G2 (amounts according to the prescribed diet) and soy oil (2 g/day) as a placebo for G3 were offered to achieve the desired amounts of dietary lipids. The volunteers were instructed to add the individualized portions of oils directly to the dish (lunch and dinner), no cooking the oil. Regarding the type of oil used to prepare food, G1 and G2 were oriented to use soybean oil and canola oil to cook, respectively, while G3 was instructed to maintain usual oil.

G1 received fish oil capsules (Vital Fish®, Vital Âtman Ltda, SP, Brazil - 2 capsules/ day containing 420 mg of eicosapentaenoic acid [EPA], 220 mg of docosahexaenoic acid [DHA] and 5 mg of vitamin E each) to achieve n-3 PUFAs intake. G2 and G3 received capsule containing 1 g of soybean oil per day (Officilab®, RJ, Brazil). The volunteers were instructed to maintain their usual physical activities during the study, and the level of physical activity was classified as sedentary or light [17].

Adherence to the dietary intervention was assessed by 3-day food record and conference of leftover sachets and capsules, and plasma fatty acids composition analysis was evaluated by gas chromatography (Agilent Technologies, model 7890 A CG System) [19]. Fatty acid composition analysis of the oils was conducted by gas chromatography [20]. For both chromatographic analyses, the internal standard C13:0 (Sigma Aldrich) at 5 mg/mL was used.

For quality control of the oils used during the dietary intervention, the physical-chemical parameters (acidity and peroxide indexes) were monitored per lot (oils and capsules). The extra virgin olive oil was controlled monthly, and the open packages of soybean oil and olive oil were monitored each 15 days for 45 days. The analysis was performed according to the methods proposed by the Institute Adolfo Lutz [21] and the results were evaluated according to the current legislation [22].

Dietary intake assessment

Dietary intake was assessed by 3-day food record (2 typical days and 1 atypical) before and during the intervention period.

The records were analyzed using the Diet Pro®5.5i software (Federal University of Viçosa) and Brazilian Table of Food Composition [16]. Tables of United States Department of Agriculture [23], University of São Paulo [24] and Philippi [25] were used in order of priority when food were not included in Brazilian Table of Food Composition.

The oil supplements (sachets and capsules) were considered in the prescribed diet calculation and in the dietary intake assessment. The chemical composition of the oil supplements was recorded in the diet analysis program based on the results obtained via gas chromatography, and oils used in the preparation of meals were considered in the analysis, as reported in the diet records.

Assessment of energy expenditure and substrate oxidation

The preparation for the EE assessment started 3 days previously. During this period, volunteers were instructed to maintain only their daily activities, avoiding any physical exercises. In addition, the volunteers were instructed not to drink alcoholic beverages, and to avoid excessive intake of foods high in fats, proteins and caffeine.

The evaluation was performed at LANUTRI, using an indirect calorimetry system with a respiratory chamber (Vmax Encore 29 Systems®, Viasys Healthcare, USA). The measurements were performed after a 12-h overnight fast. The volumes of oxygen consumption (VO_2) and carbon dioxide produced (VCO_2) were measured for 30 min, with the initial 5 min being disregarded. The appropriated equilibrium state was considered when the coefficient of variation for VO_2 and VCO_2 measurements was up to 10% in five consecutive minutes [26].

The ratio between VCO_2 and VO_2 (L/min) was used to calculate the respiratory quotient (RQ). The values used for interpreting the RQ were as follows: 0.72 for lipids, 0.80 for proteins, and 1.0 for carbohydrates [14]. The equation described by Weir [14] was used to determine REE: $([3.9 \times VO_2\ L/min] + [1.1 \times VCO_2\ L/min]) \times 1440$. To determine the TEE, the REE value was multiplied by the corresponding physical activity factor [15].

The equations described by Jéquier, Acheson, and Schutz [27] were used to calculate the substrate oxidation: lipids $= (0.75 \times NPVO_2)/2.019$ (g/min); carbohydrate $= (0.25 \times NPVO_2)/0.829$ (g/min); and proteins $= PVO_2/0.966$ (g/min). Since $NPVO_2$ = non-protein oxygen volume in L/min; PVO_2 = protein oxygen volume in L/min; in which PVO_2 (L/min) = nitrogen × 6.25 × 0.966. The $NPVO_2$ value (L/min) was obtained by the difference between VO_2 (L/min) and PVO_2 (L/min). For the calculation of PVO_2, the nitrogen excretion constant of 0.14 g/kg/day was used instead of the urinary nitrogen [28].

Table 2. Acidity and peroxide index of oils used.

Oil	Acidity[a]	Peroxide index (meq/kg)
Extra virgin olive oil (Single lot)	0.01	8.41
Soy oil capsules (Single lot)	0.53	1.99
Fish oil capsule (Single lot)	2.77	2.59
Soyoil (Lot 1)	0.44	1.74
Soyoil (Lot 2)	0.20	1.64
Soyoil (Lot 3)	0.28	1.48

KOH potassium hydroxide.
[a]mg KOH/g for soybean oil, soybean oil capsules and fish oil capsules; g/100 g in oleic acid for extra-virgin olive oil.

Anthropometric and body composition assessments

Anthropometric and body composition assessments were performed in fasting. Total body mass and height were measured using the Filizola® electronic platform scale Personal Line (0.1 kg accuracy and 150 kg maximum capacity) and Alturexata® portable vertical stadiometer (1 mm accuracy and 213 cm maximum capacity), respectively. Measures were performed with light clothing and no footwear. BMI was calculated [29]. The waist circumference (WC) was measured at the midpoint between the last rib and the iliac crest [29], using an inelastic anthropometric tape 2.0 meters long, Sanny® brand with a scale in cm.

Body composition was evaluated by single-frequency electric bioimpedance analysis (Biodynamics® model 450), the fat free mass (FFM) was calculated by the equations of Segal et al. [30], following protocol.

Statistical analysis

Results were expressed as mean and standard deviation (SD). The Kolmogorov–Smirnov test was performed, and the data had a non-normal distribution. Non-parametric tests were used to analyze the data [31]. Statistical analyzes were conducted in SPSS 20.0 program (SPSS, IBM Corporation, NY).

Comparisons between three groups were evaluated by Kruskal-Wallis test. For the variable that presented $P < 0.05$ post hoc Mann–Whitney U was conducted for comparisons between groups (G1 × G2, G1 × G3, and G2 × G3), followed by adjustment for type I error [32]. Variations between groups were similar. Differences between times in group (from baseline to final value) were evaluated using the Wilcoxon signed-rank test, with a 5% probability.

RESULTS

Quality of the oils

Acidity and peroxide index analysis of the oils used during the dietary intervention showed satisfactory results in all oils lots used (Table 2). The monthly control of extra-virgin olive oil supplied in gallons of 5 liters (Fig. 1) and biweekly monitoring of open packages of soybean oil and olive oil for a period of up to 45 days (Fig. 2) showed suitable values of acidity and peroxide index.

Volunteers and adherence to proposed dietary intervention

Of the 252 volunteers who showed interest in participating of the study, 34 were included and 32 had their data analyzed. The exclusion reasons were no adherence (< 70%) to the dietary guidelines and insufficient intake of oil supplements (sachets) (Fig. 3).

Dietary records confirmed differences in SFAs, MUFAs and PUFAs intake between groups. G1 ingested higher total PUFAs,

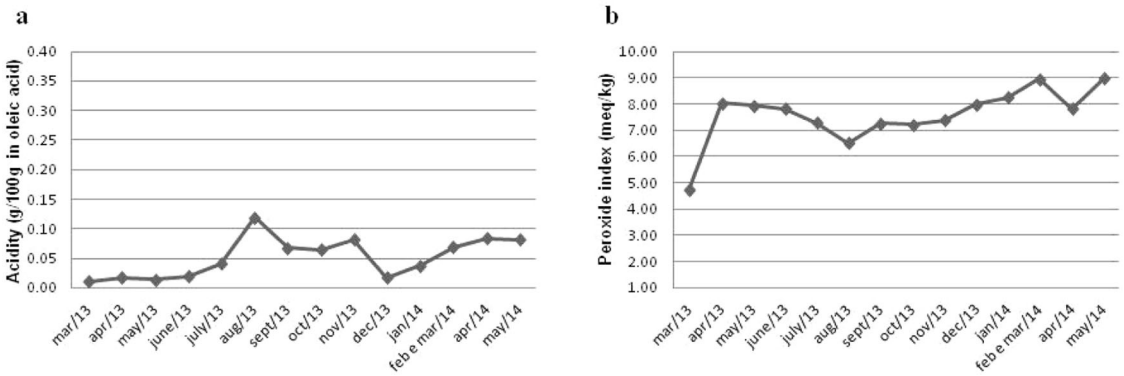

Fig. 1 Assessment of acidity and peroxide index of extra-virgin olive oil. Recommended values: acidity—up to 0.8 g/100 g in oleic acid; peroxide index—up to 20 meq/kg [22]. **a** Acidity of extra-virgin olive oil; **b** peroxide index of extra-virgin olive oil.

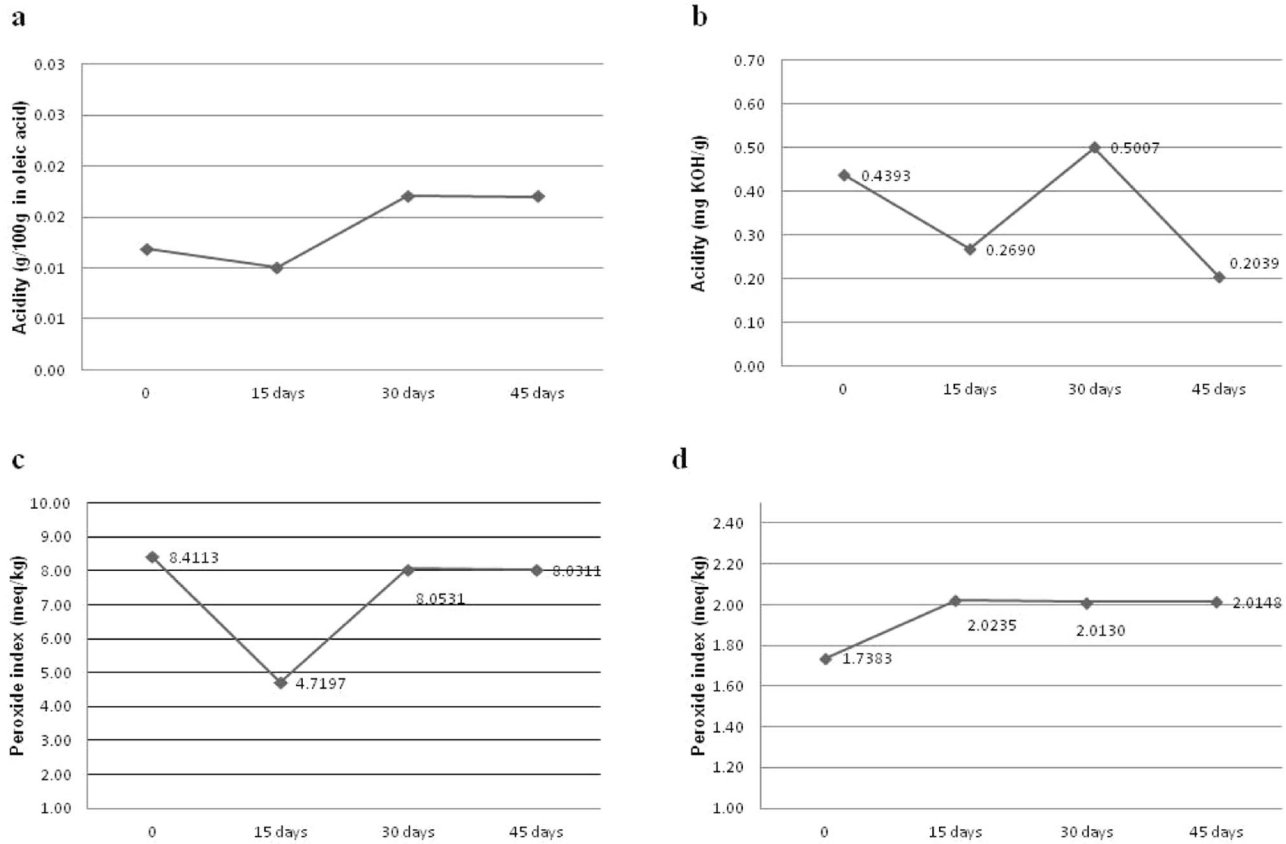

Fig. 2 Assessment of acidity and peroxide index of extra-virgin olive and soybean oil. a Acidity of extra-virgin olive oil; **b** acidity of soybean oil; **c** peroxide index of extra-virgin olive oil; **d** peroxide index soybean oil. Recommended values: olive oil acidity, up to 0.8 g/100 g in oleic acid; soybean oil acidity, up to 0.6 mg KOH/g; olive oil peroxide index, up to 20 meq/kg; soybean oil peroxide index, up to 10 meq/kg [22].

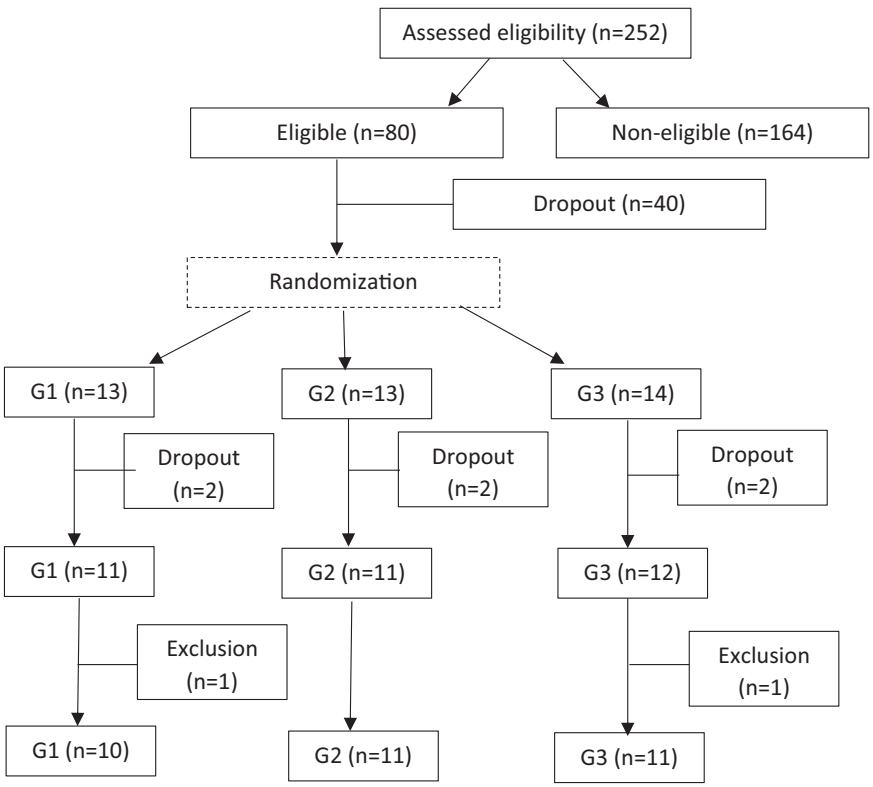

Fig. 3 Representative scheme of recruitment and selection of volunteers. G1 n-6/n-3 PUFAs-rich diet, G2 MUFAs-rich diet, G3 control group.

Table 3. Comparison of dietary intake during the study period.

	G1 ($n = 10$)	G2 ($n = 11$)	G3 ($n = 11$)	*P*-value[a]
Energy (kcal)	2079.65 ± 323.84	2016.29 ± 305.44	2270.90 ± 756.30	0.92
Carbohydrates (g)	226.24 ± 35.00	224.71 ± 48.27	287.37 ± 122.08	0.35
Carbohydrates (%)	43.73 ± 4.34	44.36 ± 4.74	49.91 ± 8.75	0.11
Proteins (g)	97.33 ± 26.72	90.74 ± 13.95	101.72 ± 42.83	0.95
Proteins(%)	18.56 ± 2.89	18.13 ± 2.40	18.04 ± 3.81	0.80
Lipids (g)	88.15 ± 15.73	84.79 ± 13.23	80.65 ± 27.36	0.61
Lipids (%)	38.15 ± 3.54	37.96 ± 3.83	32.43 ± 6.66	0.07
SFAs (g)	20.76 ± 4.63	20.67 ± 3.38	26.39 ± 10.02	0.27
SFAs(%)	8.96 ± 1.35	9.28 ± 1.27	10.63 ± 2.40	0.05
MUFAs (g)	31.74 ± 4.77	44.81 ± 9.46	25.50 ± 11.33	<0.01[b, c*]
MUFAs(%)	13.80 ± 1.27	20.00 ± 3.30	10.26 ± 3.37	<0.01[b, c*]
PUFAs (g)	27.88 ± 4.10	14.00 ± 2.14	15.67 ± 3.22	<0.01[c, d*]
PUFAs(%)	12.11 ± 0.90	6.28 ± 0.70	6.52 ± 1.26	<0.01[c, d*]
n-6 PUFAs (g)	22.69 ± 3.68	12.00 ± 1.89	13.07 ± 2.77	<0.01[c, d*]
n-6PUFAs(%)	9.86 ± 0.93	5.38 ± 0.60	5.42 ± 1.01	<0.01[c, d*]
n-3 PUFAs(g)	4.36 ± 0.47	1.86 ± 0.35	1.33 ± 0.32	<0.01[b, c, d*]
n-3 PUFAs(%)	1.91 ± 0.17	0.83 ± 0.11	0.56 ± 0.13	<0.01[b, c, d*]
EPA + DHA (g)	1.49 ± 0.05	0.06 ± 0.04	0.08 ± 0.08	<0.01[c, d*]
n-6/n-3 ratio	5.20 ± 0.30	6.52 ± 0.70	9.91 ± 1.02	<0.01[b, c, d*]

Values are represented as mean ± SD (for all values).
G1 n-6/n-3 PUFAs-rich diet, *G2* MUFAs-rich diet, *G3* control group, *SFAs* saturated fatty acids, *MUFAs* monounsaturated fatty acids, *PUFAs* polyunsaturated fatty acids, *EPA* eicosapentaenoic acid, *DHA* docosahexaenoic acid.
*$P < 0.05$.
[a]Differences between groups were analyzed using the Kruskal–Wallis test. For the variables that presented $P < 0.05$, the Mann–Whitney U test was used for comparisons between groups (G1 × G2, G1 × G3 and G2 × G3), followed by adjustment for type I error.
[b]G2 differed from G3.
[c]G1 differed from G2.
[d]G1 differed from G3.

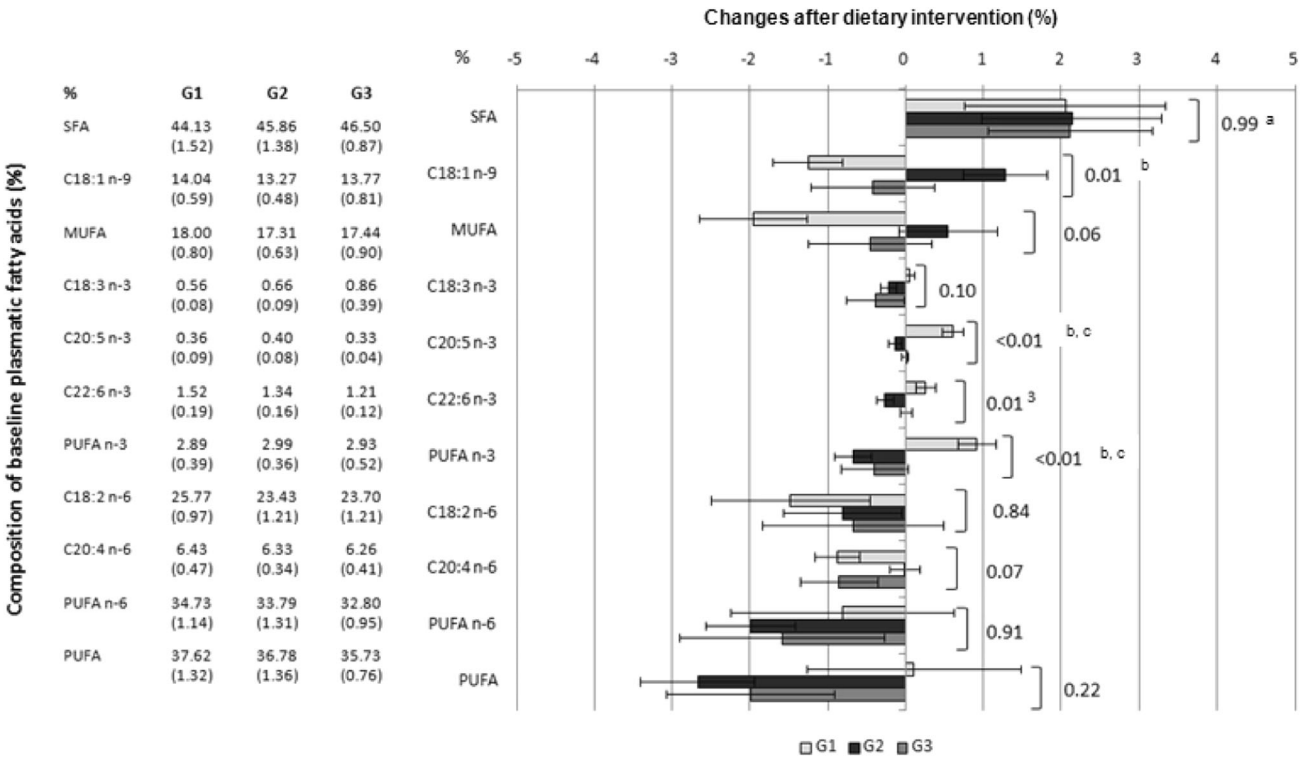

Fig. 4 Composition of plasma fatty acids (%) in baseline and changes after dietary intervention in G1, G2, and G3. Values are represented as mean ± standard error (for all values). [a]Differences between groups analyzed using the Kruskal- Wallis test (for all values). For the variables that presented $P < 0.05$, the Mann–Whitney U test was used for comparisons between groups (G1 × G2, G1 × G3 and G2 × G3), followed by adjustment for type I error. [b]G1 differed from G2. [c]G1 differed from G3. C18:1 n-9, oleic; C18:3 n-3, linolenic; C20:5 n-3, EPA; C22:6 n-3, DHA; C18:2 n-6, linoleic; C20:4 n-6, arachidonic; G1, n-3 and n-6 PUFAs-rich diet ($n = 9$); G2, MUFAs-rich diet ($n = 11$); G3, control group ($n = 10$).

Table 4. Baseline characteristics and comparison of anthropometric variables between groups at the baseline, after dietary intervention, and variations.

	G1 ($n = 10$)[a]	Δ	G2 ($n = 11$)	Δ	G3 ($n = 11$)	Δ	*P*-value[c]
Age (y)	29.70 ± 6.53		31.27 ± 2.69		31.73 ± 3.74		0.70
Weight (kg) I	89.67 ± 6.82	−0.18 ± 1.66	92.35 ± 11.09	−1.92 ± 1.99	90.40 ± 10.89	0.76 ± 0.99	0.96[e]
Weight (kg) II	89.49 ± 6.58		90.43 ± 9.93		91.16 ± 11.17		0.99[f]
P-value[b]	0.72[d]		0.02[d*]		0.05[d]		0.01[g,h*]
BMI (kg/m^2) I	33.94 ± 2.87	−0.08 ± 0.60	33.27 ± 3.24	−0.69 ± 0.70	34.18 ± 3.28	0.29 ± 0.37	0.76[e]
BMI (kg/m^2) II	33.85 ± 2.59		32.58 ± 2.80		34.46 ± 3.42		0.29[f]
P-value[b]	0.59[d]		0.02[d*]		0.04[d*]		<0.01[g,h*]
WC (cm) I	97.69 ± 8.26	−2.09 ± 3.94	94.62 ± 7.52	−1.91 ± 1.82	96.76 ± 9.39	1.22 ± 3.38	0.61[e]
WC (cm) II	95.60 ± 7.88		92.71 ± 7.52		97.98 ± 8.83		0.37[f]
P-value[b]	0.14[d]		0.02[d*]		0.08[d]		0.02[g,h*]
FFM (kg) I	47.93 ± 2.92	0.42 ± 1.59	50.00 ± 4.82	−0.78 ± 1.84	48.96 ± 4.98	0.43 ± 1.25	0.60[e]
FFM (kg) II	48.07 ± 3.59		49.22 ± 4.45		49.39 ± 4.77		0.92[f]
P-value[b]	0.77[d]		0.18[d]		0.29[d]		0.19[g]
FFM (%) I	53.63 ± 3.88	0.51 ± 1.87	54.36 ± 3.37	0.31 ± 1.57	54.32 ± 2.89	0.04 ± 1.07	0.97[e]
FFM (%) II	54.11 ± 3.33		54.68 ± 4.17		54.36 ± 2.43		0.61[f]
P-value[b]	0.95[d]		0.59[d]		0.86[d]		0.87[g]
BFM (kg) I	41.74 ± 6.27	−0.67 ± 2.43	42.35 ± 7.51	−1.14 ± 1.53	41.45 ± 6.82	0.32 ± 0.85	0.99[e]
BFM (kg) II	40.93 ± 5.15		41.21 ± 7.49		41.77 ± 6.93		0.92[f]
P-value[b]	0.59[d]		0.04[d*]		0.21[d]		0.06[g]
BFM (%) I	46.37 ± 3.88	−0.51 + 1.87	45.64 ± 3.37	−0.31 ± 1.57	45.68 ± 2.89	−0.04 ± 1.07	0.97[e]
BFM (%) II	45.89 ± 3.33		45.33 ± 4.17		45.64 ± 2.43		0.61[f]
P-value[b]	0.95[d]		0.59[d]		0.86[d]		0.87[g]

Values are represented as mean ± SD (all values).
Δ delta (after dietary intervention value – baseline value), *G1* n-3 and n-6 PUFAs-rich diet, *G2* MUFAs-rich diet, *G3* control group, *BMI* body mass index, *WC* waist circumference, *FFM* fat-free mass, *BFM* body fat mass, *I* baseline values, *II* after dietary intervention values.
*$P < 0.05$.
[a]$n = 09$ in G1 for the final variables FFM (kg) II, FFM (%) II, BFM (kg) II and BFM (%) II, as well as for the delta values and paired data analysis.
[b]Differences between times, per group, were evaluated using the Wilcoxon signed-rank test, at 5% probability.
[c]Differences between groups were analyzed using the Kruskal–Wallis test. For the variables that presented $P < 0.05$, the Mann–Whitney U test was used for group comparisons (G1 × G2, G1 × G3 and G2 × G3), followed by adjustment for type I error.
[d]Comparison of baseline and after dietary intervention, according to groups.
[e]Comparison of baseline between groups.
[f]Comparison after dietary intervention between groups.
[g]Comparison of Δ between groups.
[h]G2 differed from G3.

and n-6 and n-3 PUFAs than G2. While G2 had higher MUFAs and higher n-6/n-3 ratio intake than G1. In addition, no differences were found between groups for calories, carbohydrates (g and %), proteins (g and %), total lipids (g and %), and SFAs (g and %) (Table 3).

The intervention groups (G1 and G2) showed lower carbohydrates intake (percentage of TEE) compared to baseline (G1: 50.27% + 5.36 to 43.73% + 4.34, p = 0.02; G2: 52.55% + 5.63 to 44.36% + 4.74, p < 0.01) [data not shown].

Capsules and sachets intake were equivalent in three groups (93.92 ± 5.01%; 97.45 ± 4.00%, and 95.32 ± 5.07%; $P = 0.13$; 91.26 ± 6.47%; 93.97 ± 6.17 and 91.70 ± 7.99%; $P = 0.53$, for capsules and sachets in G1, G2 and G3, respectively).

The changes observed in the plasma fatty acids profile at the end of the study confirmed the adherence of the dietary intervention. G1 presented an increase in n-3, EPA and DHA PUFAs, while G2 showed an increase in oleic acid (Fig. 4).

Effect of intervention on anthropometric variables

In the baseline, age, BMI, WC and body fat mass (BFM) were similar between groups. All volunteers presented class I obesity, accumulation of visceral fat and high body adiposity [29]. After intervention, anthropometric and body composition variables did not differ between groups (Table 4).

Comparing anthropometric and body composition data at baseline and after intervention, only G2 showed a decrease in body weight, BMI, WC, and BFM (kg). On the other hand, G3 showed increased BMI and tendency to increase in body weight and WC (Table 4).

Effect of intervention on energy expenditure and substrate oxidation

In the baseline, there was no difference between groups for the EE-related variables. The diets did not change EE and the substrates oxidation. No differences between times and groups was observed, except for protein oxidation. G2 showed a slight increase in protein oxidation after the intervention, however, the variation was very small without clinical relevance and may be due to the small variability of the data (Table 5).

DISCUSSION

Dietary intervention and fats manipulation studies in humans are scarce, especially with individualized dietary prescription and dietary intake control. The present study showed no effect of high-PUFAs diet on anthropometric and body composition parameters. However, high-MUFAs diet caused weight loss and improvement in body composition, with a decrease in abdominal fat

Table 5. Comparison of energy expenditure variables between groups at the baseline period, after dietary intervention, and variations.

	G1 ($n = 10$)[a]	Δ	G2 ($n = 11$)	Δ	G3 ($n = 11$)	Δ	*P*-value[c]
REE (kcal) I	1480.71 ± 124.10	−19.46 ± 114.73	1468.38 ± 149.84	−28.49 ± 159.65	1549.28 ± 171.17	−48.40 ± 141.18	0.43[e]
REE (kcal) II	1474.78 ± 151.88		1439.89 ± 126.93		1500.88 ± 228.03		0.89[f]
P-value[b]	0.21[d]		0.42[d]		0.21[d]		0.96[g]
TEE (kcal) I	2287.70 ± 191.74	−30.07 ± 177.26	2268.65 ± 231.50	−44.01 ± 246.66	2410.19 ± 274.83	−91.32 ± 262.25	0.39[e]
TEE (kcal) II	2278.53 ± 234.66		2224.64 ± 196.12		2318.86 ± 352.31		0.89[f]
P-value[b]	0.21[d]		0.42[d]		0.21[d]		0.96[g]
RQ I	0.81 ± 0.04	−0.01 ± 0.05	0.77 ± 0.06	0.02 ± 0.06	0.80 ± 0.04	−0.00 ± 0.05	0.20[e]
RQ II	0.80 ± 0.03		0.78 ± 0.04		0.80 ± 0.05		0.57[f]
P-value[b]	0.78[d]		0.16[d]		0.80[d]		0.71[g]
Proteins oxidation (g/m) I	0.05 ± 0.01	0.00 ± 0.01	0.05 ± 0.01	0.01 ± 0.01	0.05 ± 0.01	0.00 ± 0.01	0.31[e]
Proteinsoxidation (g/m) II	0.05 ± 0.00		0.05 ± 0.01		0.06 ± 0.01		0.98[f]
P-value[b]	0.14[d]		0.03[a]		0.13[a]		0.32[g]
Lipids oxidation (g/m) I	0.06 ± 0.01	−0.00 ± 0.01	0.06 ± 0.01	−0.00 ± 0.01	0.06 ± 0.01	−0.00 ± 0.01	0.31[e]
Lipidsoxidation (g/m) II	0.06 ± 0.01		0.06 ± 0.01		0.06 ± 0.01		0.90[f]
P-value[b]	0.14[d]		0.48[d]		0.37[d]		0.95[g]
Carbohydrates oxidation (g/m) I	0.05 ± 0.01	−0.01 ± 0.01	0.05 ± 0.01	−0.00 ± 0.01	0.05 ± 0.01	−0.00 ± 0.01	0.31[e]
Carbohydrates oxidation (g/m) II	0.05 ± 0.01		0.05 ± 0.00		0.05 ± 0.01		0.90[f]
P-value[b]	0.14[d]		0.48[d]		0.37[d]		0.95[g]

Values are represented as mean ± SD (for all values).
Δ delta (after dietary intervention value – baseline value), *G1* n-3 and n-6 PUFAs-rich diet, *G2* MUFAs-rich diet, *G3* control group, *REE* resting energy expenditure, *TEE* total energy expenditure, *RQ* respiratory quotient, *I* baseline values, *II* after dietary intervention values.
*$P < 0.05$.
[a]$n = 09$ in G1 for the final variables REE II and TEE II, as well as for the delta values and paired data analysis.
[b]Differences between times, per group, evaluated using the Wilcoxon signed-rank test, at 5% probability.
[c]Differences between groups analyzed using the Kruskal-Wallis test.
[d]Comparison of baseline and after dietary intervention, according to groups.
[e]Comparison of baseline between groups.
[f]Comparisonafter dietary intervention between groups.
[g]Comparison of Δ between groups.

accumulation. These results suggest a strategy that may improve management the body weight of women with obesity.

Piers et al. [33] found that the high-MUFAs diet decreases body weight and fat when compared to low-MUFAs diet. Weech et al. [34] observed a reduction in WC after high-MUFAs intervention, but no difference between results provided by high-SFAs or high-n-6 PUFAs diets. According Tutinchi et al. [11], diets enriched in oleic acid can influence fat balance, body weight, central obesity, and possibly energy expenditure. The potential mechanisms involved are regulation of food intake, stimulation lipid oxidation, decreased adipose tissue inflammation and adipogenesis.

Long-term dietary intervention has shown UFAs induce greater EE, DIT, and fat oxidation compared to SFAs [10]. Chain size and saturation degree of fatty acids have been pointed as mechanisms responsible for these effects. DeLany et al. [35] observed that PUFAs and MUFAs are higher oxidized, and SFAs oxidation decreases based on the increase of carbon numbers. However, these effects did not occur in the present study. G2 decreased body weight and fat mass without EE and lipid oxidation alteration.

Studies are inconsistent about the effects of UFAs on weight loss and body fat [36, 37]. Nevertheless, MUFAs resulted in weight loss, and a decrease in WC, BMI, and body fat. These effects may be correlated with the greater satiety promoted by MUFAs [38]. G2 showed lower caloric intake than other groups, but no significant difference between groups.

In addition, G2 and G1 reduced carbohydrates intake (% of TEE) during the intervention when compared to usual intake ($P < 0.05$) (data not shown), which did not occur in control group (G3), although carbohydrates intake during the intervention was similar in all groups. According to Paniagua et al. [39], increased carbohydrates intake may increase abdominal fat accumulation, and the opposite could be observed with MUFA-rich diet.

Some evidence suggests that n-3 PUFAs provides an additional effect to caloric restriction on weight loss, WC, and body fat decrease [12, 40]. Nevertheless, other study did not verify these effects [41], confirming our results. These divergences may be related to the offered amounts of PUFAs and gender-related differences [40,42]. Crochemore et al. [42] found higher weight loss and decrease in WC with low dose of n-3 PUFAs (540 mg EPA and DHA/day vs 900 mg EPA and DHA/day). Thorsdottir et al. [40] observed additional benefits in fish intake or n-3 PUFAs oil supplementation in men, but not in women. We emphasize that the dose offered in the present study (1.28 g per day of EPA and DHA) was higher than doses used by Crochemore et al. [42].

The limitations of study include the small sample size and short intervention period. Considering these limitations, the controversial results, and the scarcity of long-term investigations, we emphasize the need to conduct new researches with standardized protocols and long-term intervention to evaluate the influence of UFAs in weight loss, body composition and energy metabolism.

High-MUFAs diet for 60 days contributed to weight loss, especially body and abdominal fat, but did not influence EE and substrate oxidation. However, high-n-3 and n-6 PUFAs diet did not change the variables studied. This result suggests a possible benefit of a high-MUFAs diet for the treatment of obesity, however further studies are needed to clarify the effects of different lipids on individuals with obesity.

DATA AVAILABILITY

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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AUTHOR CONTRIBUTIONS

MCOSL concived and designed the experiments, contributed to data extraction, data analysis, and helped write the manuscript, VCK concived and designed the experiments, contributed to data extraction, data analysis, and helped write the manuscript, LC contributed to data extraction and helped write the manuscript, DPC contributed reagentes and materials, provided feedback on the report, ELR concived and designed the experiments, data analysis and provided feedback on the report designed experiments and helped write the manuscript.

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COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

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