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ORIGINAL ARTICLE

Effects of Dietary Coconut Oil on the Biochemical and Anthropometric Profiles of Women Presenting Abdominal Obesity

Monica L. Assunção · Haroldo S. Ferreira · Aldenir F. dos Santos · Cyro R. Cabral Jr · Telma M. M. T. Florêncio

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Abstract The effects of dietary supplementation with coconut oil on the biochemical and anthropometric profiles of women presenting waist circumferences (WC) >88 cm (abdominal obesity) were investigated. The randomised, double-blind, clinical trial involved 40 women aged 20–40 years. Groups received daily dietary supplements comprising 30 mL of either soy bean oil (group S; $n = 20$) or coconut oil (group C; $n = 20$) over a 12-week period, during which all subjects were instructed to follow a balanced hypocaloric diet and to walk for 50 min per day. Data were collected 1 week before (T1) and 1 week after (T2) dietary intervention. Energy intake and amount of carbohydrate ingested by both groups diminished over the trial, whereas the consumption of protein and fibre increased and lipid ingestion remained unchanged. At T1 there were no differences in biochemical or anthropometric characteristics between the groups, whereas at T2 group C presented a higher level of HDL (48.7 ± 2.4 vs. 45.00 ± 5.6; $P = 0.01$) and a lower LDL:HDL ratio (2.41 ± 0.8 vs. 3.1 ± 0.8; $P = 0.04$). Reductions in BMI were observed in both groups at T2 ($P < 0.05$), but only group C exhibited a reduction in WC ($P = 0.005$). Group S presented an increase ($P < 0.05$) in total cholesterol, LDL and LDL:HDL ratio, whilst HDL diminished ($P = 0.03$). Such alterations were not observed in group C. It appears that dietetic supplementation with coconut oil does not cause dyslipidemia and seems to promote a reduction in abdominal obesity.

Keywords Medium chain fatty acids · Lauric acid · Dyslipidemia

Abbreviations

BMI	Body mass index
BMR	Basal metabolic rate
CVD	Cardiovascular disease
LCFA	Long chain fatty acids
MCFA	Medium chain fatty acid
SFA	Saturated fatty acid
TC	Total cholesterol
TEV	Total energy value
UFA	Unsaturated fatty acid
US-CRP	Ultra-sensitive C reactive protein
WC	Waist circumference

Introduction

In many countries obesity has reached epidemic levels, representing a serious public health problem and a matter of considerable concern for government and health services. In Brazil, the prevalence of obesity has risen from 5.7 to 10.8% of the adult population within the last 30 years, and currently 40.5% of adults are considered to

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M. L. Assunção · H. S. Ferreira (✉) · C. R. Cabral Jr · T. M. M. T. Florêncio
Faculdade de Nutrição, Universidade Federal de Alagoas, Maceió, AL 57072-970, Brazil
e-mail: haroldo.ufal@gmail.com

A. F. dos Santos
Faculdade de Ciências Biológicas e da Saúde, Fundação Jayme de Altavila, Maceió, AL 57051-160, Brazil

be overweight. Although dramatic increases in average body weight have affected the entire socioeconomic strata, they have been proportionally greater amongst poor women [1, 2].

Obesity results from a positive energy balance that leads to the accumulation of body fat at various anatomical sites. If fat accumulation occurs predominantly in the abdominal region the condition is known as visceral obesity, a factor that is strongly associated with increased risk of cardiovascular diseases (CVDs) [3]. The type of fat provided by the diet influences the incidence of obesity and also plays a significant role in the aetiology of various metabolic disorders including dyslipidemia [4]. Although many theories concerning the types of fatty acids that are beneficial or deleterious to health are contentious, it is generally considered that saturated fatty acids (SFAs) are hypercholesterolemic whereas unsaturated fatty acids (UFAs) are hypocholesterolemic [5].

Oil from coconuts (*Cocos nucifera* L.) contains a high proportion of medium chain fatty acids (MCFAs), principally the SFA lauric acid (12:0), in proportions that range from 45 to 50% [6]. A possible association between dietary intake of coconut oil and the incidence of CVDs was suggested four decades ago following studies carried out by the food industry involving hydrogenation of the oil and modification of some of the UFAs present, particularly oleic and linoleic acids. On the basis that the harmful effects of coconut oil observed in experimental animals resulted from the hydrogenation of UFAs, health professionals began to recommend the replacement of coconut oil by UFA-rich soy bean (*Glycine max* L.) oil [7]. However, studies involving African and South Pacific populations, whose diets contain large proportions of coconut oil (80% of daily lipid intake), have revealed that there is no association between the ingestion of coconut oil and the occurrence of obesity and/or dyslipidemia [8–10]. Additionally, coconut oil is frequently used in the treatment of obesity by virtue of its high content of MCFAs [11], since such lipids are easily oxidised and are not normally stored in the adipose tissue, thus diminishing the basal metabolic rate (BMR). However, the use of coconut oil in the diet remains controversial owing to the possible detrimental effects of SFAs and their association with dyslipidemia and CVDs [4, 12, 13].

Obesity is a particularly serious problem in the State of Alagoas, which is located in the north-east of Brazil and is a major producer of coconut oil. Considering the lack of evidence concerning the physiological effects of intake of coconut oil, the aim of the present study was to investigate the effects of dietary supplementation with coconut oil on the biochemical and anthropometric profiles of a population of women of low socioeconomic status living in Alagoas and suffering from abdominal obesity.

Materials and Methods

Study Design and Subjects

The project of work was submitted to and approved by the Ethical Committee in Research of the Faculdade de Ciências Biológicas e da Saúde, Fundação Educacional Jayme de Altavila, Maceió, AL, Brazil. Written informed consent was obtained from all participants prior to the commencement of the study.

The study took the form of a randomised, double-blind, clinical trial involving 40 women selected from those attending an outpatients unit located in Marechal Deodoro, AL, Brazil. The selection criteria were: (1) age between 20 and 40-years-old, (2) low socioeconomic status (per capita family income <USD$1/day), and (3) presenting abdominal obesity as defined by waist circumference (WC) >88 cm [14]. Pregnant women were excluded from the selection, as were subjects presenting arterial hypertension, chronic degenerative diseases, endocrinopathies or body mass index (BMI) >35 kg/m^2. The exclusion of women >40-years-old and with a BMI >35 kg/m^2 was intended to reduce the probability of including individuals with unknown or undiagnosed morbidities in the sample population, as well as women with menopausal hormone alterations and those presenting a degree of obesity unresponsive to dietetic treatment.

The study population was randomly divided in two groups of 20, paired according to BMI, one of which (group S) received soy bean oil as dietary supplement, while the second (group C) received coconut oil. Individual dietary counselling for all participants was provided by a nutritionist throughout the entire experimental period of 12 weeks. Subjects were instructed to follow a diet that was balanced in respect of macronutrients with increased consumption of fruits and vegetables and reduced consumption of simple carbohydrates and animal fat. Participants were also advised to drink adequate amounts of water and to reduce or eliminate alcohol consumption and smoking. Under the supervision of a fitness trainer, all of the participants took part on 4 days every week in a physical activity program comprising elongation followed by 50 min walking. Both, the physical trainer and the nutritionist were not aware of the distribution of the individuals between the study groups.

The dietary intervention consisted in the daily ingestion of either 30 mL of soy bean oil (group S) or 30 mL of coconut oil (group C) distributed through the three main meals. Participants were instructed that the daily oil supplement should be used in the preparation of food as normal, but that the nutritional recommendations provided must be followed. Each subject received a supply of oil (sufficient for the experimental period) contained in a

numbered flask, together with a standardised measuring spoon. In order to ensure that neither the participant nor the researchers had knowledge of the dietary supplement applied, the oil flasks were filled and numbered according to a supplement key prepared by an investigator, who was not involved in the collection of data, and the flasks were distributed to the participants by another researcher who had no knowledge of the contents of the supplement key. Data relating to the variables of interest were collected 1 week before the start of dietary intervention (T1) and again 1 week after the 12th week of intervention (T2). The supplement key was not revealed to the participants or to the research team until all data had been collected and analysed.

Origin and Composition of Oil Supplements

Filtered coconut oil, obtained by pressing coconut pulp that had been dehydrated at 60 °C, was provided by a local manufacturer (Sococo Indústria Alimentícia, Maceió, AL, Brazil). Soy bean oil (Bunge Alimentos, Pernambuco, Brazil) was purchased from a local supermarket. The compositions of the oil supplements (Table 1) were determined by an accredited laboratory (SFDK Laboratório de Análise de Produtos Ltda, São Paulo, SP, Brazil). The two types of oil were of a similar colour but differed slightly with respect to odour and flavour. In order to conceal the differences, the oils were bottled in 600 mL amber flasks and could not be readily distinguished except by direct comparison.

Socioeconomic Evaluation

Socioeconomic information was obtained from each participant by application of a specific questionnaire relating to the type of accommodation occupied, number of rooms, sanitary facilities, schooling, occupation and family income, number of family members, alcohol intake and smoking habits, and physical activities.

Table 1 Main fatty acids present in soy bean and coconut oils

Fatty acid	Composition (%)	
	Soy bean[a] oil	Coconut[b] oil
Lauric acid (12:0)	0	49.0
Myristic acid (14:0)	0.1	17.5
Palmitic acid (16:0)	10.3	9.0
Stearic acid (18:0)	3.8	3.0
Oleic acid (18:1 ω-9)	22.8	5.0
Linoleic acid (18:2 ω-6)	51.0	1.8
Total	100.0	100.0

Source: SFDK Laboratório de Análise de Produtos Ltda., São Paulo, SP, Brazil

[a] *Glycine max* L.

[b] *Cocos nucifera* L.

Anthropometric Evaluation

The body mass of each subject wearing light clothes was determined using Filizola SA (São Paulo, SP, Brazil) Personal PL 180 portable digital scales with a maximum capacity of 180 kg and a precision of 100 g. Heights of subjects (with bare feet) were measured using a WCS (Curitiba, PR, Brazil) stadiometer connected to a non-extendable 220-cm measuring tape (1 mm precision). WC was measured (with subjects standing upright and immediately after exhalation) at the mid point between the last rib and the anterior superior iliac spine using a non-extendable measuring tape of 200 cm. BMI was calculated by dividing body mass (kg) by the square of the height (m). Values were categorised in accordance with the cut-off points recommended by the World Health Organisation guidelines [14] for BMI [<18.5, 18.5–24.9 (or <25.0), 25.0–29.9 (or <30.0) and >30.0; kg/m^2] and WC (</>88 cm) or were considered as continuous variables.

Dietary Evaluation

In order to evaluate compliance with expected dietary intake, the effects of lipid supplementation, and possible confusing effects between the different groups, a 24 h dietary recall was applied to subjects for a 3 day period (1 day of which was during a weekend) immediately before and 12 weeks after dietary intervention. Images depicting different quantities of food were used to assist participants in assessing the amounts of food consumed [15]. Data were collected by the researcher in charge of nutritional supervision and were processed using Nutwin software (Departamento de Informática em Saúde, Universidade Federal de São Paulo, SP, Brazil). BMR values were calculated according to the procedure of the Food and Agriculture Organisation [16] taking into consideration differences between the age groups as follows: BMR (18–30 years) = (14.7 × weight) + 496 BMR (30–60 years) = (8.7 × weight) + 829.

Biochemical Evaluation

Blood samples were collected 1 week before the start of dietary intervention (T1) and 1 week after the 12th week of intervention (T2). In each case blood was taken in the morning following a 12 h overnight fast. Glucose, triglycerides, total cholesterol (TC) and LDL/HDL fractions were determined in triplicate for each sample using standard laboratory kits (Boehringer Mannheim, Germany).

The quotients TC/HDL and LDL/HDL were calculated on the basis of the plasma lipoprotein values. The levels of fibrinogen, ultra-sensitive C reactive protein (US-CRP) and insulin were determined by thermo-precipitation, nephelometry and chemiluminescence, respectively. The secretory function of pancreatic beta cells and insulin resistance were calculated using the homeostatic model assessments (HOMA-β% and HOMA-S, respectively) according to the equations proposed by Mathews et al. [17].

$$\text{HOMA-}\beta\% = \frac{20 \times \text{insulin}(\mu\text{U/mL})}{\text{glucose(mmol/L)} - 3.5}$$

$$\text{HOMA-S} = \frac{\text{insulin}(\mu\text{U/mL}) \times \text{glucose}}{22.5}$$

Statistical Analysis

The experiment was carried out according to a totally randomised design and took the form of a 2 × 2 factorial scheme (factor A—type of oil tested on the two groups; factor B—two assay periods during the 12 weeks of dietary intervention). There were four treatments: (1) soy bean oil at zero time, (2) soy bean oil after 12 weeks, (3) coconut oil at zero time; and (4) coconut oil after 12 weeks. The interaction between factors A and B was analysed statistically.

In order to reduce the magnitude of the error variance, analysis of covariance (ANCOVA), was performed considering or not the significant differences between the two groups in relation to the supposed covariate age. In this context, H_o implies that all β_I are equal to β, whilst H_1 requires at least one β_I is not equal to β for the same factor means. The equation applied was $y_{ij} = \mu + \tau_i + \beta_i x_{ij} + e_{ij.}$ The χ^2 test was used to determine if there were significant differences between the two groups of women with respect to frequency of alcohol consumption or smoking habit. The normality of the distribution and the homoskedasticity of the residual variances were analysed using Shapiro–Wilk and Levene tests, respectively, for all studied variables. These tests were also used to determine if body mass, BMI, US-CRP, triglycerides, insulin, HOMA-β% and HOMA-S obeyed the parametric assumptions. The values relating to these variables were then subjected to logarithmic transformation, i.e. $y_i = \log(X_i)$. The percentage values of protein components were arsine transformed, i.e. $y_i = \text{arcsine } X_i$. After the application of the Snedecor test (F test), multiple comparisons between treatments were carried out using the post hoc Bonferroni test ($P < 0.05$) with correction factor. Both tests were applied to all dependent variables.

The level of significance was established at 5% for all statistical tests. However, when the probability of the experimental error was >5 and <10% (i.e. $0.05 > P < 0.1$), the difference was taken to indicate the probability of a biological significance.

Results

Most participants (60%) lived in brick houses comprising three rooms on average but lacking sewage facilities. Most women were illiterate (68.2%) and their sole occupation was to act as housewife (90%). Each family unit consisted on average of 5 members with a per capita income of R$ 1.86/day (equivalent to US$ 0.82/day in January 2006). Approximately a quarter of the women (22.5%; 4 in group S and 5 in group C) smoked regularly, and more than half of the participants (52.5%; 11 in group S and 10 in group C) ingested alcohol occasionally. There were no significant differences ($P \geq 0.05$) between the two groups with respect to the variables alcohol and smoking habits (χ^2 alcoholism = 0.10 and χ^2 smoking = 0.143; df = 1 for both variables; P values = 0.759 and 0.705, respectively), indicating that these covariables exerted no significant antagonism or synergism on the outcomes.

The median height of the studied population was found to be 1.55 m, corresponding to an estimated ideal weight of 51.63 kg and an ideal daily energy intake of 1,975.6 kcal. The daily energy consumption of the population was determined to be 1,893 kcal on average at the start of the trial, and this corresponded to 95.8% of the ideal requirement estimated on the basis of assessed height.

The results at T1 showed that the diet of the participants was poor in lipids [19% of total energy value (TEV)] and proteins (0.77 g/kg of body mass per day), and this was associated with an elevated ingestion of carbohydrates (69% of TEV). Tubers, flour and bread derived from traditional carbohydrate-rich plants were consumed with abundance. The frequency of ingestion of foods with cardio-protective properties, such as fruit and vegetables, was practically zero, while the intake of fibre was around 10 g a day.

The relative contribution of macronutrients to the daily energy intake varied little between T1 and T2 for the S and C groups. The TEV supplied by carbohydrates was reduced from 69 to 61.8% during this period, whilst the TEV supplied by lipids remained almost unaltered (changing from 19 to 20.8%) despite supplementation of the diet with 30 mL/day of oil (equivalent to 270 kcal). Regarding proteins, at T1 the participants ingested a hypoproteic diet whereas at T2 the diet was considered normoproteic (0.99 g/kg of weight a day). The mean TEV was reduced from 1,893 (T1) to 1,732 kcal/day (T2) as a result of the hypocaloric diet presented to the subjects and supervised by researchers during the 12 week dietary intervention.

The results of the 24 h dietary recall performed at T1 and T2 are presented in Table 2. The energy intake and the amounts of carbohydrates, lipids, proteins and fibre were similar in both S and C groups. Within the groups, however, some differences of similar magnitude could be

Table 2 Dietetic profiles of women presenting abdominal obesity determined before and after dietary supplementation with soy bean (group S) and coconut (group C) oils during a 12-week period

Variables	24 h Dietary recall	Study population		*P* value[b]
		Group S ($n = 20$)[a]	Group C ($n = 20$)[a]	
Energy (kcal)	First evaluation (T1)	1,887.3 ± 163.9	1,894.0 ± 190.6	0.916
	Second evaluation (T2)	1,700.1 ± 154.7	1,696.9 ± 176.5	0.958
	Δ%[c]	−9.9**	-10.4**	
Protein (g)	First evaluation (T1)	54.2 ± 7.6	58.8 ± 7.2	0.084
	Second evaluation (T2)	62.4 ± 6.3	61.7 ± 7.2	0.769
	Δ%[c]	15.0***	4.9*	
Carbohydrates (g)	First evaluation (T1)	327.2 ± 33.6	325.2 ± 36.0	0.874
	Second evaluation (T2)	272.2 ± 28.6	272.9 ± 33.8	0.946
	Δ%[c]	−16.8***	−16.1***	
Lipids (g)	First evaluation (T1)	40.2 ± 1.6	39.8 ± 2.7	0.607
	Second evaluation (T2)	40.2 ± 2.3	39.8 ± 2.1	0.653
	Δ%[c]	0.2 ns	0,1 ns	
Fibres (g)	First evaluation (T1)	10.03 ± 1.0	9.97 ± 1.1	0.872
	Second evaluation (T2)	14.98 ± 1.6	15.02 ± 1.8	0.944
	Δ%[c]	49.4***	50.7***	

ns Not significant

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

[a] Mean values ± standard deviations

[b] According to Bonferroni test

[c] $\Delta\% = [(T2 - T1)/T1] \times 100$

observed. For example, there was a reduction of 10% in total energy intake and 16% in the ingestion of carbohydrates. In contrast, there was an increase (of approximately 50%) in the ingestion of proteins and fibres. The ingestion of lipids, however, remained unaltered.

The biochemical and anthropometrical data collected at T1 and T2 are presented in Table 3. ANCOVA analysis showed that there were no significant differences ($P \geq 0.05$) between groups S and C concerning the covariate age as determined by the H_o value. The Snedecor test (F test) showed a significant difference ($P < 0.05$) between the two groups concerning the type of oil (factor A) and assessment time (factor B). However, there was no significant interaction between factors A and B ($P \geq 0.05$) for any of the variables in this study (Table 3).

At the end of the trial period, reductions in body mass and BMI were observed in both groups ($P < 0.05$), but only group C presented a significant reduction ($P = 0.005$) in WC. The levels of total cholesterol, LDL and LDL:HDL ratio were significantly ($P < 0.05$) increased in group S, whilst the level of HDL was significantly ($P = 0.03$) diminished. Such alterations in the lipid profile did not occur in group C. In contrast, this group presented a tendency towards increased HDL although the alteration was not statistically significant ($P = 0.09$). At T1 the HDL level of group S tended ($P = 0.08$) to be higher than that of group C, whilst at T2 the situation was reversed and the HDL level of group C was significantly higher, as shown by a P value of 0.03 consequently, at T2 the LDL:HDL ratio of group C was significantly ($P = 0.04$) lower than that of group S. According to the LDL:HDL ratios determined in the present study, the S and C groups cannot be considered to be at risk with respect to CVDs since the values were below 3.5.

The glucose levels of the S and C groups at T1 and T2 were similar. There was no change in the release of insulin in group S, whereas in group C the increase in hormone release observed, although not statistically significant ($P = 0.09$), was sufficient to increase the HOMA-S value of group C to a level significantly higher ($P = 0.03$) than that of group S. There were no significant differences between the S and C groups with respect to the remaining parameters (i.e. height, age, levels of triglycerides, US-CRP, fibrinogen, glucose and HOMA-β%) at both evaluations.

Discussion

The two groups of women presenting abdominal obesity endured similar socioeconomic conditions and lifestyles. The reductions in body mass and BMI of both S and C

Table 3 Metabolic and anthropometric profiles of women presenting abdominal obesity determined before and after dietary supplementation with soy bean (group S) and coconut (group C) oils during a 12-week period

Variables	Desirable values	Group S ($n = 20$)				Group C ($n = 20$)			
		First evaluation (T1)[1]	Second evaluation (T2)[1]	Δ (T2 − T1)	*P*	First evaluation (T1)[1]	Second evaluation (T2)[1]	Δ (T2 − T1)	*P*
Height (m)	–	1.56 ± 0.07	–	–	–	1.54 ± 0.05	–	–	–
Age (years)	–	28.5 ± 6.7	–	–	–	31.0 ± 6.4	–	–	–
Body mass (kg)	–	76.0 ± 9.0	75.0 ± 9.1	−1.0*	0.02	73.2 ± 9.0	72.1 ± 9.1	−1.1*	0.002
BMI (kg/m^2)	18.5–24.9	31.1 ± 3.2	30.7 ± 3.3	−0.4*	0.02	31.0 ± 3.6	30.5 ± 3.6	−0.5*	0.003
Waist circumference (cm)	<88	96.4 ± 5.1	97.0 ± 6.5	0.6	0.39	98.8 ± 6.7	97.4 ± 7.0	−1.4*	0.005
Total cholesterol (mg/dL)	<200	189.5 ± 22.2	209.3 ± 28.5	19.8*	0.001	192.5 ± 41.2	198.1 ± 39.0	5.6	0.69
HDL (mg/dL)	40–60	51.5 ± 10.0[C]	45.00 ± 5.6[A]	−6.5*	0.04	45.5 ± 7.1[c]	48.7 ± 2.4[a]	3.2[‡]	0.11
LDL (mg/dL)	<100	108.6 ± 15.9	134.1 ± 28.7	25.5*	0.01	112.6 ± 37.8	116.5 ± 36.8	3.9	0.69
Triglycerides (mg/dL)	<150	147.2 ± 75.2	148.2 ± 64.8	1.0	0.93	172.8 ± 88.1	179.7 ± 93.7	6.9	0.65
LDL:HDL [18]	≤2.9	2.2 ± 0. 5	3.1 ± 0.8[B]	0.9*	0.002	2.49 ± 0.8	2.41 ± 0.8[b]	−0.1	0.73
US-CRP (mg/L)	<1	4.9 ± 4.0	4.2 ± 3.2	−0.7	0.52	5.7 ± 4.9	3.7 ± 1.7	-2.0	0.11
Fibrinogen (mg/dL)	200–400	241.5 ± 41.5	243.6 ± 43.9	2.1	0.91	254.0 ± 42.2	243.8 ± 41.9	−10.2	0.85
Glucose (mg/dL)	70–100	81.8 ± 9.0	78.5 ± 9.9	−3.3	0.36	83.5 ± 7.8	82.8 ± 5.4	−0.7	0.81
Insulin (μU/mL)	<29.1	8.9 ± 3.3	7.6 ± 2.1	−1.3	0.27	9.0 ± 4.5	9.8 ± 4.1	0.8[‡]	0.09
HOMA-S% [19]	1.66 ± 0.81	1.83 ± 0.82	1.48 ± 0.45[D]	−0.4	0.20	1.8 ± .0.9	2.0 ± 0.9[d]	0.2	0.11
HOMA *β*% [19]	> 100%	35.3 ± 13.0	31.8 ± 9.8	−3.5	0.44	36.1 ± 20.1	39.4 ± 18.0	3.3	0.20

US-CRP Ultra-sensitive C reactive protein

* Difference statistically significant ($P < 0.05$) and [‡]difference biologically significant ($P \geq 0.05$ to <0.1) according to Bonferroni test

[1] Mean values ± standard deviations

[A/a,B/b] Difference statistically significant ($P < 0.05$) according to Bonferroni test

[C/c,D/d] Difference biologically significant ($P \geq 0.05$ to <0.1) according to Bonferroni test

groups observed at T2 may be attributed to the negative energy balance resulting from healthier habits (i.e. a more balanced diet and additional physical activity) arising from the counselling administered during dietary intervention. The reduction in abdominal fat in individuals of group C can be explained by their consumption of MCFA-rich coconut oil, since these components are not readily incorporated into the triglycerides of adipose tissue [20]. Moreover, unlike triglycerides containing long chain fatty acids (LCFAs), those comprising MCFAs are more susceptible to oxidation even under resting conditions. This difference may be explained by the fact that LCFAs are dependent on carnitine for mitochondrial transport, whilst MCFAs are transported through the inner mitochondrial membrane independent of the carnitine acyl transferase system [21].

Studies on the effects of a diet rich in MCFAs (equivalent to 45% of TEV) compared with a diet rich in LCFAs administered over a 21-day period showed that plasma TC, LDL and triglyceride levels increased by 11, 12 and 22%, respectively, as a result of the ingestion of MCFAs, while HDL levels remained unchanged [22]. However, a diet providing an intake of MCFAs in excess of 30 g/day would be nutritionally unbalanced and likely to produce undesirable collateral effects, which might explain the results observed [23]. In the present study, the hypocaloric diet adopted by the individuals of group C was supplemented with MCFA-rich coconut oil (as the main source of lipids), but no undesirable alterations were observed in the lipid profile of the participants after 12 weeks. In contrast, some researchers [24] have observed that subjects supplied with an amount of lauric acid significantly greater than that used in the present study, exhibited hypercholesterolemia. These results may be explained, however, by the unbalanced relation between saturated, monounsaturated and polyunsaturated fatty acids in the diet applied.

The majority of studies that have focussed on the hypercholesterolemic effect of MCFAs have employed hyperlipidic diets from which essential mono and polyunsaturated fatty acids, such as linoleic (18:1) and arachidonic (20:4) acids, were absent. Arachidonic acid is an important modulator of anti-inflammatory responses and is a precursor of the eicosanoids (leukotrienes, prostaglandins, thromboxanes and lipoxins), which present diverse biochemical

and physiological roles [25]. Such diets would, therefore, have been unbalanced and could have given rise to numerous metabolic disorders.

The reduction in HDL levels within group S observed in the present study may be explained by the high content of linoleic acid (51%) present in the soy bean oil, which may have undergone conformational changes during cooking. The process of heating transforms *cis*-linoleic and *cis*-linolenic acids into their respective *trans* isomers leading to alterations in lipid metabolism and to the under-expression of LDL receptors and, consequently, to an increase in blood LDL [26]. Whilst no evidence was obtained in the present study to confirm the formation of *trans* UFAs, the ingestion of soy bean oil by subjects of group S definitely induced an increase in the concentration of serum LDL after 12 weeks. There is a consensus amongst researchers concerning the deleterious effects on the levels of LDL of *trans* UFAs in comparison with SFAs. For example, Roos and co-workers [27] carried out a comparative study of a diet rich in lauric acid and a diet rich in *trans* UFAs derived from soy bean oil and observed that individuals supplied with the former presented a healthier lipid profile. In the present study, it is clear from the 24 h dietary recall that the participants used the oil provided for frying foods and as a supplement to the diet. However, the time spent in cooking, the temperature used in the processes, and the possible formation of *trans* isomers were not directly investigated. Undoubtedly, the influence of such variables warrants consideration and such a study would contribute to the knowledge already gathered. Nevertheless, it is important to emphasise that prior to nutritional intervention, several participants reported that they used the same cooking oil twice or three times as an economy measure. Although such practice was strongly discouraged during the study and the amount of oil supplied was ample for the needs of the participants and their families, full compliance was out of the control of the research team.

Much research has focussed on attempts at elucidating the real effects of SFAs on the lipid profile and the possible association with CVDs [28–30] but the results achieved are controversial since the amounts of oil employed, together with their fatty acid compositions, varied in the different studies. Further factors that could also explain the diverse results deriving from the various investigations include the duration of the dietary intervention, the ingestion of dietetic cholesterol, and the level of antioxidants ingested. In the present study, the daily ingestion of 30 g of coconut oil during a 12-week period did not alter the lipid metabolism of the women of group C, including those who had exhibited some degree of hypertriglyceridaemia at T1. This result may be explained by the rapid oxidation of MCFAs, particularly lauric acid, as well as by their low incorporation into VLDLs. MCFAs are poor substrates for Δ^6 and Δ^9 fatty acid desaturases and, unlike palmitic acid (16:0), cannot undergo elongation. The hypercholesterolemic activity of palmitic acid is a consequence of such elongation reactions and the subsequent formation of LCFAs [31]. The presence of antioxidant polyphenols in the coconut oil could also have contributed to the results obtained in the present study. It is of interest to note that reduced levels of LDL, VLDL and total cholesterol, together with increased levels of HDL, have been reported in experimental animals that had been fed with coconut oil [32].

Regarding the carbohydrate profile, it has been postulated that the secretion of insulin varies according to the type of fatty acids supplied in the diet [33]. UFAs, such as linoleic and linolenic acids, potentiate insulin secretion in response to the basal concentration of glucose, whereas SFAs diminish the response of the islets of Langerhans to glucose concentration [34]. In the present study, individuals of group C exhibited a tendency towards increased insulin secretion following 12 weeks on a diet rich in lauric acid, a finding that is in agreement with a previous report that supplementation of lauric acid in experimental animals stimulated the secretion of insulin [35].

It is important to emphasise that the HOMA-S values representing insulin resistance were higher than normal in both groups and at both evaluations, a condition probably associated with abdominal obesity that was common to all participants of the study. The levels of USCRP, an inflammatory marker and an independent predictor of CVD risk [35], were also above the normal limit (<1 mg/L) in both groups and at both evaluations. The levels of fibrinogen were, however, normal at both T1 and T2. This protein is an important component of platelet aggregation and is an indicator of acute conditions. When the levels are normal, fibrinogen performs a cardio-protective function even in the presence of high levels of cholesterol [36].

It is important to stress that the background diet of participants in the present study was richer in carbohydrates (about 70%) than that of most Western populations (around 50–55%). Stable isotope experiments have shown that individuals submitted to carbohydrate-rich diets exhibit higher rates of de novo lipogenesis [37, 38] and decreased VLDL clearance and fat oxidation [39] compared with those ingesting diets poor in carbohydrates and rich in fat. Considering that the subjects of this study did not increase fat intake significantly, and that the carbohydrate intake diminished by more than 15%, it is possible that metabolic adaptations were induced by their normal diet pattern that could have influenced the results obtained. For this reason, the effects of coconut oil reported here cannot be extrapolated to other populations. Additionally, the 24-h dietary recall presented various limitations that must be considered in the evaluation of food consumption. Among such limitations are recall bias and the fact that a

single dietary recall does not characterise the habitual feeding habits of an individual owing to high intra-personal variability [40]. In order to minimise reporting bias a photo album containing images of different portions of food was employed, and to reduce intra-personal variability three different 24 h dietary recalls were conducted including one at the weekend when variations in the usual consumption pattern are most likely to occur.

In conclusion, the ingestion of coconut oil did not produce undesirable alterations in the lipid profile of women presenting abdominal obesity, although dietary supplementation with this oil did give rise to a reduction in WC, which is considered to confer some protection against CVDs. On the other hand, the ingestion of coconut oil appeared to have induced an increase in peripheral insulin resistance. The results presented here indicate that SFAs cannot be characterised as the sole aetiological cause of obesity, dyslipidemia and risk factor for CVDs, but that the overall composition of the diet, particularly the fatty acid, cholesterol and antioxidant fractions, as well as the lifestyle of the individuals, must be taken into consideration. Within this context, it would be important to evaluate the effects of coconut oil over a prolonged period, and to investigate the composition and effects of the polyphenolic fraction of the oil.

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