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RESEARCH ARTICLE

Almonds and Walnuts Consumption Modifies PUFAs Profiles and Improves Metabolic Inflammation Beyond the Impact on Anthropometric Measure

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Abstract:

Purpose:

To evaluate changes in serum fatty acids, metabolic profile and inflammation markers after a dietary intervention of 15g of walnuts and 15g of almonds for 8 weeks in obese subjects,

Patients and Methods:

We studied a total of 48 sedentary obese grade I subjects (13 men and 35 women). Anthropometric measures, body composition, serum glucose, lipid profile, insulin, lipocalina-2, high sensitivity C-reactive protein (hsCRP), adiponectin, and fatty acids profile were analyzed at the baseline and after dietary intervention.

Results:

The adiponectin (30.4%, $p = 0.007$), and lipocalin-2 concentrations (17.9%, $p = 0.014$), and total Polyunsaturated Fatty Acids (PUFAs) percentage (1.6% $p = 0.040$) significantly increased after the intervention; particularly the eicosapentaenoic acid and docosahexaenoic acid percentages were increased marginally. A significant decrease in saturated fatty acids levels (3%, $p = 0.001$), in particular the C:14, C:16, in total cholesterol (6.7%, $p = 0.01$), LDL (11.4%, $p = 0.002$) levels, and in all adiposity measures (weight, waist circumference, hip circumference, BMI and fat mass, $p < 0.0001$) was found. The effect size was large for all adiposity measures, except for BMI as well as for adiponectin which was moderate.

Conclusion:

The intake of almonds and walnuts to short-time may improve significantly the metabolic profile and decrease adiposity.

Keywords: Obesity, Walnuts, Almonds, Adiponectin, Lipocalin-2, Metabolic profile.

1. INTRODUCTION

Obesity has emerged as a serious public health threat of the 21st century [1]. According to the 2016 National Health

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and Nutrition Survey in the Mexican population, the prevalence of overweight and obesity is 72.5% in adults aged 20 and older [2]. Recent evidence suggests that the amount and type of fat included in the diet contributes to the development of obesity and insulin resistance, influencing the secretion of adipokines from obese adipose tissue [3].

Several studies have demonstrated that the consumption of almonds [4, 5], walnuts [6, 7] or mixed nuts [8] has a favorable effect on the lipid profile and insulin resistance. Walnuts have a high content of Polyunsaturated Fatty Acids (PUFAs) 47.2%, especially α-linolenic acid (C:18:3 n-3) 9.1%, and linoleic acid (C:18:2 n-6) 38.1%. [9] Also, nuts are rich in other bioactive compounds, such as phytosterols [9]. The richness in bioactive compounds may be responsible for a significant increase in High-Density Lipoprotein cholesterol (HDL) and a reduction in Low-Density Lipoprotein cholesterol (LDL), achieved after consumption of 30 g of walnuts for 6 months in fifty-eight subjects aged 59.3 ± 8.1 years [10]. A meta-analysis investigating the impact of walnut consumption on blood lipids showed that walnut-enriched diets significantly decreased total cholesterol and LDL when compared with the control diet [11]. Since almonds contain a high content of Monounsaturated Fatty Acids (MUFAs) (31.5%), and PUFAs (12.1%), [9] a reduction in LDL range appears to be between 11.6 and 13.4% following the consumption of almonds [12, 13]. However, other reports have failed to show significant changes in weight or Body Mass Index (BMI) after the intake of almonds and walnuts were observed [14, 15]. The intake of 30 g/day of raw nuts (15 g walnuts, 7.5 g/almonds and 7.5 g hazelnuts [16] is not associated with a reduction in LDL, as neither the consumption from 63 to 108 g/d of walnuts decreased the total cholesterol, LDL, and triglycerides levels [17].

Adiponectin is an anti-inflammatory adipocytokine that has been suggested to play a causative role in the development of insulin resistance, diabetes, and atherosclerosis [18]. An increase in circulating concentrations of adiponectin is observed after a short-term dietary intake of walnuts in obese subjects [19]. Lipocalin-2 (LCN2) is an extracellular protein expressed by adipose tissue that induces the expression of peroxisome proliferator activated receptor gamma (PPARγ), and its target genes, adiponectin and lipoprotein lipase [20]. An association between LCN2 and BMI, waist circumference, fat percentage, hypertriglyceridemia, and insulin resistance [21] has been reported, but another study failed to find increased circulating LCN2 concentrations in obese patients [22]. It had also been reported that the circulating LCN2 levels were decreased in patients with long-term type 2 diabetes [23]. Therefore, the role of LCN2 as a potential metabolic and cardiovascular risk factor remains to be fully elucidated. These data show that the beneficial effects on the amount of the intake and type of nuts on metabolism and inflammation are still controversial. In addition, there are few reports that examined the effect of nut consumption on circulating adiponectin levels, and even, it is not known whether consumption of nuts increases LCN2 concentrations. The aim of this work was to evaluate changes in serum Fatty Acids (FA), metabolic profile and inflammation markers after a diet supplemented with a daily intake of 15g of walnuts and 15g of almonds for 8 weeks in obese subjects.

2. MATERIAL AND METHODS

2.1. Participants and Study Design

The present study comprised a clinical trial with 8 weeks of follow-up. A total of 61 obese grade I subjects were included, 13 did not finish the intervention; 10 of them were excluded due to lack of adherence to diet and 3 of them presented a migraine crisis. A preliminary report comprising 30 subjects was done previously. In this study, a total of 48 subjects completed the intervention. The subjects were selected by means of advertisements in local media and shoe factories for a period of 14 months. Obese subjects of 30 to 50 years old with BMI between 30 and 34.9 kg/m^2 (grade I obesity) without evidence of chronic degenerative, infectious or neoplastic diseases, neither medication nor actually a dietary treatment, and a physical exercise level < 2 hours/week were recruited. All participants gave their written informed consent to participate in the study. The study was approved by the Institutional Ethics Committee of the Universidad de Guanajuato and was conducted according to the ethical standards laid down in the Declaration of Helsinki 1983 and in agreement with the Good Clinical Practice guidelines.

2.2. Dietary Assessment Method

On baseline and at the end intervention, the usual dietary intake was evaluated by direct application of 24-hour reminders (two weekdays and one weekend), using the United States Departament of Agriculture (USDA) Five-Step Multiple Penetration Automated Method [24]. The energy and nutrient intake were evaluated with the databases of the USDA [25] and SNUT software (National Institute of Public Health, Mexico) [26].

2.3. Intervention Protocol

The habitual diet was enriched with 15g of almonds and 15g of walnuts per day for eight weeks, indicating its consumption as a snack, supplied directly to the patient by the researcher. These amounts of walnuts and almonds contain PUFAs approximately 2.02g of omega 3 (ω-3) (alpha-linolenic acid C:18:3 ω-3) and 11.1g of omega 6 (ω-6), linoleic acid C:18:2 ω-6 mostly. In order to evaluate adherence, volunteers were visited every two weeks. Positive Adherence was considered when the consumption of walnuts and almonds was not modified more than ± 20% of the amount indicated by the researcher. For analysis, only those who adhered at least 80% of the consumption of walnuts and almonds were considered.

2.4. Anthropometric and Clinical Measures

Weight was measured with a roman type Tanita BC533 scale, height was measured using a Stadiometer SECA 406. Waist and hip circumferences were measured using a tape SECA 206 according to the technique of Lohman TG [27]. The fat mass percentage was determined by bioimpedance using a Tanita BC533 instrument. The obtained weight and height were used to calculate the BMI. Systolic and diastolic blood pressures were measured in a sitting position after a ten-minute rest. All measures were conducted in duplicate by standardized personnel. All anthropometric and metabolic measures were evaluated at baseline and after eight weeks of treatment.

2.5. Biochemical Measurement

Blood samples were withdrawn after 12 h of fasting to quantify circulating levels of serum glucose, lipids, insulin, LNC2, high-sensitivity CRP (hsCRP) and adiponectin. Serum samples were separated and frozen to -80ºC until analysis. Serum glucose (coefficient of variation 5.6%) and lipids profile were measured using enzymatic methods with a chemistry analyzer (Auto KEM II, Kontrollab, Italy). The coefficient of variation was 4.3% for total cholesterol, 6% for Triglycerides, and 3% for HDL. Serum insulin was measured by radioimmunoassay with a commercial kit (BI-Insulin-IRMA, Cisbio Bioassay, Codolet, France), with an intra-assay Variation Coefficient (VC) of 3.9%. Serum LNC2 was measured using Quantikine ELISA (R & D Minneapolis MN, USA) with 4.4% CV. The hsCRP was quantified by means of hsCRP ELISA Kit (ALPCO Immunodiagnostic AG, Stubenwald-Allee, Bensheim) with 5.5 CV%. For quantification of adiponectin, a radioimmunoassay kit (Millipore, St. Charles, Missouri, USA) with CV of 3.6% was used; while the Homoeostatic Model Assessment was used to estimate insulin resistance (HOMA-IR) [28].

2.6. Serum fatty Acids Measurement

Serum samples were withdrawn at the baseline and 8 weeks after dietary intervention. The serum was dried under a gentle stream of nitrogen at room temperature and its residues were dissolved in 1 ml of NaOH and 0.5M of methanol. An internal standard consisting of 10 µl of nonadecanoic acid (C19:0, 5 mg/ml) was added. The temperature of the solution was held at 90°C for 1h; each sample was then cooled to room temperature and 1ml of boron trifluoride etherate in methanol (Sigma-Aldrich) was gently added. The samples were reincubated at 90°C for 30 minutes. The solutions were cooled and transferred to a test tube, where 2ml of deionized water and 4ml of hexane were added, after which the organic phase was separated. Each solution was dried again under a stream of nitrogen at room temperature and dissolved in 400µl of isooctane (Fisher Chemical). After that, an aliquot was injected into the chromatograph. Fatty acids were chromatographed on a 30m fused-silica column Zebron ZB-WAX (0.25 mm i.d.). The analysis was performed with an Agilent Technologies 7890A gas chromatograph equipped with a flame ionization detector Agilent Technologies 5975C. The column temperature was held at 50°C for 3 min, and subsequently increased in a stepwise fashion (10ºC/ min) to a plateau of 250°C, and then held for 3 min. The injection temperature was 220°C. Helium was used as carrier gas at 2ml/min. The results of measurements of FA are expressed in percentage (*i.e.* % serum FA).

2.7. Statistical Analysis

The anthropometric and metabolic data are expressed as the mean ± standard deviation. Data normality was tested using the Kolmogorov-Smirnov's test. Differences between groups were examined using the paired t-test. Non-parametric variables were transformed to logarithms. In order to examine the size effect of each anthropometric and metabolic indicator, we used the Cohen *d*. Statistical significance that was set at $p < 0.05$. Data analysis were performed with the Statistica 6410 for Windows (Statsoft, Tucson AZ) statistical software.

3. RESULTS

Forty-eight participants finished the intervention (73% women/ 27% men). The Mean age was 37 ± 4 years old, and BMI of 32 ± 2 kg/m^2. Significant increase of circulating adiponectin (30.4%, p = 0.003) and LCN2 (17.8%, p = 0.017) concentration were found at the end of the intervention. On average, the participants lost 3.7% (p < 0.0001) of their initial BMI and weight, 4% of waist circumference (p = 0.000002), 2.6% of hip circumference (p = 0.00002), 6.4% of fat mass (p = 0.000005), total cholesterol (6.3%, p = 0.011) and LDL (11.4%, p = 0.002) reduced, and triglycerides concentrations (8.8%, p = 0.06) nominally decreased (Table **1**).

Table 1. Anthropometric and metabolic characteristics of obese subjects before and after nutritional intervention.

Variable	Baseline n = 48	Final n = 48	–	–
	Mean ± SD	Mean ± SD	t Student	p
Weight (kg)	82 ± 11	79 ± 10	7.68	<0.0001
Waist circumference (cm)	102 ± 7.5	98 ± 7	5.42	<0.0001
Hip circumference (cm)	112 ± 7	109 ± 7	4.72	0.0001
BMI (kg/m^2)	32.4 ± 2	31 ± 2	7.77	<0.0001
Fat mass (%)	39 ± 7.5	36.5 ± 7	5.07	0.0001
Muscle mass (kg)	47.3 ± 8.5	48.1 ± 8	0.72	0.46
Systolic blood pressure (mmHg)	110 ± 13	108 ± 10	0.82	0.30
Diastolic blood pressure (mmHg)	76 ± 7	75 ± 8	0.45	0.12
Glucose (mmol/L)	4.8± 0.83	4.7 ± 0.83	0.25	0.56
Total cholesterol (mmol/L)	4.43 ± 0.56	4.15 ± 0.87	2.61	0.01
HDL-c(mmol/L)	1.49 ± 0.15	1.47 ± 0.18	1.26	0.28
LDL-c (mmol/L)	2.21 ± 0.54	1.96 ± 0.59	3.16	0.002
Triglycerides (mmol/L)*	2.26± 0.78	2.06 ± 0.53	1.92	0.060
LNC-2 (ng/mL)	76 ± 29	89.6 ± 34	-2.53	0.014
hsCRP (mg/L)*	4.0 ± 3.2	3.9 ± 3.2	0.16	0.31
Adiponectin (µg/mL)	9.2 ± 7.9	12 ± 10.2	-2.79	0.007
Insulin (pmol/L)*	61.2 ± 35.4	53.4 ± 31.8	1.34	0.45
HOMA-IR	2.1 ± 1.3	1.9 ± 1.2	0.72	0.27

BMI=Body mass index; HDL-c= High density lipoprotein; LDL-c=Low density lipoprotein; LNC-2= Lipocalin-2; hsCRP=High sensitivity C-reactive protein; HOMA-IR=Homeostasis model assessment insulin resistance. Differences between groups were examined using the paired t test. * = Differences between groups were examined using the Wilcoxon test. *logarithmic transformation.

There were changes in the circulating lipid profile, the percentage of Saturated Fatty Acids (SFA) in serum was decreased after the treatment (3%, p = 0.001), in particular, myristic acid (C:14) (8.6%, p = 0.007) and palmitic acid (C:16) (2.8%, p = 0.002). The percentage of palmitoleic acid also decreased (C:16:1) (8.8%, p = 0.0003). In contrast, the total PUFAs percentage (1.6%, p = 0.04) increased, particularly the Docosahexaenoic Acid (DHA) was increased marginally (C:22:6 ω3) (4.5%, p = 0.07) (Table **2**). In women, the percentage of oleic acid was significantly lower, but the percentage of DHA was higher than in men at baseline (Fig. **1**). these differences were not significant at the end of the intervention. We observed an unexpected decrease in energy intake of 288 Kcal/day (17%, p < 0.001), but there were no significant changes in the percentages of the macronutrient as compared with habitual consumption.

Table 2. Serum fatty acid levels in obese subjects before and after nutritional intervention.

Variable	Baseline n = 48	Final n = 48	–	–
	Mean ± SD	Mean ± SD	t Student	p
% Fatty acid in blood				
C:12:0*	0.08 ± 0.04	0.07 ± 0.04	1.88	0.059
C:14:0	1.0 ± 0.2	0.86 ± 0.2	2.84	0.007
C:16:0	21.2 ± 1.4	20.6 ± 1.4	3.30	0.002
C:16:1 (ω-9)	3.4 ± 0.7	3.1 ± 0.5	3.87	0.0003
C:18:0	7.5 ± 0.8	7.4 ± 0.8	1.28	0.20
C:18:1 (ω-9)	23.2 ± 1.8	23.6 ± 2.2	-1.25	0.21
C:18:2 (ω-6)	26.8 ± 2.8	27.4 ± 2.8	-1.60	0.12

(Table 2) contd.....

Variable	Baseline n = 48	Final n = 48	–	–
	Mean ± SD	Mean ± SD	t Student	p
C:18:3 (ω-3)	1.1 ± 0.2	1.1 ± 0.2	0.90	0.37
C:20:0	0.06 ± 0.02	0.07 ± 0.02	-1.81	0.08
C:20:1 (ω-9)	0.47 ± 0.1	0.49 ± 0.1	1.26	0.21
C:20:3 (ω-6)	2.1 ± 0.4	1.9 ± 0.4	1.44	0.15
C:20:4 (ω-6)	6.8 ± 1.2	6.9 ± 1.3	-1.31	0.15
C:20:5 (ω-3)	3.7 ± 2.9	3.9 ± 3	-1.89	0.066
C:22:6 (ω-3)	2.2 ± 0.6	2.3 ± 0.6	-1.81	0.076
SFA	29.9 ± 1.7	29 ± 1.8	3.55	0.001
MUFAs	27.2 ± 2.1	27.1 ± 1.9	0.18	0.86
PUFAs	43 ± 3	43.7 ± 3	-2.11	0.040
• ω-6	35.8 ± 3	36.3 ± 3	0.58	0.56
• ω-3	7.1 ± 2.8	7.3 ± 3.1	-0.944	0.35

SFA=Saturated fatty acids; MUFAs=Monounsaturated fatty acids; PUFAs=Polyunsaturated fatty acids, ω-3= Polyunsaturated fatty acids omega-3; ω-6=Polyunsaturated fatty acids omega-6. Differences between groups were examined using the paired t- test. *logarithmic transformation.

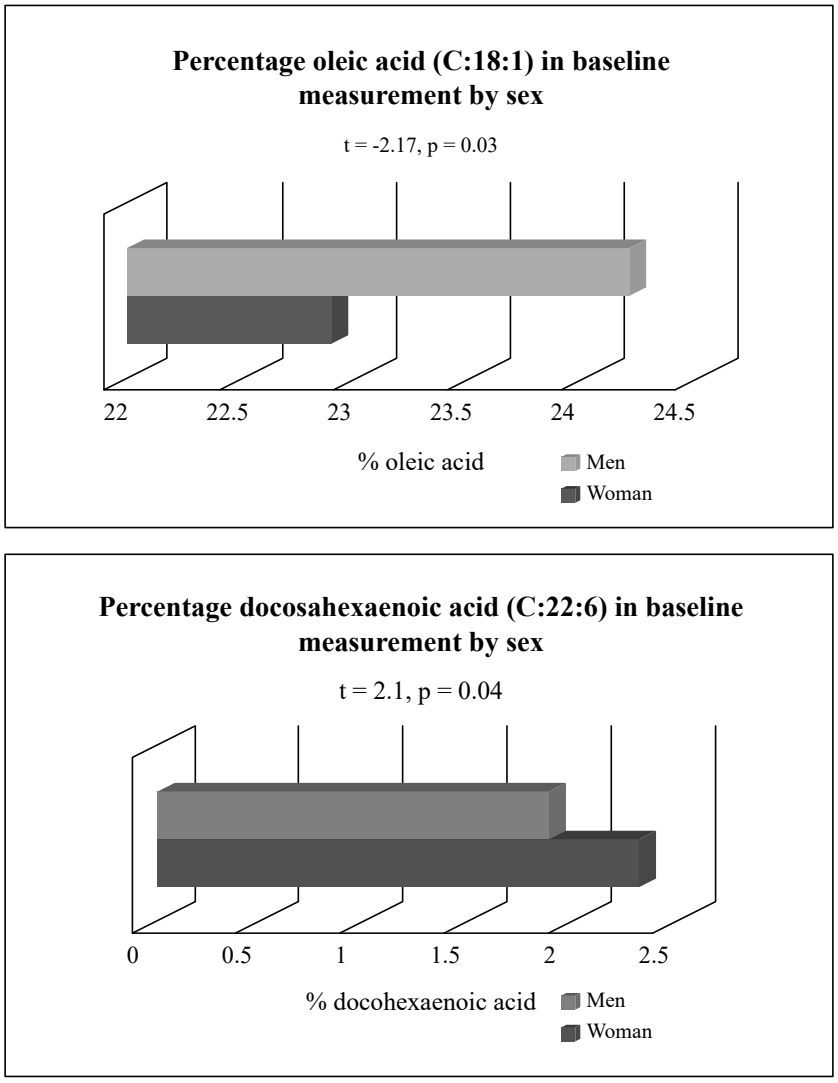

Fig. (1). Percentage of oleic acid and DHA between men and women.

To know the effect of the intervention on each variable, we calculated the effect size, which was greater in waist circumference, weight, hip circumference, and fat mass. For BMI, adiponectin, LCN2, SFA, C:16:0, C:16:1, the effect

size was moderate (Table **3**). In the baseline, positive correlations of lauric and stearic acids with hsCRP, and oleic acid with triglycerides were found. Also, negative correlations of the arachidonic acid with BMI and triglycerides, and DHA with BMI were found. At the end of the intervention, a positive correlation of oleic acid with triglycerides and a negative correlation of arachidonic acid with BMI were maintained. In addition, dihomo gamma linoleic acid (20:3 ω-6) positively correlated with LCN2 concentrations. All correlation analyses were adjusted for sex (Table **4**).

Table 3. Effect size analysis after intervention.

Variable	Cohen's d test	Variable	Cohen's d test
Weight (kg)	1.13	C:14:0	0.19
Waist circumference (cm)	1.19	C:16:0	0.50
Hip circumference (cm)	0.77	C:16:1 (ω-9)	0.54
BMI (kg/m^2)	0.67	C:18:0	0.15
Fat mass (%)	0.74	C:18:1 (ω-9)	0.19
Systolic blood pressure (mmHg)	0.11	C:18:2 (ω-6)	0.30
Diastolic blood pressure (mmHg)	0.19	C:18:3 (ω-3)	0.04
Glucose (mmol/L)	0.08	C:20:1 (ω-9)	0.30
Total cholesterol (mmol/L)	0.32	C:20:3 (ω-6)	0.11
HDL-c (mmol/L)	0.13	C:20:4 (ω-6)	0.26
LDL-c (mmol/L)	0.46	C:20:5 (ω-3)	0.17
Triglycerides (mmol/L)	0.11	C:22:6 (ω-3)	0.37
LNC-2 (ng/mL)	0.49	SFA	0.55
hsCPR (mg/L)	0.15	MUFAs	0.01
Adiponectin (μg/mL)	0.51	PUFAs	0.36
Insulin (pmol/L)	0.11	ω-6	0.21
HOMA-IR	0.11	ω-3	0.21
C:12:0	0.12		

BMI=Body mass index; HDL-c= High density lipoprotein; LDL-c=Low density lipoprotein; LNC-2= Lipocalin-2; hsCRP=High sensitivity C-reactive protein; HOMA-IR=Homeostasis model assessment insulin resistance; SFA= Saturated fatty acids; MUFAs= Monounsaturated fatty acids; PUFAs= Polyunsaturated fatty acids.

4. DISCUSSION

In this study, circulating adiponectin levels increased by 30% in obese subjects supplemented with a daily intake of approximately 2g of ω-3 and 11g of ω-6 fatty acids for eight weeks from two sources: 15 g of walnuts and 15 g of almonds. In conformity with our results, previous reports showed an increase of 6.4% in circulating adiponectin after short-term walnut 48 g/d consumption (four days) [19], and an increase of 23% in circulating concentrations of adiponectin can be observed in women who most closely followed a Mediterranean diet [29]. The circulating LCN2 concentrations also were increased by 17.9% after the intervention. The LCN2 has been reported as a novel regulator of brown adipose tissue activation by modulating the adrenergic independent p38MAPK-PGC-1α-UCP1 pathway [30]. It has also been reported that saturated fats might contribute to the circulating LCN2 concentrations in obese patients with insulin resistance [31]. The evidence and our results suggest that the improvement observed in the metabolic state may be due to, or at least, in part to PPARγ activation and adiponectin expression promoted by the increase in circulating LCN2 concentrations.

In our work, we found a decrease in %SFA, particularly C:14, C:16, and C:16:1. Similar data have previously been reported after walnuts consumption for four weeks.[6] It is possible that PUFAs contained in walnuts and almonds might suppress the activity of sterol regulatory element-binding transcription factor 1 (SREBP1c), decreasing the synthesis of fatty acids as described [32].

Table 4. Correlation of basal and final fatty acids concentrations with adiposity and metabolic markers in obese subjects (n=48)

–		Basal		Final	
Variables	–	**r**	**p**	**r**	**p**
SFA	–	–	–	–	–
Total SFA	hsCRP*	0.52	0.003	0.20	0.22

(Table 4) contd.....

–		Basal		Final	
Variables	**–**	**r**	**p**	**r**	**p**
C:12*	hsCPR*	0.43	0.017	0.17	0.30
C:14	Triglycerides*	0. 682	<0.001	-0.63	0.70
C:18	hsCRP*	0.45	0.013	0.22	0.17
MUFAs					
C:18:1	Triglycerides*	0.43	0.018	0.41	0.009
PUFAs					
C:20:3 ω-6	LNC-2	-0.13	0.43	0.40	0.011
C:20:4 ω-6	BMI	-0.41	0.010	-0.42	0.008
	Triglycerides*	-0.64	<0.001	-0.13	0.41
C:22:6 ω-3	BMI	-0.35	0.030	0.51	0.76

BMI=Body mass index; LNC-2= Lipocalin-2; hsCRP=High sensitivity C-reactive protein; MUFAs=Monounsaturated fatty acids; PUFAs=Polyunsaturated fatty acids; SFA=Saturated fatty acids. *logarithmic transformation. Relationships between variables were examined using the Pearson correlation coefficient.

In addition, we found a significant decrease in body weight, fat mass, hip and waist circumference, total cholesterol, and LDL concentrations in obese subjects after the intervention. In concordance with our results, various several studies have shown a significant decrease in weight and BMI with the consumption of 50 or 56g of almonds for 3 or 6 months in overweight and obese individuals [4, 5]. Another report showed a decrease in body weight of 18 kg after a low-calorie diet enriched with 84g/d of almonds for a period of 24 weeks in overweight and obese individuals [33]. We indicated a combination of 15 g/d of walnuts and 15g/d almonds, noting that weight loss is significant with a lower consumption for a shorter period. However, previous studies with a diet including almonds have shown no significant effect on body weight, BMI, as compared with control [34, 35].

We also found a decrease of 6.3% in the total cholesterol and 11.3% in the LDL concentrations. A similar decrease of (6%) using 36g of walnuts for 6 weeks was found in another study [36]. Tapsell et al. used 30g of walnuts for 6 months to achieve a 10% reduction in LDL in type 2 diabetic patients [10]. Another study that used 60 g/d almonds for 12 weeks resulted in a significant reduction of the body fat, total cholesterol and LDL concentrations in type 2 diabetic patients [12]. It has also been reported that endothelial function, total and LDL cholesterol improved significantly, but BMI, body fat percent, visceral fat, and fasting glucose did not change after consumption of 56 g/d of walnuts [37].

Through the calculation of size effect, we could identify which indicator was more modified for the effect of the intervention. The waist circumference, weight, hip circumference, and fat mass were indicators that experimented the majors effects. Only one report showed a greater weight-loss after a hypocaloric almonds-enriched diet [4]. However, it did not evaluate the effect size on other indicators as BMI, adiponectin and LCN2 levels.

The walnut contains a variety of bioactive compounds, such as vitamin E and polyphenols that possess antioxidant and anti-inflammatory activity. Almonds are rich in MUFAs, fiber, α-tocopherol, magnesium and copper that may contribute to hypocholesterolemic benefits. Therefore, it is possible that in addition to the PUFAs, other components to be able also act to bring about the anthropometric and metabolic changes reported in this work. The lack of a control group may be a weakness; however, in a previous report done in 30 subjects we found several miRNAs modified after the intake of walnuts and almonds, including decrease of miR-328, miR-330-3p, miR-221, and miR-125a-5p, and increase mir-192, miR-486-5p, miR-19b, miR-106a, miR769-5p, miR-130b, and miR-18a. In addition miR-106a variations in plasma positively correlated with the changes in PUFAs [38]. In addition, previous studies have reported that total cholesterol and LDL concentration in adults that complied with the walnut diet were lower than in those who followed diet control [11, 39]. The consumption of almonds (50-100g/d) and walnuts (40-84g/d) also decreased the total cholesterol and LDL concentrations, compared with subjects consuming controlled diet [40]. Weight, BMI, waist circumference and total cholesterol decreased significantly in the almonds group compared to the nut-free group [5].

CONCLUSION

In conclusion, significant increases of 30% in adiponectin and LCN2 (17.9%) concentrations were found. Additionally, a significant decrease in all adiposity measures (weight, waist circumference, hip circumference, BMI and fat mass), total cholesterol and LDL concentrations after the intervention was observed. The SFA percentage significantly decreased, in particular, the C:14, C:16, and the MUFAs C:16:1. The total PUFAs percentage significantly increased, and particularly the percentages of the eicosapentaenoic acid (EPA, C:20:5 ω-3) and DHA C:22:6 ω-3 marginally increased. The effect size was large for all adiposity measures, except for BMI as well as for adiponectin

which was moderate. Our data show that the consumption of almonds and walnuts for a short-time may improve the metabolic inflammation and measure adiposity.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was approved by the Institutional Ethics Committee of the Universidad de Guanajuato.

HUMAN AND ANIMAL RIGHTS

No Animals were used in this study. All human research procedures followed were in accordance with the ethical standards laid down in the Declaration of Helsinki 1983 and in agreement with the Good Clinical Practice guidelines.

CONSENT FOR PUBLICATION

All participants submitted their written informed consent to participate in the study.

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CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest.

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Declared none.

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