Nutrición Hospitalaria

Nutr Hosp. 2015;31(6):2561-2570
ISSN 0212-1611 • CODEN NUHOEQ
S.V.R. 318

Original / *Ancianos*

Impact of the consumption of a rich diet in butter and it replacement for a rich diet in extra virgin olive oil on anthropometric, metabolic and lipid profile in postmenopausal women

Hazel Ester Anderson-Vasquez[1,2,4], Pablo Pérez- Martínez[4,5], Pablo Ortega Fernández[1,3] y Carmina Wanden-Berghe[6,7]

[1]*Universidad del Zulia,* [2]*Laboratorio de Investigación y Desarrollo en Nutrición.* [3]*Instituto de Investigaciones Biológicas, Venezuela.* [4]*Universidad de Córdoba, Doctorado en Nutrición y Metabolismo.* [5]*Unidad de Lípidos y Ateroesclerosis. IMIBIC/Hospital Universitario Reina Sofia, Universidad de Córdoba, and CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, España.* [6]*Hospital General Universitario de Alicante.* [7]*Universidad CEU Cardenal Herrera (Elche), España.*

Abstract

Objective: **to analyze the impact of the substitution of a rich diet in saturated fats with a rich diet in monounsaturated fats on anthropometric, metabolic and lipid profile in postmenopausal women.**

Material and methods: **a prospective, longitudinal and comparative study where 18 postmenopausal women participated in two periods of dietary intervention of 28 days each one: 1) (SAT diet) consumed butter. Caloric formula (CF) = 15% protein, 38% fat. [20% saturated fat (SFA), 12% monounsaturated fat (MUFA) and 47% carbohydrates and 6% polyunsaturated (PUFA)]. b) Period MONO: with extra virgin olive oil (EVOO). CF = 15% protein, 38% fat (<10% SFA, 22% PUFA and 6% MUFA) and 47% carbohydrates. Size and body composition, glucose, insulin, HOMA, TC, HDL-C, LDL-C, VLDL-C, TG, TC/HDL-C, LDL-C/HDL-C, TG/HDL-C and non-HDL-C/HDL.C were measured; dietary Anamnesis/24 hours, daily food record. ANOVA and Bonferroni statistical analysis (SPSS 20) was applied.**

Results: **the age was 56 ± 5 years, BMI 29.8 ± 3.1 kg/m², waist circumference: 93.2 ± 10.1 cm, waist/hip ratio: 0.86 ± 0.14, waist/height: 0.59 ± 0.06 and 38.6 ± 4% body fat (NS). Lipid profile: SAT diet increased TC (p <0.001), LDL-C (p <0.002) and non HDL-Cholesterol (p <0.000), HDL-C increased in MONO diet (p <0.000). SAT diet: TC/HDL-c ratio, Non col HDL-c/HDL-c, LDL-c/HDL-c (p <0.000) and TG/HDL-c (p <0.000). In MONO diet decreased TC/HDL-c (p <0.015) and TG/HDL-c (p <0.016).**

Correspondence: Carmina Wanden-Berghe
Universidad CEU Cardenal Herrera. Elche.
Hospital General Universitario de Alicante
E-mail: carminaw@telefonica.net – cwanden.el@uch.ceu.es

Recibido: 28-I-15.
1.ª Revisión: 3-III-15.
Aceptado: 7-III-2015.

IMPACTO DEL CONSUMO DE UNA DIETA RICA EN MANTEQUILLA Y SU REEMPLAZO POR UNA DIETA RICA EN ACEITE DE OLIVA VIRGEN EXTRA SOBRE EL PERFIL ANTROPOMÉTRICO, METABÓLICO Y LIPÍDICO EN MUJERES POSTMENOPÁUSICAS

Resumen

Objetivo: **analizar el impacto de la sustitución de una dieta rica en grasas saturadas por una dieta rica en grasas monoinsaturadas sobre el perfil antropométrico, metabólico y lipídico en mujeres postmenopáusicas.**

Material y método: **estudio prospectivo, longitudinal y comparativo en el que 18 mujeres postmenopáusicas participaron en dos períodos de intervención dietética de 28 días cada uno: 1) (dieta SAT) consumieron mantequilla. Fórmula calórica (FC) = 15% de proteínas, 38% grasas. [20% grasas saturadas (AGS), 12% grasas monoinsaturadas (AGM) y 47% carbohidratos y 6% poliinsaturadas (AGPI)]. 2) Periodo MONO: con aceite de oliva virgen extra (AOVE). Fórmula calórica = 15% de proteínas, 38% grasas (<10% AGS, 22% AGM y 6% AGPI) y 47% carbohidratos. Se midieron dimensión y composición corporal, glicemia, insulina, HOMA, CT, HDL-C, LDL-C, VLDLC, TG, CT/HDL-C, LDL-C/HDL-C/ TG/HDL-C y CT no HDL-C/HDLC. Anamnesis dietética/24 horas, registro diario de alimentos. Para el análisis estadístico se aplicó ANOVA y BONFERRONI (SPSS 20).**

Resultados: **la edad fue de 56 ± 5 años, IMC: 29,8 ± 3,1 kg/m², circunferencia de cintura (CCi): 93,2 ± 10,1 cm, circunferencia cintura/cadera (IC/C): 0,86 ± 0,14, relación cintura/estatura (ICE): 0,59 ± 0,06 y 38,6 ± 4 % de grasa corporal (%GC) (NS). CCi, Dieta SAT al Inicio: 55,6% = RCV, final = 66,7%, dieta MONO = 55,6%. Perfil lipídico: dieta SAT aumentaron CT (p<0,001), LDL-C (p<0,002) y colesterol NO HDL-c (p<0,000), HDL-C aumentó en dieta MONO (p<0,000). Dieta SAT: Rel. CT/HDL-c, Col No HDL-c/HDL-c, LDL-c/HDL-c (p<0,000) y TG/HDL-c (p<0,000). En dieta MONO disminuyeron CT/HDL-c (p<0,015) y TG/HDL-c (p<0,016).**

Conclusions: **the SAT diet increased cardiovascular risk, while the MONO diet decreased the risk to develop the metabolic syndrome components and choronary heart disease.**

(*Nutr Hosp*. 2015;31:2561-2570)

DOI:10.3305/nh.2015.31.6.8732

Keywords: *Postmenopause. Cholesterol dietary. Olive oil. Butter. Diet therapy.*

Conclusiones: **la dieta SAT aumentó el riesgo cardiovascular, mientras que la dieta MONO disminuyó el riesgo de desarrollar los componentes del síndrome metabólico y enfermedades coronarias.**

(*Nutr Hosp*. 2015;31:2561-2570)

DOI:10.3305/nh.2015.31.6.8732

Palabras clave: *Menopausia. Perfil lipídico. Aceite de oliva. Mantequilla. Intervención dietética.*

Abbreviations:
SFA: Saturated fatty acids.
MUFA: Monounsaturated fatty acids.
PUFA: polyunsaturated fatty acids.
EVOO: extra virgin olive oil.
WC: Waist circumference.
TC: Total cholesterol.
TC/HDL-C: Total cholesterol/HDL-C ratio.
Col No HDL: Colesterol no HDL.
DIET SAT: Diet with butter.
DIET MONO: Extra virgin olive oil diet.
CVD: Cardiovascular Diseases.
CVRF: Cardiovascular risk factor.
CVR: Cardiovascular risk.
HDL-C: High density lipoproteins.
BMI: Body Mass Index.
W/Hip I: Waist Hip Index.
W/Height I: Waist Height index.
LDL-C/HDL-C: LDL-C/HDL-C ratio.
LDL-C: Low Density Lipoproteins.
PMW: Postmenopausal Women.
No HDL-C/HDL/C: no HDL-C/HDL-C Ratio.
TG: Triglycerydes.
TG/HDL-C: Triglycerydes/HDL-C Ratio.
VLDL-C: Very Low Density level Lipoproteins.

Introduction

Menopause is the permanent suspension of menstrual function caused by reduced estrogen secretion, due to the loss of follicular function[1] which brings the end of the reproductive life of women, so bodily changes and biochemical characteristics occur that bring on a number of risk factors associated with obesity abdominal and insulina resistance[2]; which includes hypertension, diabetes and dyslipidemia that increase cardiovascular disease favoring higher mortality in this cycle of life[3]. Early, natural or surgical menopause is associated with endothelial dysfunction and increases 3 times the risk of coronaria artery disease[4].

In this period of life, decreased circulating levels of estrogen brings about changes in the size and body composition, favoring weight gain of adipose tissue and increased fat distribution in the abdominal area[2]. Currently there are various models, methods and techniques for anthropometric assessment, where it is important to obtain information in determining the nutritional status as well as their influence on the cardiometabolic risk[5].

On the one hand, another important aspect is the lipid profile[6-8]. Postmenopausal women have a lipid profile characterized by high concentrations of total cholesterol, LDL-C and TG and low HDL-C called dyslipidemia atherogenic[8]. It is considered that the Castelli atherogenic index and LDL-C / HDL-C ratio are good predictors of the clinical benefit degree on lipids intervention, although studies have been conducted mainly in pharmacological therapies[9]. Acevedo M et al showed in their study that the TC / HDL-C ratio and cholesterol no-HDL-C were the best associated, among different lipoprotein markers, to subclinical atherosclerosis. The HDL cholesterol proved to be the best protector of carotid thickness[10].

In Venezuela women over 45 years accounted for 17.05% of the total female population for 1995 and projection for 2035 is 35.76%, with a life expectancy of 74.73 years old. The age for menopause comprises between 48.7 ± 4.6 years, this indicates that consequent alterations of endocrinological, biological and clinical processes of this gonadal cessation, will accompany them for a third of their lives[11]. This aspect reflects the great importance of the study and management of environmental factors that influence their lifestyle, specifically food. However, few studies of dietary intervention on lipids in this cycle of life is conducted in the region.

From the nutrition point of view, epidemiological studies have highlighted the role of vegetable fats in the cardiovascular disease (CVD), neurodegenerative and cancer prevention[12,13]. Lopez et al[14] analyzed the association between dietary patterns and the presence of cardiovascular risk factors (CVR) in the National Health and Nutrition Examination Survey (NHANES) 2001-2002, These authors evaluated 1,313 women, and founded a significant association between dietary patterns and increased CVR. Cardiovascular protection decreases with age after menopausia[15], so it is possible that dietary intervention of monounsaturated fatty acids instead of saturated fatty acids in healthy diet, can induce different effects on metabolic risk factors in postmenopausal women[16].

When it comes to foods rich in saturated fatty acids (SFA) and its effect on CVR factors, it is butter where palmitic and stearic fatty acids predominate. These two SFA have the greatest potential impact on LDL-C[17]. The Health of Nurses, after tracking 80,082 women for 14 years, has estimated that an increase of 2% of caloric intake of SFA produces a 28% increased risk of coronary heart disease [18,19].

On the other hand, aliments rich in monounsaturated fats (MFA), the main representative being extra virgin olive oil (EVOO), which has a higher anti-atherogenic capacity, improves metabolic syndrome, facilitates glucose control, blood pressure and has beneficial effects on platelet function, thrombogenesis and fibrinolisis[20].

According to the above stated, the present research aimed to determine the impact of two dietary interventions in order to measure effects on anthropometric, metabolic and lipid profile before and after consumption of a diet high in saturated fat (diet SAT), compared to its replacement on a diet rich in monounsaturated fatty acids (diet MONO) supplied to a group of postmenopausal women, to generate dietary interventions aimed at improving the quality of life of women during this cycle of life.

Materials and methods

Study design

It is a prospective, longitudinal, experimental and comparative research. It consisted of 18 postmenopausal women (PMW) selected according to the inclusion criteria and subjected to two dietary interventions. Menopause was defined according to published criteria[1,6]. In the pre and post intervention periods, a menu was provided with a similar fat composition to be consumed in each diet. The Shaw et all[21] criteria for dietary intervention protocol was used.

Study population

From a universe of 100 PMW attending the Outpatient Nutrition Clinic, from March 2011 to March 2012; after explanation of the protocol, 18 patients who met the following criteria were selected: a) Between 50 and 65 years old. b) Being postmenopausal. c) Not having evidence of chronic disease, including liver, kidney, thyroid or heart dysfunction. d) Not following a low calorie diet. e) Not receiving drugs that could affect lipid metabolism. f) Has not received hormone replacement therapy; and g) Agreeing in writing to participate in the study. This research was approved by the Ethics Committee of the Laboratory for Research and Development in Nutrition, School of Nutrition and Dietetics, Faculty of Medicine, University of Zulia.

Methodology

Anthropometric Assessment

The measurements were performed by a specialist in Clinical Nutrition, previously trained and standardized in anthropometry. Women were assessed only with the minimum of clothes, no shoes or accessories. The technical error of measurement was within the range of values considered adequate: weight: 0.00 g, size: 0,01cm and triceps skinfold (PTr) 0.1 mm. Skinfolds were measured with a Lange® grease caliper (Cambridge Scientific Instruments, Cambridge, MD) with a measuring range of 0-60 mm, graduation 0.3 mm and a constant pressure of 10 g/mm^2. Scales INNER-Scan Body Composition Monitor by Tanita, Ironman TM-BC554-3901 was used to determine body composition. For perimeters a 150 cm long anthropometric measuring tape was used. The measures of size and body composition[22] were taken according to the criteria of the WHO[23] .

Biochemical Evaluation

The sampling was performed at the Laboratory for Research and Development in Nutrition, after an overnight fast of 12 hours in a conditioned room where patients remained seated and relaxed. Then it was proceeded to attain the right antecubital vein, collecting 5 ml of blood in dry tube, samples were taken at baseline and 28 days after that at the end of each period; then it was proceeded to determine the levels of glucose, insulin, TG, TC, HDL-C, LDL-C, VLDL-C.

Insulin was determined by ELISA (DRG Instruments GmbH, Germany, Division of DRG International, Inc.) and blood glucose by the enzymatic colorimetric method of Glucose Oxidase (Human Gesellschaft für Biochemica und Diagnostica mbh). With both data it was proceeded to calculate HOMA-IR (Homeostasis Model Assessment) index, using the parameters of insulin and basal glucose following formula: IR (HOMA-IR) = Insulin fasting (uU / mL) × fasting glucose (mmol /L)/22.5[24].

Different concentrations of lipids were analyzed using commercial colorimetric methods (Human Gesells Chaftfür Biochemical und Diagnostica mbh). LDL-C cholesterol was calculated using the Friedewald formula: LDL-C=CT (HDL-C + TG/5)[24]. It was used as a reference for the lipid profile ATP III[25] criteria. Regarding lipoprotein[9] ratios were determined as follows:

– For the atherogenic index Castelli[26] equals relation to TC/HDLC-C, the following were taken as cutoff points: <4.5 lower risk, from 4.5 to 7 moderate risk and >7 high risk.

- For the LDL-C/HDL-C index <3 was taken as the cutoff point.
- For the relation CHOLESTEROL No HDL/HDL-c, <4.5 was taken as the cutoff.
- For the TG/HDL-C ratio, > = 3.0 was used as the cutoff.

Dietary Assessment

During the initial dietary assessment[27] was performed by applying the 24-hour recall[28] in a standardized manner to identify the habits and preferences of the participants taking into account two nonconsecutive days (usual day and not usual), emphasis was placed on the amounts and types of food as well as specials preparations, recording measurements and ingredients used. During the execution of the study to assess diets compliance, their homes were visited and 3 phone calls were made weekly. To calculate the contribution of energy and nutrients a computer program with data from the Table of Food Composition of Venezuela[29] was used.

Dietary intervention

All volunteers participated in two dietary intervention periods of 28 days each one, isocaloric in relation to their usual previous consumption in order to keep their weight stable. There was no washout period. At the beginning and end of each dietary intervention period the respective biochemical tests were performed. PMW consumed the SAT diet which comprises 15% protein, 47% carbohydrate (CHO) and 38% fat (20% SFA, 12% MUFA, and PUFA 6%) and the MONO diet which comprised 15% protein , 47% CHO, 38% fats (<10% SFA, 22% MUFA and PUFA 6%). Cholesterol intake was less than 300 mg/day for the two periods of dietary intervention. With the results of dietary assessment it was proceeded to calculate the average caloric formula and the drafting of the regimes used in different periods according to the type of fat used.

The diet was made based on 7 rotating menus previously established with an isocaloric formula which providing for the SAT DIET 1926 kcal, 80.7 g fat; while where provided for the MONO diet 1951 kcal and 82.7 g of fat. Both regimens provided 79 g protein and 222 g carbohydrate. These diets were consumed by patients at home. The participants in the first stage received the SAT diet where they consumed 50 g of butter a day as a source of saturated fat, which they smeared on white bread or corn products; subsequently, they received the OLIVA diet, which included 50 cc of EVOO, which was consumed in soup (added raw) or tablespoons.

Statistical analysis

For the statistical treatment of the data, the statistical program package SPSS (Statistical Package for Social Sciences) version 17,0[29] was used. Descriptive Statistics were used to represent the results as mean ± standard deviation and change percentages for the control diet in tables. The Kolmogorov – Smirnov test was used to compare the normal distribution of the variables. The Levene test was used in the contrast of the homogeneity test. To determine the effect of the different diets on the studied variables, the analysis of variance (ANOVA) was used in comparing arithmetic means and post hoc, Bonferroni was applied. $p < 0.05$ was considered as the significant statistical value.

Results

Women studied were of 56±5 years old, with a range between 50 and 64. Table 1 shows the anthropometric variables of body dimension, it appears that at the beginning of the SAT diet, BMI was 29.8±3.1 kg/m^2, waist circumference (WC), 93.2±10.1 cm, waist/hip circumference (W/HC) of 0.86±0.14 and waist/height relationship (W/HR) of 0.59±0.06. Also, regarding the body composition, the subjects presented a body fat percentage of 38.6±4%. There were no significant differences in anthropometric measurements between the two diets. However, while analyzing the effects of the anthropometric indicators of cardiovascular disease risk, it was found that the cardiovascular disease risk had increased during the consumption of the SAT diet for all indicators; which then decreased during the period of the MONO diet consumption.

In table 3 a metabolic and lipidic profile is presented for both periods. Regarding the metabolic profile, glycemia and the HOMA-IR index significantly increased during the SAT diet, while not presenting any modifications at the end of the MONO period. Regarding the lipidic profile, it was observed that during the SAT diet there was a significant increase in TC ($p < 0.001$), LDL-C ($p < 0.002$) and observed the no cholesterol HDL-c ($p < 0.000$). Additionally, no significant changes were observed in the VLDL-C, nor TG on any of the dietary interventions. Nevertheless, while percentage comparing it was found that during the MONO period the TG and VLDL-C decreased by 7.4% and 7.2%, respectively.

When it comes to the HDL-C, it was observed that in the MONO diet the response was significant ($p < 0.000$). Another aspect evaluated in table 3 were the lipoproteics indicators where it is evident that the relation CT/HDL-C and the relation Col No HDL-C/HDL-C increased the cardiovascular disease risk in the SAT diet ($p < 0.000$); the same aspect decreased in the MONO diet ($p < 0.015$ and $p < 0.016$ respectively). The relation LDL-C/HDL-C increased during the SAT diet ($p < 0.000$).

Table I
Comparison of anthropometric variables of participants body size and composition in the two periods of dietary intervention.

Antropometric Variable	*Dietary Intervention Type*		
	Diet SAT		*Diet MONO*
	BEGINNING (n=18)	*ENDING (n=18)*	*(n=18)*
Body size			
Body weight (kg)	72.4±10.3	72.2±10.0	71.9±10.2
BMI (kg/m^2)	29.8±3.1	29.7±2.9	29.7±2.9
Torax (cm)	103.1±8.9	102.8±9.5	101.7±9.0
Waist circumference (cm)	93.2±10.1	93.3±9.5	93.2±10.1
Hip circumference (cm)	108.0±7.2	107.7±6.3	106.0±6.7
Waist/Hip Circumference	0.86±0.14	0.86±0.15	0.87±0.15
Waist/Height ratio	0.59±0.06	0.59±0.06	0.58±0.05
Thigh circumference (cm)	106.6±5.2	106.6±5.4	106.7±4.9
Media right arm/ Circunferemce (cm)	32.6±3.1	32.6±2.8	32.2±2.8
Right wrist (cm)	16.7±1.3	16.7±1.1	16.5±1.0
Body built	9.2±0.54	9.1±0.46	9.23±0.42
Right thigh circumference (cm)	63.1±3.6	63.7±3.5	62.1±2.5
Right calf circumference (cm)	37.9±2.1	37.8±2.2	37.2±2.4
Body composition			
Biceps skinfold (mm)	11.8±3.2	11.3±2.7	11.2±2.6
Triceps skinfold (mm)	17.1±4.6	19.0±3.0	18.9±3.1
Subscapular skinfold (mm)	22.0±5.2	20.3±4.6	20.3±3.3
Suprailiac skinfold (mm)	26.7±6.4	27.1±3.2	25.4±3.2
Abdominal skinfold (mm)	27.3±5.5	27.0±4.6	25.4±4.3
Total body water (%)	42.7±2.4	42.3±2.3	41.4±2.0
Fat free area (kg)	41.8±3.8	41.2±3.7	40.6±3.8
Bone tissue (kg)	2.2±0.1	2.2±0.2	2.1±0.2
Body fat (%)	38.6±4.0	39.2±4.0	40.4±3.5
Body fat (kg)	28.2±6.4	28.5±6.2	29.5±5.8
Visceral fat (%)	9.3±1.9	9.5±1.9	9.8±1.8
Visceral fat (kg)	6.8±2.1	6.9±2.1	7.1±2.1

Values represent the mean ± D. Diet SAT: Butter diet. Diet MONO: Extra virgin olive oil diet.

Table IV represents percentage ratio comparison in regards of the lipid profile of both types of diets. Regarding the cholesterol, it was observed that after the ingestion of butter the cardiovascular disease risk increased in 77.8% of the patients, said risk decreased after the consumption of olive oil in 61.1% of the patients. Table 4 also shows that the levels of LDL-C, after the consumption of butter, increased in all of the patients, while decreasing during the consumption of EVOO, and optimal values were observed in 11.1% of the patients.

For a more specific analysis the percentage evaluation of the lipoprotein ratios of cardiovascular disease risk in every patient was considered. In that sense, table 4 showing the CT/HDL-C relation, it is observed that during the dietary intervention with butter, the risk of cardiovascular disease increased in all patients; while decreasing during the extra virgin olive oil intervention, in such a way that 33.3% presented low risk. In the same order of ideas, table 4 shows similar results for the LDL-C/HDL-C relation and the TG/HDL-C. Furthermore, it is shown that in the NO cholesterol

Table II
Comparison of anthropometric indicators of cardiovascular risk in the two periods of dietary intervention.

Anthropometric Indicator	*Diet SAT*		*Diet MONO*
	BEGINNING (n=18)	*ENDING (n=18)*	*(n=18)*
Body Mass Index			
Normal	5 (27.8%)	4 (22.2%)	4 (22.2%)
Overweight	3 (16.7%)	5 (27.8%)	5 (27.8%)
Obesity	10 (55.6%)	9 (50.0%)	9 (50.0%)
Waist Circunference			
Without risk	8(44.4%)	6 (33.3%)	8(44.4%)
With risk	10 (55.6%)	12 (66.7%)	10 (55.6%)
Waist/Hip Index			
Low risk	2 (11.1%)	1 (5.6%)	2 (11.1%)
Moderated risk	8(44.4%)	10 (55.6%)	8(44.4%)
High risk	8(44.4%)	7 (38.9%)	8(44.4%)
Waist/Height Index			
Without risk	2 (11.1%)	0 (0.0%)	1 (5.6%)
With risk	16 (88.9%)	18 (100.0%)	17 (94.4%)

Diet SAT= butter diet
Diet MONO= extra virgin olive oil diet

HDL-C/HDL-C relation, during the SAT period, the cardiovascular disease risk increased for 83.3% of the patients to high risk; however, these values improved in the MONO diet for 66.7% of the subjects.

Discussion

Despite major advances in the pharmacological management of dyslipidemia in recent decades, nutrition continues to play a key role in primary and secondary prevention of CVD. The main objective of this study was to determine the effect of a dietary intervention that included the replacement of saturated fatty acids represented by butter, as base grease of the typical Western diet based on monounsaturated fatty acids using extra virgin olive oil, as fat source of the Mediterranean diet, over atherogenic dyslipidemia of PMW; obtaining as a result that the SAT diet severely increased cardiovascular disease risk, which improved by receiving the MONO diet.

During menopause, cardiovascular risk factors are of greater impact due to hormonal changes, age, weight gain, inactivity and altered lipidic profile[3]. Women in this study were overweight and fat distribution of android type, with high cardiovascular metabolic risk according to the WC, W/Hip and W/Height index, which can foster the development of insulin resistance and its clinical consequences: carbohydrate intolerance and type 2 diabetes, hypertension, dyslipidemia and coronary disease[1,2].

This investigation the studied patients were overweight with abdominal fat distribution. Similar results were reported by Meertens et al.[30] who evaluated 129 PMW aged 52.52±5.48 years old, they found a BMI of 28.07±5.78 kg/m^2, and WC of 89.16±11.13 cm with similar cardiometabolic risk; either than Rosety-Rodriguez et al[31] were aimed at clarifying whether central obesity measurements assessed by dual X-ray absorptiometry may predict metabolic syndrome in 1326 Spanish postmenopausal women aged > 45 years old, during the observation period 537 women, representing 40.5% of the total studied, met the diagnostic criteria for metabolic syndrome. Also differs from those values reported by Miguel-Soca et al.[8] who studied 298 menopausal women aged 59.5±9.7 years finding a BMI of 30.46±5.23 kg/m^2, and WC of 102.16±11.58 cm. Menopause brings with it increased fat due to age decreases of the basal metabolic rate associated with a progressive reduction in physical activity, decreased estrogen controlling leptin and possibly regulating appetite, inadequate food habits, socioeconomic conditions and genetic[8].

The high cardiovascular risk observed in these women justifies the need for studies on the lipid profile diet effects, that being the main objective of this re-

Table III
Comparison of metabolic and lipid profile of the participants in the two periods of dietary intervention.

Biochemicals	***Dietary Intervention Type***				
	Diet SAT		*Bonferroni*	*Diet MONO*	*Bonferroni*
	Beginning (n=18)	*Ending (n=18)*	*p<0,005*	*(n=18)*	*p<0,005*
Glycemia (RV: 70-100 mg/dl)	85.0±6.1	95.4±11.9 (↑11.7%)	0,008	94.0±10.8 (↓1.5%)	NS
Basal Insulin (RV:< 25 UI/l)	9.5±2.6	11.1±2.3 (16.8%)	NS	10.2±1.8 (↓8.9%)	NS
HOMA (RV: <2,5)	1.99±0.66	2.62±0.77 (↑31.6%)	0,029	2.36±0.67 (↓10%)	NS
Total Cholesterol (RV:<200 mg/dl)	182.7±16.2	223.2±30 (↑22.1%)	0,001	211.7±39.8 (↓5.1%)	NS
HDL-C (RV: <45 mg/dl)	39.3±2.7	40.0±1.9 (↑1.7%)	NS	43.4±1.6 (↑8.5%)	0,000
LDL-C (RV: 100-140mg/dl)	110.1±12.6	149.0±30 (↑35.3%)	0,002	140.9±43.3 (↓5.5%)	NS
VLDL-C (<25 mg/dl)	33.1±1.7	33.6±4.6 (↑1.5%)	NS	31.2±4.2 (↓7.2%)	NS
No HDL-C Cholesterol (< 140 mg/dl)	143.3±13.7	183.1±13.7 (↑27.7%)	0,000	169.7±37.3 (↓7.4%)	NS
Triglycerides (<150 mg/dl)	164.9±6.4	168.1±23.0 (↑1.9%)	NS	155.8±21.7 (↓7.4%)	NS
CT/HDL-C Rel (RV: 00-4,5)	4.61±0.25	5.5±0.7 (↑19.3%)	0,000	4.8±0.9 (↓12.7%)	0,015
LDL-C/HDL-C Rel (RV: 0,0-3)	2.83±0.22	3.8±0.7 (↑34.2%)	0,000	3.5±0.9 (↓7.8%)	NS
TG/HDL-C Rel (RV:0,0-3)	4.22±0.30	4.20±0.68 (↓0.47%)	NS	3.90±0.87 (↓7.1%)	0,003
Col No HDL-C/HDL-C Rel (RV: 00-4,5)	3.63±0.20	4.47±0.58 (↑23.1%)	0,000	3.63±0.20 (↓18.7%)	0,016

Values represent the mean ± DE
Diet SAT: Butter diet. Diet MONO: Extra virgin olive oil diet.
HDL-C: High density lipoproteins. LDL-C: Low density lipoproteins, VLDL-C: Very low density lipoproteins, Col HDL-C: cholesterol no HDL-C , TG: Triglycerides, Rel: Relación.

search. In terms of diet in different population groups, it has been reported that it is especially important in the prevention of mentioned diseases[12,32] Hu et al.[32] conducted a study of 80,082 women between 34 and 59 years old, which showed that the replacement of saturated fat and unsaturated fatty acids trans for monounsaturated or polyunsaturated fatty acids prevents the development of coronary heart disease, more significantly compared with only reduce consumption of saturated fat.

The present study at the end of the SAT period, an increase in TC at the expense of LDL-C was observed. It has been demonstrated that LDL-C is one of the key factors in the development of atherosclerosis, since they are more susceptible to oxidation because they are better able to enter the macrophage monocyte arterial wall system; the opposite effect occurred when the extra virgin olive oil was consumed as monounsaturated fatty acids present in this oil are incorporated to the lipoproteic particle[15]. Concerning HDL-C was observed, that EVOO diet significantly improved HDL-C by 8.5% compared with the diet butter. It has been reported that the concentration of HDL-C is the best predictor of coronary heart disease risk in women, and their risk decreases by 3% for every 1 mg/dl increasing HDL-C in plasma[32].

In this study, cardiovascular disease predictive index in the 2 periods of dietary intervention for com-

Table IV
Comparison of lipoproteins ratio and cardiovascular risk of the participants in the two periods of dietary intervention.

Indicator	*Dietary Intervention Type*		
	Diet SAT		Diet MONO
	BEGINNING (n=18)	ENDING (n=18)	(n=18)
Total Cholesterol			
Desirable	17 (94.4%)	4 (22.2%)	7 (38.9%)
High	1 (5.6%)	7 (38.9%)	7 (38.9%)
Very high	0 (0.0%)	7 (38.9%)	4 (22.2%)
Low density lipoproteins			
Optimum	4 (22.2%)	0 (0.0%)	2 (11.1%)
Intermediate	14 (77.8%)	6 (33.3%)	7 (38.9%)
High limit	0 (0.0%)	6 (33.3%)	4 (22.2%)
High	0 (0.0%)	5 (27.8%)	1 (5.6%)
Very high	0 (0.0%)	1 (5.6%)	4 (22.2%)
Cholesterol total/HDL-C Relationship			
Low risk	3 (16.7%)	0 (0.0%)	6 (33.3%)
Moderate risk	15 (83.3%)	17 (94.4%)	12 (66.7%)
High risk	0 (0.0%)	1 (5.6%)	0 (0.0%)
LDL-C/HDL-C Relationship			
Low risk	2 (11.1%)	0 (0.0%)	3 (16.7%)
Moderate risk	16 (88.9%)	5 (27.8%)	7 (38.9%)
High risk	0 (0.0%)	13 (72.2%)	8 (44.4%)
Triglycerides/HDL-C Relationship			
Low risk	0 (0.0%)	0 (0.0%)	1 (5.6%)
High risk	18 (100.0%)	18 (100.0%)	17 (94.4%)
Colesterol NO HDL-C/HDL-C Relation			
Low risk	18 (100.0%)	3 (16.7%)	12 (66.7%)
High risk	0 (0.0%)	15 (83.3%)	6 (33.3%)

Values represent the mean ± DE
Diet SAT: Butter diet. Diet MONO: Extra virgin olive oil diet.
HDL-C: High density lipoproteins. LDL-C: Low density lipoproteins, VLDL-C: Very low density lipoproteins, Col HDL-C: cholesterol no HDL-C , TG: Triglycerides , Rel: Relationship.

paring the use impact of diet high in SFA and its replacement by a diet rich in MUFA were calculated; in this sense the Castell index and LDL-C/HDL-C ratio reflect increased atherogenic lipoproteins (LDL-C), and the decrease in HDL-C correlating with cardiovascular risk factors such as the components of metabolic syndrome (MS). Moreover, it has been shown that the predictive value of TG/HDL-C ratio is high for heart disease, and there is a direct relationship with hypertension and MS[9].

In this line of thought, the results of this work at the beginning there was no PMW with high CV risk; which is why they differ from E Chavez Gonzalez et al.[33] who evaluated the rate of Castell in 112 menopausal study reporting that 9.3% had low risk, 68.6% moderate risk and 22% high cardiovascular risk. Similarly, regarding the dietary intervention periods, the lipoprotein indicators showed high atherogenic and MS risk in patients following consumption of a diet rich in butter; as well as the beneficial effect of consuming a diet rich in EVOO.

The consumption of olive oil has been widely studied[13,15,21]. The PREDIMED study with 7,216 subjects between 55 and 80 years old, including both genders, with high cardiovascular risk, concluded that consumption of olive oil, especially extra-virgin variety was associated with a significantly reduced risk of cardiovascular events and cardiovascular mortality. It is important to consider that for every 10 g per day increase in total consumption of olive oil is associated with a 16% reduction in cardiovascular mortality, based on the findings in the EPIC-España[34] study. No literature that would allow comparison of lipid profile dietary intervention periods applying the aforementioned ratios is found.

This study has some limitations. The most important one was the small sample size, due to the high cost of chemical reagents, its scarcity and high inflation in the country where the study took place. As well as consumer acceptance of extra virgin olive oil because it is not part of the food patterns within the Western diet.

Conclusion

The results of this study indicates that replacing saturated fat butter and extra virgin olive oil improved atherogenic dyslipidemia, but did not influence the metabolic profile in these postmenopausal women.

Conflicts of interest

No conflicts of interest.

References

1. Zárate A, Saucedo R, Basurto L, Martínez C. La enfermedad cardiovascular como amenaza actual para la mujer adulta mayor. *Ginecol Obstet Mex*.2007; 75: 286-92.
2. Rojas J S, Lopera V J S, Cardona V J, Vargas G N, Hormaza A M P. Síndrome metabólico en la menopausia, conceptos clave. *Rev. chil. obstet. ginecol*. 2014; 79(2) : 121-128.
3. Pinto López E, Rice C and Weddle D O. The relationship among cardiovascular risk factors, diet patterns, alcohol consumption, and ethnicity among women aged 50 years and older. *J. Am Diet Assoc*. 2008; 108(2): 248-256.
4. Velázquez-Maldonado Elsy M. Prevención cardiovascular en la mujer postmenopáusica. Rev. Venez. Endocrinol. *Metab*. 2009 Jun; 7(2): 3-16.
5. Rosales Ricardo Y. Antropometría en el diagnóstico de pacientes obesos: una revisión. *Nutr. Hosp*. 2012 Dic; 27(6): 1803-1809.
6. Arca M, Lena-Vega G, Grundy S. Hypercholesterolemia in post-menopausal women. *JAMA*.1994; 27: 453-459.
7. Villasmil E R, Guerra V M, Torres M M, Reyna N, Mejía M J. Perfil lipídico en mujeres pre menopáusicas y posmenopáusicas. *Rev Obstet Ginecol Venez*. 2007; 67(2): 107-114.
8. Miguel-Soca PE, Rivas-Estévez M, Sarmiento-Teruel Y, Mariño-Soler AL, Marrero-Hidalgo M, Mosqueda-Batista L. Factores de riesgo de enfermedad cardiovascular en mujeres con menopausia. *Rev Fed Arg Cardiol*. 2014; 43(2): 90-96.
9. Millán J, Pintó X, Muñoz A, Zúñiga M, Rubíes-Prat J, Pallardo L F et al. Cocientes Lipoproteicos: significado fisiológico y utilidad de los índices aterogénicos en prevención cardiovascular. *Clin Invest Arterioscl*. 2010; 22(1): 25-32.
10. Acevedo M, Krämer V, Tagle R, Corbalán R, Arnaíz P, Berríos X, Navarrete C. Relación colesterol total a HDL y colesterol no HDL: los mejores indicadores lipídicos de aumento de grosor de la íntima media carotidea. *Rev Med Chile* 2012; 140: 969-976.
11. CEPAL América Latina y el Caribe: el envejecimiento de la población. 1950-2050. Santiago de Chile: Boletín Demográfico. Centro Latinoamericano y Caribeño de Demografía (CELADE); 2003.
12. Carrillo Fernández L, Dalmau Serra J, Martínez Álvarez JR, Solà Alberich R, Pérez-Jiménez F. Grasas de la dieta y salud cardiovascular. *Nutr. clín. diet. Hosp*. 2001; 31(2): 6-25.
13. Pérez-Martínez P, Moreno-Conde M, Cruz-Teno C, Ruano J, Fuentes F, Delgado-Lista J, Garcia-Rios A et al. Dietary fat differentially influences regulatory endothelial function during the postprandial state in patients with metabolic syndrome: from the LIPGENE study. *Atherosclerosis*. 2010; 209(2): 533-8.
14. López EP, Rice C, Weddle DO, Rahill GJ. The relationship among cardiovascular risk factors, diet patterns, alcohol consumption, and ethnicity among women aged 50 years and older. *J Am Diet Assoc*. 2008 Feb; 108(2): 248-56. doi: 10.1016/j.jada.2007.10.043.
15. Rodenas S, Rodríguez-Gil S, Cruz Merinero M, Sánchez-Muniz FJ. Dietary Exchange of an Olive Oil and Sunflower Oil Blend for Extra Virgin Olive Oil Decreases the Estimate Cardiovascular Risk and LDL and Apolipoprote in AII Concentrations in Postmenopausal Women. Rev. Journal of the American *College of Nutrition*. 2005; 24(5): 361–369.
16. Tardivo AP, Nahas-Neto J, Nahas EA, Maesta N, Rodrigues MA, Orsatti FL. Associations between healthy eating patterns and indicators of metabolic risk in postmenopausal women. *Nutr J*. 2010 Dec 8; 9:64. doi: 10.1186/1475-2891-9-64.
17. Cicerale S, Conlan XA, Sinclair AJ, and Keast RSJ. Chemistry and Health of Olive Oil Phenolics. Critical Reviews in Food *Science and Nutrition*. 2009; 49: 218–236.
18. Sun J, Buys NJ, Hills AP. Dietary pattern and its association with the prevalence of obesity, hypertension and other cardiovascular risk factors among Chinese older adults. Int J Environ *Res Public Health*. 2014 Apr 10; 11(4): 3956-71. doi: 10.3390/ijerph110403956.
19. Vilela AA, Sichieri R, Pereira RA, Cunha DB, Rodrigues PR, Gonçalves-Silva RM, Ferreira MG. Dietary patterns associated with anthropometric indicators of abdominal fat in adults. *Cad Saude Publica*. 2014 Mar; 30(3): 502-10.
20. Pérez- Martínez P, López- Miranda J, Delgado-Lista J, López-Segura F y Pérez - Jiménez F. Aceite de oliva y prevención cardiovascular: más que una grasa. *Clin Invest Arterioscl*. 2006; 18(5): 195-205.
21. Shaw, D.I., Tierney A.C., McCarthy S., Upritchard J., Vermunt S., Gulseth H.L. et al. J.A. LIPGENE food-exchange model for alteration of dietary fat quantity and quality in free-living participants from eight European countries. *British Journal of Nutrition*. 2009; 101(5): 750-759. ISSN 0007-1145.
22. Onis M, Habicht P. Anthropometric reference data for international use: recommendations from a World Health Organization Expert Committee. 1996.
23. ISAK. International Standards For Anthropometric Assessment. International Society for the Advancement of Kinanthropometry. *National Library of Australia*. 2001: 1-123.
24. Díaz Portillo J, Staff V.V., Fernández del Barrio M.T., Paredes Salido F. Determinaciones bioquímicas: Utilidad y valoración clínica Editorial Elsevier, España, 2004. ISBN 8445813684, 9788445813683. 524 pp.
25. Castelli WP. Epidemiology of coronary heart disease: The Framingham Study, *Am J Med*, 1984; 76: 4-9.
26. ATP III Guidelines At-A-Glance Quick Desk Reference. Third Report of the Expert Panel on. National Cholesterol Education Program. National. institutes of Health. National Heart, Lung, and Blood Institute. *NIH Publication*. 2001; No. 01-3305:1-6.
27. Palma SG, Puchal A, Vilà M C, Miserachs M, Illan M . Entrevista dietética: Herramientas útiles para la recogida de datos. *Rev Med Univ Navarra*. 2006; 50(4): 46-55.

28. Instituto Nacional de Nutrición. Tabla de Composición de Alimentos para uso práctico. Publicación Nº 52. Caracas (Venezuela). *Serie Cuadernos Azules*. 2001: 15-77.
29. Manual del Usuario de SPSS (2010) IBM_SPSS_Statistics_Base_20.148151002 SPSS Inc., an IBM Company Headquarters, 233 S. Wacker Drive, 11th floor. Chicago, Illinois 60606.
30. Pavón de Paz I., Alameda Hernando C y Olivar Roldán J. Obesidad y Menopausia. *Nutr. Hosp*. 2006; 21(6): 633-637.
31. Meertens L, Díaz N, Fraile C, Riera M, Rodirguez A, Rodríguez L y cols . Estado nutricional, indicadores antropométricos y homocisteína sérica en mujeres posmenopáusicas venezolanas. *Rev. chil. nutr.* 2011 Sep; 38(3) : 278-284.
32. Hu FB, Stampfer MJ, Manson JE, Rimm E, Colditz GA, Rosner BA et al. Dietary fat intake and the risk of coronary heart disease in women. *N Engl J Med*.1997; 337: 1491–1499.
33. Chávez González E, Romero Romero N, Pardo Morales RV. Índice aterogénico en pacientes Perimenopáusicas. *Archivos de Investigación Materno Infantil*. 2011 mayo-agosto; III (2): 73-76. URL:www.medigraphic.org.mx.
34. Guasch-Ferré M, Hu FB, Martínez-González MA, Fitó M, Bulló M, Estruch R et al. Olive oil intake and risk of cardiovascular disease and mortality in the PREDIMED Study. *BMC Med*. 2014 May 13;12:78. doi: 10.1186/1741-7015-12-78.