Nor. Afr. J. Food Nutr. Res. 2020; 4(9): S7-S16

https://doi.org/10.51745/najfnr.4.9.S7-S16

https://www.najfnr.com

Special Issue.
Nutritional Situation In Africa: An Update (UPNIA-2020)

ORIGINAL ARTICLE

Cross-sectional association between lifestyle behavior and cardiometabolic biomarkers in west Algerian postmenopausal women

Amina Tiali [1], Djahida Chenni [2], Mohamed Benyoub [3], Khedidja Mekki [1]*

[1] Laboratory of Clinical and Metabolic Nutrition, University Oran 1, Oran 31100, Algeria
[2] Etablissements Publics de Santé de Proximité (EPSP) Messerguine, Oran, Algeria
[3] Department of Epidemiology, University Hospital of Oran, Oran 31037, Algeria

Abstract

Background: Abdominal adiposity, insulin resistance dyslipidemia, and endothelial dysfunction emerge during menopause. **Objectives:** To assess the relationship between lifestyle, eating behavior, and cardiometabolic biomarkers in Algerian postmenopausal women. **Subjects and Methods:** A prospective cross-sectional survey was conducted among 228 postmenopausal women (57.65±6.42 years) in Oran (Algeria). Women were divided into quartiles according to their waist circumference (WC); Q1 (69-89cm), Q2 (90-98.5cm), Q3 (99-108cm), and Q4 (108-125cm). We assessed for 3 days, food consumption by the 24h recall and record method, and daily energy expenditure (DEE). In serum, we analyzed the lipid profile, inflammation markers, and oxidative status. **Results**: DEE and total energy intake were similar in all groups. A decrease in metabolism equivalent tasks (Mets) was observed according to WC increase (<1.5). The Mets was negatively correlated with LDL-cholesterol, triacylglycerols (TG), lipid accumulation products (LAP), CRP, thiobarbituric acid reactive substances (TBARS), TBARS-LDL, and carbonyls and positively correlated with the activity of lecithin cholesterol acyltransferase (LCAT), superoxide dismutase (SOD) and catalase. An inverse relationship was noted between the intake of meats, poultry, eggs, fish, and antioxidant enzymatic activities. Fat intake was positively correlated with lipid accumulation products (r=0.293, p<0.001) and negatively with HDL-cholesterol (r=-0.396, p<0.001), LCAT activity (r= -0.275, p<0.001) and C-Reactive Protein (CRP) (r= -0.315, p<0.001). Fruits and vegetables intake was negatively correlated with LDL-Cholesterol (r=- 0.279, p<0.001) and LDL-TBARS (r= -0.284, p<0.001). **Conclusion:** Unhealthy diet and sedentary lifestyle were associated with high cardiometabolic risk factors in postmenopausal women and exposed them to cardiovascular diseases.

Keywords: Lifestyle behavior, Cardiometabolic biomarkers, Waist circumference, Postmenopausal women.

Received: July 13, 2020 / **Accepted**: October 05, 2020 / **Published**: October 16, 2020

1 Introduction

The midlife period is a critical window for the development of cardiovascular diseases (CVD) [1]. Diabetes and/or strokes are a consequence of estrogen deficiency or results from a higher prevalence of cardiometabolic risk factors such as abdominal obesity, insulin resistance, dyslipidemia, and endothelial dysfunction, which occur with aging [2-5].

Menopause can promote insulin resistance, conferring an increased risk of type 2 diabetes. Furthermore, it is associated with a weight gain then a shift from a gynoid to an android profile.

Lifestyle can be responsible for up to 40% of premature death from cardiovascular disease. The reduction in morbidity and cardiovascular mortality is constantly observed with high adherence to the Mediterranean diet consisting of fish, unsaturated fats, whole seeds, fruits and vegetables, nuts, and legumes. Around menopause energy requirements decrease and are related to the decrease in basal metabolism [6]. These changes, associated with a sedentary lifestyle and eating disorders, are responsible for the high prevalence of visceral obesity. Inappropriate eating habits, characterized by high-energy dense food and low nutrients, are responsible for significant weight gain[7]. Moreover, vasomotor symptoms (hot flushes, night sweats, and insomnia) are linked to a 70% increase in cardiovascular disease by the genesis of inflammation [8].

The concomitant presence of android obesity, dyslipidemia, and hypertension and the decrease in insulin sensitivity leads to metabolic syndrome (MS), a high-risk metabolic and cardiovascular entity. MS being prevalent in Algerian menopausal women (57.9%) is characterized by a high prevalence of abdominal obesity (67.2%) [9].

Inflammation being the fundamental mediator of CVD[10], as well as oxidative stress, is defined as a disturbance in the balance between the production of reactive oxygen species (ROS) and antioxidant defenses are interdependent and play a crucial role in the development of CVD [11, 12]. Estrogen deficiency plays an

* **Corresponding author:** Khedidja Mekki, Laboratory of Clinical and Metabolic Nutrition, University Oran 1, Oran 31100, Algeria. Tel : + 213 552649929 E-mail: khmekki@hotmail.com

important role in the etiology and pathophysiology of chronic inflammatory and degenerative diseases [13]. However, abdominal obesity alone is currently considered a chronic state of inflammation and oxidative stress, even in the absence of other risk factors for CVD.

The aim of the current study was to evaluate the relationship between lifestyle, eating behavior, and cardiometabolic biomarkers in west Algerian postmenopausal women with abdominal obesity. We assessed food intake and energy expenditure and correlated them with lipids profile, inflammatory markers, and oxidant/antioxidant status.

2 Subjects and Methods

2.1 Study setting

An observational cross-sectional study was conducted at the Gynecological department Clinic TOULOUSE 1 and polyclinic of "*Misserghuin*" in Oran (West of Algeria). Two hundred twenty-eight (n=228) post-menopausal women (Table 1) were enrolled for the study. Women were considered postmenopausal after twelve months and more physiological amenorrhea.

We excluded subjects taking antioxidant supplements, anti-inflammatory and lipid-lowering drugs, undergoing hormonal therapy, radiotherapy, and women suffering from thyroid and kidney diseases. All women selected for the study included 54 diabetics women treated with biguanides, and 63 hypertensives treated with converting enzyme inhibitors alone (7.14%) or combined with diuretics (29%), others by calcium channel blockers (21%), vasodilator nitrates (7%) or selective B-blockers (21%).

The purpose of this study was explained to all women and the investigation was carried out with their consent. The experimental protocol was approved by the Committee for Research on Human Subjects of Oran.

2.2 Lifestyle assessment

Daily energy expenditure (DEE) was assessed on 3 days using an adapted questionnaire inspired by the International Physical Activity Questionnaire (IPAQ). This variable being primarily influenced by lean body mass and the type, duration, and intensity of physical activity, was assessed using the following formula: DEE (Kcal/d) = ∑Factorial daily expenditure (FDE) [14] and was calculated as FDE (Kcal/d) = Resting Metabolism*Metabolic equivalent of task* duration expressed in hours / 24 hours. The formula of Black [15] was used to calculate the resting metabolism (RM), which represents the largest component of total energy expenditure and is a major contributor to energy expenditure. Metabolic equivalent tasks (METS) was used to define sedentary and active behavior. Sedentary activity: 1-1.59 METS; light activity: 1.6-2.9 METS; moderate activity: 3-5.9 METS; intense activity: ≥6 METS [16].

2.3 Cardiometabolic biomarkers assessment

Blood samples were drawn after 12 hours overnight fast from antecubital venipuncture. Tubes containing the lithium heparin were used for biochemical experiments. A tube containing Ethylenediaminetetraacetic acid (EDTA) was used to prepare lipoproteins fractions. We collected serum by low-speed centrifugation at 3000X g, at 4°C for 15 min. The serum was removed, aliquoted, and stored at -20° C.

Lipoproteins were separated by precipitation[17] using $MgCl_2$ and dextran sulfate weight 500,000 (Sigma Chemical Company, France). Triacylglycerols (TG) and total cholesterol (TC) were determined in serum and lipoproteins by colorimetric methods (Spinreact, Spain).

Lipid Accumulation Products (LAP) was defined as [(WC (cm) - 58)*(TG (mmol/L)] for women. LAP is an index of central lipid accumulation to predict the risk of metabolic syndrome. The formula includes the minimum WC values (58 cm for women) at NHANES III (Third National Health and Nutrition Examination Survey) [18]. In our population sample, the minimum WC value was 69 cm.

Lecithin cholesterol acyltransferase (LCAT) activity was determined by endogenous method [19]. The enzyme esterifies the unesterified cholesterol (UC) into a lecithin fatty acid, which leads to a disappearance of UC and the appearance of esters of cholesterol (EC). UC was analyzed by enzymatic colorimetric technique (Biolabo, France). The activity of LCAT is based on the disappearance of UC during four hours of assessment and was calculated following the formulae: LCAT activity = (CLt0h - CLt4h) / 4 and is expressed as mmol/L/h.

C-reactive protein (CRP) was measured using duplicate samples with an immunometric assay kit (ELISA) (Cayman Chemical's human EIA kit) with a range of 0–3000 pg/mL with a limit of detection of approximately 50 pg/mL. The quantitative determination of fibrinogen in plasma according to Von Clauss [20] was carried out on an analyzer automate ACL ELITE model (Series Coagulation 2015) using fibrinogen kit-C-0020301100 from HemosIL ®.

For lipid peroxidation assessment, we measured the concentration of thiobarbituric acid reactive substances (TBARS) using Tetramethoxypropane (Prolabo) as a precursor of malondialdehyde [21]. One milliliter of the diluted sample (protein concentration about 2mg/mL) was added to 2 mL of thiobarbituric acid (final concentration. 0.017mmol/L) and butylated hydroxytoluene (concentration 3.36mmol/L) and incubated for 30 min at 85°C. After cooling and centrifugation, the absorbance of the supernatant was measured at 535 nm. Data were expressed as mmol of TBARS produced/mL of serum.

Table 1: Clinical characteristics of postmenopausal women according to waist circumference quartiles

Parameters	Q 1 (69-89cm)	Q 2 (90-98.5cm)	Q 3 (99-108cm)	Q 4 (108-125cm)
Age (years)	57.59±6.76	57.22±5.79	56.48±6.23	59.20±6.72 p=0.028[c]
Age at menopause (years)	49.04±0.03	48.33±4.16	48.87±6.11	47.41±4.78
Weight (Kg)	59.94±8.09	67.05±7.56 p<0.001[a]	77.02±7.91 p<0.001[a, b]	83.35±9.41 p<0.001[a, b, c]
WC (cm)	83.50±4.68	93.91±2.51 p<0.001[a]	103.07±2.55 p<0.001[a, b]	114.07±4.82 p<0.001[a, b, c]
Height (m)	1.59±0.071	1.58±0.056	1.61±0.068	1.58±0.06 p=0.044[c]
BMI (kg/m^2)	23.72±3.30	26.79±2.92 p<0.001[a]	29.88±3.065 p<0.001[a, b]	33.36±3.89 p<0.001[a, b, c]
Waist / hip	0.90±0.07	0.91±0.06	0.91±0.068	0.92±0.068
SBP mm Hg	131.76±16.30	126.58±19.58	135.98±18.75 p=0.010[b]	145.35±23.022 p=(<0.001[a, b], 0.020[c])
DBP mmHg	85.55±11.73	84.37±13.27	84.67±10.64	93.62±12.061 p<0.001[a, b, c]
Blood glucose (g/L)	0.94±0.19	0.91±0.18	0.91±0.17	0.89±0.16
Urea (mmol/L)	4.66±1.29	4.78±1.18	4.73±1.23	4.88±1.19
Creatinine (µmol/L)	76.39±8.40	74.82±9.28	75.41±13.01	75.58±10.02
Uric acid (mg/L)	55.43±12.89	55.56±12.24	58.24±10.55	54.88±11.34
Proteins (g/L)	74.45±7.61	74.08±7.06	75.29±6.91	74.05±7.13
Albumin (g/L)	45.89±7.64	46.92±6.26	47.76±6.36	46.44±7.13

BMI: Body mass index; WC: waist circumference; SBP: Systolic blood pressure; DBP: Diastolic blood pressure. Data are presented as means±SD. After analysis of variance (ANOVA), means were compared by Student's t-test. a: (Q2, Q3, Q 4) vs Q1; b(Q3, Q4) vs Q2; c: Q4 vs Q3. p< 0.05 was considered statistically significant.

Table 2: Energy balance in postmenopausal women according to waist circumference quartiles

	Q 1 (69-89cm)	Q 2 (90-98.5cm)	Q 3 (99-108cm)	Q 4 (108-125cm)
TEI (MJ/d) [min- max] values	6.7±1.67 [2.61-10.60]	7.02±1.80 [3.27-10.60]	6.98±1.78 [3.27-10.61]	6.38±1.24 p=(0.035[b], 0.026[c]) [3.99-9.09]
DEE (MJ/d) [min- max] values	7.78±0.76 [6.19- 9.15]	7.89±0.93 [6.14- 9.78]	7.77±0.84 [5.93- 9.79]	7.97±0.70 [6.59-9.51]
TEI/DEE [min- max] values	0.88±0.22 [0.31-1.46]	0.93±0.28 [0.36-1.61]	0.93±0.27 [0.45-1.72]	0.81±0.18 p=(0.007[b] 0.008[c]) [0.52-1.32]
RM (MJ/d) [min- max] values	5.15±0.43 [1.06-1.59]	5.41± 0.39 p=0.001[a] [4.28-6.15]	5.62 ±0.40 p=(<0.001[a],0.007[b]) [4.52-6.94]	5.91± 0.43 p<0.001[a, b, c] [5.17-6.94]
%RM (proportion of DEE) [min- max] values	66.52 [55.62- 79.67]	69.12 p=0.025[a] [59-79.67]	72.84 p=(<0.001[a], 0.002[b]) [55.62- 81.52]	74.55 p<0.001[a, b] [55.62- 81.27]
METS [min- max] values	1.5±0.13 [1.25-1.79]	1.44±0.13 p=0.018[a] [1.25-1.79]	1.39±0.14 p=(<0.001[a], 0.021[b]) [1.22-1.79]	1.35±0.12 p<0.001[a, b] [1.23-1.66]

TEI: Total energy intake; DEE: Daily Energy Expenditure; RM: Resting Metabolism; Mets: Metabolic equivalent task.
[min-max] values: interval of minimum and maximum values.
Data are presented as means ± SD. After analysis of variance (ANOVA), means were compared by Student's t-test. a: (Q2, Q3, Q 4) vs Q1; b(Q3, Q4) vs Q2; c: Q4 vs Q3. p< 0.05 was considered statistically significant.

Oxidized proteins were evaluated by the analysis of carbonyls concentrations [22] using the 2,4-dinitrophenylhydrazine (DNPH). Superoxide dismutase (SOD) activity was determined in serum at 420nm by measuring the auto-oxidation of pyrogallol [23]. Catalase (CAT; EC 1.11.1.6; $2H_2O_2$ oxidoreductase) activity was measured at 420 nm by assessing the H_2O_2 decomposition rate [24]. The assay was performed on a 250-µl sample. 250 µL of H_2O_2 30mmol/L (dilute 0.34 mL in 100mL phosphate buffer 50mmol / L) and 250 µL of phosphate buffer were added, the solution was then stirred and incubated for 5 min, and results were expressed as U/mL.

2.4 Statistical analysis

Women were categorized according to quartiles of waist circumference and analysis was performed using SPSS 20.0 (IBM SPSS Statistics. Armonk. NY). Data were expressed as the means ± SD (standard deviation). Comparison between groups was performed using the unpaired Student's t-test, One-way analysis of variance (ANOVA). The correlations were established by Pearson linear regression test. P < 0.05 was considered statistically significant.

3 Results

3.1 Lifestyle factors

Lifestyle factors evaluation (Table 2) shows that total energy intake (TEI) was similar in Q2, Q3, and Q4, compared to Q1 and the means were less than 8 MJ/d. Compared to Q2, TEI decreased in Q4 ($p=0.035$) and was similar in Q3 (p=0.846). A significant decrease was noted in Q4 (p=0.026) compared to Q3. Daily energy expenditure (DEE) was similar in all groups.

Resting metabolism was significantly more important in all groups compared to Q1. The percentage of RM increased significantly by +3.91% in Q2 (p=0.025), +9.50% in Q3 and +12.07% in Q4 (p<0.001), compared to Q1. Moreover, the increase was by +5.38% in Q3 (p=0.021) and by +2.35% in Q4 (p<0.001), compared to Q2.

Sedentary behavior was noted in women. The Mets decreased significantly by -4%in Q2 (p=0.018), -7.33% in Q3 (<0.001) and -10% in Q4 (<0.001) compared to Q1. Significant decrease by -3.47% was noted in Q3 (p=0.021), -2.88% in Q4 compared toQ2. No difference was noted between Q3 and Q4.

Table 3 shows the food composition in postmenopausal women according to the WC quartile. Expressed in percentage of TEI, we noted that protein intake was more than 16% and values were higher in Q2, Q3 and Q4, compared to Q1 (p<0.001).

Carbohydrates energy intake was similar in Q1 and Q2 (55% of TEI) and decreased in Q3 (49%) and Q4 (47%). Lipids energy intake was more important (p= 0.005) in Q4 than Q1 and Q2 (p<0.001).

Qualitative contribution of nutrients showed increased intake of animal proteins and decreased intake of vegetable proteins (p=0.003) in Q4 compared to Q2. We noted a decrease in polyunsaturated fatty acids (PUFA) intake in Q4 compared to Q1 (p= 0.056) and increased monounsaturated fatty acids (MUFA) intake in Q2 compared to Q1 (p= 0.046).

The intake of food groups (Table 4) showed higher consumption of meat, poultry, fish, and eggs in Q3 and Q4 (p<0.001), compared to Q1. Moreover, this intake was more important in Q4 compared to Q2 (p= 0.021). Compared to Q1, the intake of milk and dairy products was similar in Q2 and Q3 but increased in Q4 (p= 0.001). A significant increase was also noted in Q4 compared to Q2 (p= 0.013) and compared to Q3 (p= 0.016). Low consumption of fruits and vegetables was noted in all groups. Compared to Q1, we noted a significant decrease in fruits and vegetable intake in Q2 (p= 0.001), Q3 (p=0.032), and Q4 (p=0.046). Fat intake was similar in all groups. It was increased in Q4 compared to Q1 (p=0.008), Q2 (p= 0.003) and Q3 (p= 0.010). High consumption of sugar and sweet products was observed and values were identical in all groups.

3.2 Lipids profile

Compared to Q1, LCAT activity (Table 5) was similar in all groups, but a decrease by -14.75% was noted in Q4 compared to Q1 (p= 0.002) and by -10.16% compared to Q2 ((p= 0.0054). Compared to Q1, TC increased in Q3 (p=0.024) and Q4 (p<0.001). TG increased by +16.39% in Q2 (p=0.038), +39.34% in Q3 and +35.24% in Q4 (p<0.001) compared to Q1. Compared to Q1, VLDL-C, HDL-C, TC/HDL-C, and LDL-C/HDL-C ratio values were similar in all groups. However, we noted that HDL_2-C was significantly increased in Q2 (p = 0.042), Q3 (p= 0.004) and Q4 (p= 0.007) compared to Q1.

LAP were increased in all groups compared to Q1 (p = <0.001). A significant increase in WC was noted (p<0.001).

3.3 C-Reactive protein and Oxidant/Antioxidant status

CRP values were 1.39-fold higher in Q2, 1.61-fold in Q3, and 2.25-fold in Q4, compared to Q1. The values of CRP were more elevated in Q4 compared to Q3 and Q2 (p<0.001) (Table 6).

TBARS increased by 41% in Q2 (p=0.003), 43.92% in Q3 (p= 0.002) and 65.42% in Q4 (<0.001), compared to Q1. Moreover, TBARS increased according to WC increase by 21.73% in Q2 (p = 0.003), 27.17% in Q3 (p = 0.003) and by 38% in Q4 (p <0.001), compared to Q1.TBARS- LDL values were more elevated in Q4 compared to Q2 (p=0.007).

Protein carbonyls concentrations were increased in Q2 (p=0.002), Q3 (p<0.001), and Q4 (p<0.001), compared to Q1.

A decrease in SOD activity was noted in Q2 (p=0.001), Q3, and Q4 (p<0.001) compared to Q1. Likewise, we noted that catalase activity was decreased in Q2 (p=0.005), Q3 (p<0.001), and Q4 (p<0.001) compared to Q1.

3.4 Relationships between lifestyle factors and cardiometabolic biomarkers

Table 7 shows the most significant correlations established between lifestyle and biomarkers. An inverse relationship between TEI and; HDL-C (r= -0.232, p<0.001) and with LDL-C (r= -0.206, p= 0.002) were found. DEE was correlated with HDL-C (r= 0.265, p<0.001).

Table 3: Food intake composition in postmenopausal women according to waist circumference quartiles

	Q 1 (69-89cm)	Q 2 (90-98.5cm)	Q 3 (99-108cm)	Q 4 (108-125cm)	MD
TEI (MJ/d)	6.7±1.67	7.02±1.80	6.98±1.78	6.38±1.24 p=(0.035[b], 0.026[c])	8
Proteins (% of TEI)	16%	19% p=0.008[a]	21% p=(<0.001[a], 0.114[b])	21% p=(<0.001[a], 0.051[b])	10%
Proteins intake (g/d)	58.86±19.28	68.75±18.30 p=0.006[a]	73.83±17.01 p<0.001[a]	69.18±13.31 p=0.001[a]	
Animal protein[$]	43%	44%	47%	54% p=(0.001[a], 0.003[b])	40%
Vegetable protein[$]	57%	56%	53%	46% p=(0.001[a], 0.003[b])	60%
Carbohydrates (% of TEI)	55%	55%	49% p=(<0.001[a], 0.004[b])	47% p<0.001[a, b]	55%
Carbohydrates intake (g/d)	223.26±50.87	224.37±64.87	218.47±73.07	210.97±82.60	
Complex carbohydrates[$]	62%	69% p=0.008[a]	69%	62% p=(0.004[b], 0.007[c])	75%
Simple carbohydrates[$]	38%	31% p=0.007[a]	31% p=0.014[a]	38% p=(0.004[b], 0.007[c])	25%
Lipids (% of TEI)	29%	27%	31%	32% p=(0.005[a], <0.001[b])	35%
PUFA [$]	22%	18%	18%	17% p=0.056[a]	25%
MUFA[$]	47%	51% p=0.046[a]	52%	51%	50%
SFA[$]	31%	31%	30%	32%	25%

MD: Mediterranean Diet (24); TEI: Total energy intake; $: Expressed in percentage of total macronutrient intake. PUFA: Polyunsaturated fatty acids; MUFA: Monounsaturated fatty acids; SFA: Saturated fatty acids. Data are presented as means ± SD. After analysis of variance (ANOVA), means were compared by Student's t-test. a: (Q2, Q3, Q4) vs Q1; b(Q3, Q4) vs Q2; c: Q4 vs Q3. p< 0.05 was considered statistically significant.

Table 4: Food groups intake in postmenopausal women according to waist circumference quartiles

Food category (g/d)	Q 1 (69-89cm)	Q 2 (90-98.5cm)	Q 3 (99-108cm)	Q 4 (108-125cm)	MD (g/d)
Meat. poultry. fish and eggs	87.00±53.54	111.45±86.15	148.82±120.21 p=0.001[a]	153.07±108.04 p=(<0.001[a], 0.021[b])	148
Milk and dairy products	169.69±137.91	196.18±148.17	194.89±155.57	271.52±176.63 p=(0.001[a], 0.013[b], 0.016[c])	168
Fruits and vegetables	353.39±179.80	269.39±164.620 p=0.010[a]	283.88±151.95 p=0.032[a]	284.44±184.48 p=0.046[a]	240-480
Cereals and starchy foods	290.05±155.48	296.29±180.12	284.35±182.37	304.71±160.81	~400
Fat	11.41±8.10	11.13±6.93	11.59±7.37	17.17±13.80 p=(0.008[a], 0.003[b], 0.010[c])	-
Sugar and sugar products	213.14±162.91	219.48±164.7	246.85±214.65	247.49±204.85	≤ 9

Data are presented as means ± SD. After analysis of variance (ANOVA), means were compared by Student's t-test. a: (Q2, Q3, Q 4) vs Q1; b(Q3, Q4) vs Q2; c: Q4 vs Q3. p< 0.05 was considered statistically significant.

Mets was correlated with LCAT activity (r=0.243, p<0.001), SOD (r= 0.374, p<0.001) and catalase (r=0.283, p<0.001). However, Mets was negatively associated with LDL-C (r= -0.153, p= 0.021), HDL-C (r= -0.161, p= 0.015), TG (r= -0.442, p<0.001), LAP (r= -0.502, p<0.001), CRP (r= -0.428, p<0.001), TBARS (r= -0.367, p<0.001), TBARS-LDL (r= -0.338, p<0.001) and carbonyls (r= -0.349, p<0.001).

Fruits and vegetables intake was positively correlated with LCAT activity (r=0.324, p<0.001) and HDL-C (r= 0.140, p= 0.035) and negatively with LDL-C (r=- 0.279, p<0.001), and TBARS-LDL (r= -0.284, p<0.001).

Fibers intake correlated positively with SOD (0.340, p<0.001) and catalase (r= 0.166, p<0.001) activities and negatively with CRP (r= -0.335, p<0.001), TBARS (r= -0.261, p<0.001), TBARS-LDL (r= -0.153, p= 0.017) and carbonyls (r= - 0.386, p<0.001). Vitamin E intake was inversely correlated with TBARS-LDL (r=- 0.278, p<0.001). Vitamin C intake correlated positively with SOD (r= 0.388, p<0.001).

Table 5: Lecithin cholesterol acyltransferase activity, lipid profile and atherogenic indices in postmenopausal women according to waist circumference quartiles

	Q 1 (69-89cm)	Q 2 (90-98.5cm)	Q 3 (99-108cm)	Q 4 (108-125cm)
LCAT (mmol/L/h)	147.87±40.77	140.31±45.41	138.71±43.93	126.05±33.97 p=(0.002[a], 0.054[b])
TC (mmol/L)	4.50±0.81	4.44±0.80	4.16±0.73 p=(0.024[a], 0.050[b])	4.74±1.03 p=0.001[c]
TG (mmol/L)	1.22±0.51	1.42±0.53 p=0.038[a]	1.70±0.50 p=(<0.001[a], 0.005[b])	1.65±0.48 p=(<0.001[a], 0.018[b])
VLDL-C (mmol/L)	0.68±0.22	0.69±0.16	0.67±0.11	0.64±0.14
LDL-C (mmol/L)	2.28±0.40	2.19±0.54	2.19±0.49	2.38±0.56 p=(0.052[b], 0.050[c])
HDL_2-C (mmol/L)	0.56±0.16	0.61±0.12 p=0.042[a]	0.63±0.10 p=0.004[a]	0.62±0.08 p=0.007[a]
HDL_3-C (mmol/L)	0.85±0.23	0.72±0.23 p=0.003[a]	0.67±0.28 p<0.001[a]	0.81±0.38 p=0.024[c]
TC/HDL-C	3.18±0.57	3.38±0.93	3.20±0.97	3.22±0.98
LDL-C/HDL-C	1.62±0.31	1.62±0.38	1.58±0.31	1.58±0.29
LAP	31.45±15.44	50.90±19.07 p=<0.001[a]	76.23±22.19 p<0.001[a, b]	91.78±27.74 p<0.001[a, b, c]

LCAT: Lecithin cholesterol acyltransferase; TC: Total cholesterol; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; TG: Triacylglycerol; LAP: Lipid accumulation products. Data are presented as means±SD. After analysis of variance (ANOVA), means were compared by Student's t-test. a: (Q2, Q3, Q 4) vs Q1; b(Q3, Q4) vs Q2; c: Q4 vs Q3. p< 0.05 was considered statistically significant.

Table 6: C-Reactive Protein and Oxidant/Antioxidant status in postmenopausal women according to waist circumference quartiles

	Q 1 (69-89cm)	Q 2 (90-98.5cm)	Q 3 (99-108cm)	Q 4 (108-125cm)
CRP (mg/L)	2.45±2.30	3.42±2.54 p=0.037[a]	3.95±2.56 p=0.002[a]	5.52±2.36 p<0.001[a, b, c]
TBARS (µmol/L)	1.07±0.79	1.51±0.78 p=0.003[a]	1.54±0.73 p=0.002[a]	1.77±0.87 p<0.001[a]
TBARS- LDL (µmol/L)	0.92±0.25	1.12±0.27 p=<0.001[a]	1.17±0.26 p<0.001[a]	1.27±0.33 (p=(<0.001[a], 0.007[b])
Carbonyls (µmol/L)	15.88±6.54	20.04±7.46 p=0.002[a]	21.48±5.41 p<0.001[a]	22.04±5.26 p <0.001[a]
SOD (UI/mL)	53.91±13.42	44.68±15.67 p=0.001[a]	35.28±11.39 p<0.001[a, b]	35.41±11.32 p<0.001[a, b]
Catalase (UI/mL)	62.77±6.30	58.97±7.67 p=0.005[a]	58.46±7.22 p=0.001[a]	56.45±8.86 p=(<0.001[a], 0.022[b])

CRP: C-Reactive Protein; TBARS: Thiobarbituric acid reactive substances; SOD: Superoxide dismutase. Data are presented as means ±SD after analysis of variance (ANOVA) between quartiles. Means were compared using the Student's t-test. a: (Q2, Q3, Q 4) vs Q1; b(Q3, Q4) vs Q2; c: Q4 vs Q3. p< 0.05 was considered statistically significant.

Meats, poultry, eggs and fish intake was correlated negatively with SOD (r= -0.339, p<0.001) and catalase activities (r= -0.206, p= 0.002). However, a positive correlation was noticed between Fat intake and LDL-C (r= 0.345, p<0.001), TG (r=0.246, p<0.001), LAP (r=0.293, p<0.001) and a negative correlation with HDL-C (r=-0.396, p<0.001), LCAT activity (r= -0.275, p<0.001) and CRP (r= -0.315, p<0.001).

Sugar, and sugar products intake was correlated negatively with C-HDL (r= -0.228, p= 0.001), LCAT activity (r= -0.217, p= 0.001) and TG (r= -0.169, p= 0.010).

4 Discussion

This study was undertaken in postmenopausal women with abdominal obesity with the objective to assess the association between lifestyle behavior and cardiometabolic biomarkers.

After menopause, fat deposition, and accrual shift to favor the visceral depot that is accompanied by an increase in cardiometabolic risk reminiscent of that seen in men [1]. There is a synergistic relationship between food quality and physical activity associated with the cardiometabolic risk including dyslipidemia, inflammation, and oxidant-antioxidant status. In the current study, women had a total energy intake (TEI) less than 8 MJ/d which is below the recommendations of ANSES (2016) [25]. On the other hand, we recorded a high intake of protein portions balanced by a low intake of carbohydrates. According to the literature, this alteration in macronutrients composition despite stability in TEI leads to unfavorable changes for health and body weight [26-28]. We thought that the imbalance of these macronutrients could be responsible for maintaining android obesity and increasing metabolic syndrome (MetS).

Table 7: Relationship between lifestyle and biomarkers in postmenopausal women

	TEI	DEE	RM	Mets	Vit E	Vit C	Fibers	Fruits and vegetables	Meat. Poultry, eggs. Fishes	Fat	Sugar and sugar products
LDL-C	r=-0.206 p=0.002	-	-	r=-0.153 p=0.021	-	-	-	r=-0.279 p<0.001	-	r=0.345 p<0.001	-
HDL-C	r=-0.232 p<0.001	r=0.265 p<0.001	-	r= 0.161 p=0.015	-	-	-	r=0.140 p=0.035	-	r=0.396 p<0.001	r=-0.228 p=0.001
LCAT	-	-	r=-0.277 p<0.001	r=0.243 p<0.001	-	-	-	r=0.324 p<0.001	-	r= -0.275 p=<0.001	r=-0.217 p=0.001
TG	-	-	r=0.317 p<0.001	r= -0.442 p<0.001	-	-	-	-	-	r=0.264 p=<0.001	r=-0.169 p=0.010
LAP	-	-	r=0.486 p<0.001	r=-0.502 p<0.001	-	-	-	-	-	r=0.293 p=<0.001	-
CRP	-	-	-	r=-0.428 p<0.001	-	-	r=-0.335 p<0.001	-	-	r=-0.315 p=<0.001	-
TBARS	-	-	r=0.198 p=0.003	r=-0.367 p<0.001	-	-	r=-0.261 p<0.001	-	-	-	-
TBARS-LDL	-	-	r=0.455 p<0.001	r=-0.338 p<0.001	r=-0.278 p<0.001	r=-0.334 p<0.001	r=-0.153 p=0.017	r=-0.284; p=<0.001	-	-	-
Carbonyls	-	-	r=0.240 p<0.001	r=-0.349 p<0.001	-	-	r=-0.386 p<0.001	-	-	-	-
SOD	-	-	r=-0.410 p<0.001	r=0.374 p<0.001	-	r=0.388 p<0.001	r= 0.340 p<0.001	-	r=-0.339 p<0.001	-	-
Catalase	-	-	r=-0.178 p=0.007	r=0.283 p<0.001	-	-	r= 0.166 p= 0.012	-	r=-0.206 p=0.002	-	-

TEI: Total energy intake; DEE: Daily Energy Expenditure; RM: Resting Metabolism; Mets: Metabolic equivalent task. LCAT: Lecithin cholesterol acyltransferase; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; TG: Triacylglycerol; LAP: Lipid accumulation products; CRP: C-Reactive protein; TBARS: Thiobarbituric acid reactive substances; SOD: Superoxide dismutase. Correlations were performed by Pearson linear regression.

Overweight and obesity are closely related to eating habits. In the present study, women displayed a tendency to consume more meats, poultry, eggs, starchy food and refined cereals, milk and dairy products which explains the high amount of SFA and simple sugars. That leads also to high intake of animal proteins and refined carbohydrates, characterized by a high glycemic load and high SFA intake. These fatty acids may increase the risk of CVD by aggravating glucose intolerance, dyslipidemia, hypertriglyceridemia and the decrease in HDL-C [2]. According to Barbara *et al.*, these changes in habits appear during perimenopause [29]. The same authors reported that more than 50% of women have an increase consumption of sweets most often chocolate as well as fatty products by consumption of sandwiches. As a result of these new habits, 71% of women had weight gain during this period [29].

Globalization has participated in the change of eating habits [26], data confirmed that in many Mediterranean countries the loss of adherence to the Mediterranean diet (MD) is continuing and increasing linked also to the current economic downturn [27-30]. To remind that MD protects against cardiovascular diseases and is characterized by intake of olive oil, fruits, vegetables, whole cereals, legumes and nuts, moderate amounts of fish and dairy products and low quantities of meat and meat products [26]. The Mediterranean region is undergoing nutritional transition, while the traditional diet was based on healthy foods. Nowadays, individuals are consuming a more Western-influenced diet that contains empty calories. Globalization has disrupted the lifestyle and eating behavior of our society. Manufactured products were more consumed and have been replaced with a diet that contains more red meat, sweets, and processed foods. In this study, food intake was characterized by an unbalanced quantitative and qualitative distribution of macronutrients. Decreased whole grain and increased proteins consumption were noted. Macronutrient calorie distribution affects the health of individuals. The amount and quality of macronutrients may play a major role in WC [31]. Our results showed a negative correlation between meats, poultry, eggs, fish consumption and antioxidant enzymes activity. Chronic high protein intake leads to an increase in ROS generation causing toxicity [32], this toxicity could be the source of a decrease in SOD. In our study, LAP was positively correlated with fat intake. LAP, a novel index of central lipid accumulation based on a combination of WC and serum TG, is a good efficiency to identify metabolic syndrome independently of the classification used to detect it especially among women. It could be associated with a dysfunctional and highly lipolytic adipose tissue that is a central abnormality behind MS and associated conditions such as CVD [33]. A recent study has shown that 57.9% of postmenopausal women from the west of Algeria have MS [9].

We noted a moderate intake of vegetables and fruits which consumption decreased according to WC quartile. It is well established that fruits and vegetables contain vitamins, carotenoids, polyphenols and other still unknown bioactive compounds, making them a food group with high dietary antioxidant capacity. These compounds promote the scavenging of ROS produced during lipid peroxidation and other metabolic processes, limiting or preventing oxidative stress [34]. Fruits and vegetables intake was correlated negatively with LDL-C, LDL-

TBARS and positively with HDL-C and LCAT activity. LCAT is an enzyme converting free cholesterol into cholesteryl ester and raising the atheroprotective HDL-C [35].

Moreover, dietary fiber intake was correlated negatively with CRP, TBARS and carbonyls and positively with SOD. We suggest the existence of a synergic effect of dietary fibers and antioxidant compounds from fruits and vegetables. It has been shown that dietary fibers may have an effect on systemic inflammation by contributing to regulation of healthy body weight [36].

Vitamin C is a recognized antioxidant nutrient, with the ability to scavenge oxygen radicals. Furthermore, we observed that E and C vitamins correlated with LDL-TBARS. Vit C also correlated positively with SOD. The two vitamins could be used to prevent the onset of various disorders associated with an age-related decrease in estrogen. These vitamins scavenge free radicals and neutralize oxidative stress [34].

MUFA are recognized as healthy fatty acids that contribute to lowering LDL-C and improve HDL-C, which in turn can lower the risk of CVD [37]. MUFA are present in olive oil, a traditional product of the Mediterranean basin. However, in our study, MUFA were essentially from manufactured dishes based on poultry, soups and cereal, which were accompanied by saturated fatty acids (SFA) and empty calories that promote visceral adiposity.

An inverse relationship was found between TEI and; HDL-C and with LDL-C. On the other hand, DEE was correlated with HDL-C. Women were sedentary; Mets was negatively correlated with LDL-C, LAP, CRP, TBARS, TBARS-LDL and carbonyls and positively with LCAT, SOD and catalase. Physical activity promotes benefits on lipid profile [38,39] for which the positive effect on biomarkers reducing CRP and interleukin were found in obese postmenopausal women [40]. Moreover, it has been shown that physical activity improves LCAT activity [41] which is positively correlated with SOD activity and the level of CRP [35].

Our findings demonstrate that an unhealthy diet and sedentary lifestyle were associated with cardiometabolic risk in postmenopausal women. The early fight against overweight and obesity by the adoption of a healthy lifestyle based on regular physical activity and a balanced diet with principles of MD are the best strategies for CVD prevention.

Author contribution: All authors approved the final version before submission, have read and agreed to the published version of the manuscript.
Funding: This research received no external funding.
Conflict of interest: The authors declare no conflicts of interest.

ORCID
Amina TIALI: https://orcid.org/0000-0002-8060-9048
Djahida CHENNI: https://orcid.org/0000-0002-0780-4373
Mohamed BENYOUB: https://orcid.org/0000-0002-0140-0888
Khedidja MEKKI: https://orcid.org/0000-0001-8783-3088

References

1. Newson, L. (2018). Menopause and cardiovascular disease. *Post Reproductive Health*, *24*(1), 44-49. https://doi.org/10.1177/2053369117749675
2. Ortega, F. B., Lavie, C. J., & Blair, S. N. (2016). Obesity and cardiovascular disease. *Circulation Research*, *118*(11), 1752-1770. https://doi.org/10.1161/circresaha.115.306883
3. Dworatzek, E., & Mahmoodzadeh, S. (2017). Targeted basic research to highlight the role of estrogen and estrogen receptors in the cardiovascular system. *Pharmacological Research*, *119*, 27-35. https://doi.org/10.1016/j.phrs.2017.01.019
4. Ko, S., & Kim, H. (2020). Menopause-associated lipid metabolic disorders and foods beneficial for postmenopausal women. *Nutrients*, *12* (1), 202. https://doi.org/10.3390/nu12010202
5. Taleb-Belkadi, O., Chaib, H., Zemour, L., Fatah, A., Chafi, B., & Mekki, K. (2016). Lipid profile, inflammation, and oxidative status in peri- and postmenopausal women. *Gynecological Endocrinology*, *32*(12), 982-985. https://doi.org/10.1080/09513590.2016.1214257
6. Duval, K., Prud'homme, D., Rabasa-Lhoret, R., Strychar, I., Brochu, M., Lavoie, J.-M., & Doucet, É. (2014). Erratum : Effects of the menopausal transition on energy expenditure : A MONET group study. *European Journal of Clinical Nutrition*, *68*(1), 142-142. https://doi.org/10.1038/ejcn.2013.246
7. Stelmach-Mardas, M., Rodacki, T., Dobrowolska-Iwanek, J., Brzozowska, A., Walkowiak, J., Wojtanowska-Krosniak, A., Zagrodzki, P., Bechthold, A., Mardas, M., & Boeing, H. (2016). Link between food energy density and body weight changes in obese adults. *Nutrients*, *8*(4), 229. https://doi.org/10.3390/nu8040229
8. Zhu, D., Chung, H., Dobson, A. J., Pandeya, N., Anderson, D. J., Kuh, D., Hardy, R., Brunner, E. J., Avis, N. E., Gold, E. B., El Khoudary, S. R., Crawford, S. L., & Mishra, G. D. (2020). Vasomotor menopausal symptoms and risk of cardiovascular disease: A pooled analysis of six prospective studies. *American Journal of Obstetrics and Gynecology*. https://doi.org/10.1016/j.ajog.2020.06.039
9. Khalfa, A., Tiali, A., Zemour, L., Fatah, A., & Mekki, K. (2017). Prevalence of metabolic syndrome and its association with lifestyle and cardiovascular biomarkers among postmenopausal women in western Algeria. *International Journal of Gynecology & Obstetrics*, *138*(2), 201-206. https://doi.org/10.1002/ijgo.12206
10. Siti, H. N., Kamisah, Y., & Kamsiah, J. (2015). The role of oxidative stress, antioxidants and vascular inflammation in cardiovascular disease (a review). *Vascular Pharmacology*, *71*, 40-56. https://doi.org/10.1016/j.vph.2015.03.005
11. Saltiel, A. R., & Olefsky, J. M. (2017). Inflammatory mechanisms linking obesity and metabolic disease. *Journal of Clinical Investigation*, *127*(1), 1-4. https://doi.org/10.1172/jci92035
12. Marseglia, L., Manti, S., D'Angelo, G., Nicotera, A., Parisi, E., Di Rosa, G., Gitto, E., & Arrigo, T. (2014). Oxidative stress in obesity: A critical component in human diseases. *International*

Journal of Molecular Sciences, *16*(1), 378-400. https://doi.org/10.3390/ijms16010378

13. Au, A., Feher, A., McPhee, L., Jessa, A., Oh, S., & Einstein, G. (2016). Estrogens, inflammation and cognition. *Frontiers in Neuroendocrinology*, *40*, 87-100. https://doi.org/10.1016/j.yfrne.2016.01.002
14. Warwick, P. M., Edmundson, H. M., & Thomson, E. S. (1988). Prediction of energy expenditure: Simplified fao/Who/Unu factorial method vs continuous respirometry and habitual energy intake. *The American Journal of Clinical Nutrition*, *48*(5), 1188-1196. https://doi.org/10.1093/ajcn/48.5.1188
15. Black, A. E., Coward, W. A., Cole, T. J., & Prentice, A. M. (1996). Human energy expenditure in affluent societies: an analysis of 574 doubly-labelled water measurements. *European Journal of Clinical Nutrition*, *50*(2), 72–92.
16. Ainsworth, B. E., Haskell, W. L., Herrmann, S. D., Meckes, N., Bassett, D. R. J., Tudor-Locke, C., Greer, J. L., Vezina, J., Whitt-Glover, M. C., & Leon, A. S. (2011). 2011 Compendium of Physical Activities : A Second Update of Codes and MET Values. *Medicine & Science in Sports & Exercise*, *43*(8), 1575–1581. https://doi.org/10.1249/MSS.0b013e31821ece12
17. Burstein, M., Scholnick, H. R., & Morfin, R. (1970). Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. *Journal of Lipid Research*, *11*(6), 583-595.
18. Kahn, H. S. (2005). The "lipid accumulation product" performs better than the body mass index for recognizing cardiovascular risk: A population-based comparison. *BMC Cardiovascular Disorders*, *5*(1). https://doi.org/10.1186/1471-2261-5-26
19. Albers, J. J., Chen, C., & Lacko, A. G. (1986). Isolation, characterization, and assay of lecithin-cholesterol acyltransferase. *Methods in Enzymology*, 763-783. https://doi.org/10.1016/0076-6879(86)29103-x
20. Clauss A. Gerinnungsphysiologische Schnellmethode zur Bestimmung des Fibrinogens [Rapid physiological coagulation method in determination of fibrinogen]. Acta Haematol. 1957 Apr;17(4):237-46. German. https://doi.org/10.1159/000205234.
21. Quintanilha, A. T., Packer, L., Davies, J. M., Racanelli, T. L., & Davies, K. J. (1982). Membrane effects of vitamin E deficiency: Bioenergetic and surface charge density studies of skeletal muscle and liver mitochondria. *Annals of the New York Academy of Sciences*, *393*(1 Vitamin E), 32-47. https://doi.org/10.1111/j.1749-6632.1982.tb31230.x
22. Levine, R. L., Garland, D., Oliver, C. N., Amici, A., Climent, I., Lenz, A., Ahn, B., Shaltiel, S., & Stadtman, E. R. (1990). [49] determination of carbonyl content in oxidatively modified proteins. *Oxygen Radicals in Biological Systems Part B: Oxygen Radicals and Antioxidants*, 464-478. https://doi.org/10.1016/0076-6879(90)86141-h
23. Marklund, S., & Marklund, G. (1974). Involvement of the superoxide anion radical in the Autoxidation of pyrogallol and a convenient assay for superoxide Dismutase. *European Journal of Biochemistry*, *47*(3), 469-474. https://doi.org/10.1111/j.1432-1033.1974.tb03714.x
24. Aebi, H. (1974). Catalase. *Methods of Enzymatic Analysis*, 673-684. https://doi.org/10.1016/b978-0-12-091302-2.50032-3
25. ANSES. (2016). French food composition table. *French Agency for Food, Environnemental and Occupationnel health and safety*. Available at https://ciqual.anses.fr
26. Widmer, R. J., Flammer, A. J., Lerman, L. O., & Lerman, A. (2015). The Mediterranean diet, its components, and cardiovascular disease. *The American Journal of Medicine*, *128*(3), 229-238. https://doi.org/10.1016/j.amjmed.2014.10.014
27. Bonaccio, M., Di Castelnuovo, A., Bonanni, A., Costanzo, S., De Lucia, F., Persichillo, M., Zito, F., Donati, M., De Gaetano, G., & Iacoviello, L. (2014). Decline of the Mediterranean diet at a time of economic crisis. Results from the moli-sani study. *Nutrition, Metabolism and Cardiovascular Diseases*, *24*(8), 853-860. https://doi.org/10.1016/j.numecd.2014.02.014
28. Hall, K. D., Heymsfield, S. B., Kemnitz, J. W., Klein, S., Schoeller, D. A., & Speakman, J. R. (2012). Energy balance and its components: Implications for body weight regulation. *The American Journal of Clinical Nutrition*, *95*(4), 989-994. https://doi.org/10.3945/ajcn.112.036350
29. Barbara, M.M., Hans, W.H., Claudia, I.R., Georg, K., Harisson, G.P., Johann, K. (2013). The Menopausal Transition – A Possible Window of Vulnerability for Eating Pathology. *Int J Eat Disord*, 46: 609-616. https://doi.org/10.1002/eat.22157
30. Bach-Faig, A., Berry, E. M., Lairon, D., Reguant, J., Trichopoulou, A., Dernini, S., Medina, F. X., Battino, M., Belahsen, R., Miranda, G., & Serra-Majem, L. (2011). Mediterranean diet pyramid today. Science and cultural updates. *Public Health Nutrition*, *14*(12A), 2274-2284. https://doi.org/10.1017/s1368980011002515
31. Vergnaud, A., Norat, T., Mouw, T., Romaguera, D., May, A. M., Bueno-de-Mesquita, H. B., Van der A, D., Agudo, A., Wareham, N., Khaw, K., Romieu, I., Freisling, H., Slimani, N., Perquier, F., Boutron-Ruault, M., Clavel-Chapelon, F., Palli, D., Berrino, F., Mattiello, A., … Peeters, P. H. (2013). Macronutrient composition of the diet and prospective weight change in participants of the EPIC-PANACEA study. *PLoS ONE*, *8*(3), e57300. https://doi.org/10.1371/journal.pone.0057300
32. Van Raamsdonk, J. M. (2015). Levels and location are crucial in determining the effect of ROS on lifespan. *Worm*, *4*(4), e1094607. https://doi.org/10.1080/21624054.2015.1094607
33. Taverna, M. J., Martínez-Larrad, M. T., Frechtel, G. D., & Serrano-Ríos, M. (2011). Lipid accumulation product: A powerful marker of metabolic syndrome in healthy population. *European Journal of Endocrinology*, *164*(4), 559-567. https://doi.org/10.1530/eje-10-1039
34. Hermsdorff, H. H., Barbosa, K. B., Volp, A. C., Puchau, B., Bressan, J., Zulet, M. Á., & Martínez, J. A. (2011). Vitamin C and fibre consumption from fruits and vegetables improves oxidative stress markers in healthy young adults. *British Journal*

of Nutrition, *107* (8), 1119-1127. https://doi.org/10.1017/s0007114511004235

35. Dullaart, R. P., Tietge, U. J., Kwakernaak, A. J., Dikkeschei, B. D., Perton, F., & Tio, R. A. (2014). Alterations in plasma lecithin: cholesterol acyltransferase and myeloperoxidase in acute myocardial infarction: Implications for cardiac outcome. *Atherosclerosis*, *234*(1), 185-192. https://doi.org/10.1016/j.atherosclerosis.2014.02.026
36. Saijo, H., Hirohashi, Y., Torigoe, T., Horibe, R., Takaya, A., Murai, A., Kubo, T., Kajiwara, T., Tanaka, T., Shionoya, Y., Yamamoto, E., Maruyama, R., Nakatsugawa, M., Kanaseki, T., Tsukahara, T., Tamura, Y., Sasaki, Y., Tokino, T., Suzuki, H., Kondu T., Takahashi H., Sato, N. (2016). Plasticity of lung cancer stem-like cells is regulated by the transcription factor HOXA5 that is induced by oxidative stress. *Oncotarget*, *7*(31), 50043-50056. https://doi.org/10.18632/oncotarget.10571
37. Mensink, R. P., & World Health Organization. (2016). *Effects of saturated fatty acids on serum lipids and lipoproteins : A systematic review and regression analysis*. World Health Organization; WHO IRIS. Available at URL address: https://apps.who.int/iris/handle/10665/246104
38. Loprinzi, P. D., & Addoh, O. (2016). The association of physical activity and cholesterol concentrations across different combinations of central adiposity and body mass index. *Health Promotion Perspectives*, *6*(3), 128-136. https://doi.org/10.15171/hpp.2016.21
39. Lira, F. S., Yamashita, A. S., Uchida, M. C., Zanchi, N. E., Gualano, B., Martins, E., Caperuto, E. C., & Seelaender, M. (2010). Low and moderate, rather than high intensity strength exercise induces benefit regarding plasma lipid profile. *Diabetology & Metabolic Syndrome*, *2*(1). https://doi.org/10.1186/1758-5996-2-31
40. Imayama, I., Ulrich, C. M., Alfano, C. M., Wang, C., Xiao, L., Wener, M. H., Campbell, K. L., Duggan, C., Foster-Schubert, K. E., Kong, A., Mason, C. E., Wang, C., Blackburn, G. L., Bain, C. E., Thompson, H. J., & McTiernan, A. (2012). Effects of a caloric restriction weight loss diet and exercise on inflammatory biomarkers in overweight/Obese postmenopausal women: A randomized controlled trial. *Cancer Research*, *72*(9), 2314-2326. https://doi.org/10.1158/0008-5472.can-11-3092
41. Belalcazar, L. M., Haffner, S. M., Lang, W., Hoogeveen, R. C., Rushing, J., Schwenke, D. C., Tracy, R. P., Pi-Sunyer, F. X., Kriska, A. M., & Ballantyne and the Look AHEAD Actio, C. M. (2013). Lifestyle intervention and/or statins for the reduction of C-reactive protein in type 2 diabetes: From the look AHEAD study. *Obesity*, *21*(5), 944-950. https://doi.org/10.1002/oby.20431

Cite this article as: Tiali, A., Chenni, D., Benyoub, M., & Mekki, K. (2020). Cross-sectional association between lifestyle behavior and cardiometabolic biomarkers in west Algerian postmenopausal women 2020. *The North African Journal of Food and Nutrition Research,* 4(9): S7-S16. https://doi.org/10.51745/najfnr.4.9.S7-S16