Progress in Nutrition 2025; Vol. 27, N. 4: 17968 DOI: 10.23751/pn.v27i4.17968

Original article

Healthy lifestyle promotion improves cardiometabolic markers in premenopausal women with abdominal obesity

Azzeddine Senouci[1,2], Latifa Imen Benharrat[2], Wassila Benhabib[3], Rahmouna Fizi[4]

[1]Department of Agri-Food, Faculty of Science and Technology, University of Ain Temouchent-Belhadj Bouchaib, Ain Temouchent, Algeria; [2]Laboratory of Clinical and Metabolic Nutrition, Faculty of Natural and Life Sciences, University Oran 1, Oran, Algeria; [3]Department of Epidemiology, University Hospital of Oran, Oran, Algeria; [4]Department of Public Health, Public Health Establishment of Proximity Es Senia, Oran, Algeria.

Abstract. *Background and aims:* To assess the effect of the Mediterranean diet (MD) pattern on lipid profile, inflammatory markers, and markers of oxidative stress in perimenopausal women with abdominal obesity. *Material and methods:* Ninety women (48±3 years) with abdominal obesity (waist circumference 105.48±12.33 cm) were randomly assigned to two groups: 45 received nutritional counseling in accordance with MD and the practice of regular activity for 8 weeks, and 45 women were used as controls. At baseline (T0) and 8 weeks after the initiating of nutritional intervention (T8), we assessed cardiometabolic biomarkers. *Results:* Compared to the control group, women in the intervention group showed overall improvements in lipid profile, with reductions in total cholesterol and a better TC/HDL-C ratio. Inflammatory status improved through a decrease in TNF-α levels, while oxidative stress was reduced, as reflected by lower lipid peroxidation and enhanced antioxidant defenses. Other metabolic markers remained largely unchanged. *Conclusions:* MD pattern improves dyslipidemia, inflammation and oxidative stress and represent the best choice a particularly relevant nutritional approach for managing cardiometabolic risk and prevent cardiovascular disease in perimenopausal women.

Key words: abdominal obesity, perimenopausal women, mediterranean diet, lipid profile, inflammation, oxidative status.

Background and Aims

The perimenopause or menopausal transition is the transitional period before menopause. Oestrogen insufficiency is associated with alterations in endocrine, biological, and clinical features (1). Perimenopause can last anywhere from a few months to 10 years. It's characterized by a progressive decline in oestrogen production, a menstrual cycle irregularities and vasomotor symptoms (2). During this time, a number of symptoms, such as hot flushes, night sweats, sadness, irritability, headaches, sleep difficulties and cognitive impairment, may happen more frequently (3). Key risk variables of cardiovascular disease (CVD) have been found to deteriorate throughout the menopause transition, independently of the effects of aging alone. As a result, it is believed that the transition through menopause has a significant role in the development of CVD in women (4). Moreover, the perimenopause is a time of significant metabolic alterations linked to elevated atherogenic lipid profiles, impaired inflammatory profiles, and insulin resistance, all of which are known to raise the risk of CVD (5). In this period, type 2 diabetes, a major risk factor for cardiovascular disease, also increases (6). The most common abnormality during perimenopause is the high waist circumference.

Cardiometabolic syndrome is associated with central obesity and chronic low-grade inflammation. It's established that visceral adiposity is generally associated with increased plasma C-reactive protein (CRP), an inflammatory marker that predicts CVD. CVD is also associated with high blood pressure, which is more prevalent during menopause (7). Also, numerous studies suggested links between some menopausal symptoms (hot flashes, insomnia) and CVD (4). Studies showed that vasomotor symptoms are linked to hypertension (8), insulin resistance, diabetes (9) and dyslipidemia. The proinflammatory profile and oxidative stress were associated with climacteric symptoms (10).

In CVD pathogenesis, oxidative stress plays a crucial role. Since the menopausal transition and up to the post menopause, oxidative stress is primarily caused by oestrogen deprivation in women (11). Oestrogen is a potent antioxidant that inhibits lipid peroxidation, thus when it is lacking after menopause, OS increases. Reactive oxygen species (ROS) (free radicals) generation and antioxidant defense are out of balance during oxidative stress (12). Nearly all of the key cellular components, including proteins, DNA, and membrane lipids, can be damaged by oxidative stress, which may induce cell death (13). The impact of lifestyle changes in lowering cardiometabolic risk and promoting women's health during menopause is becoming more well acknowledged (14). The Mediterranean diet (MD) is an effective strategy for controlling and reducing cardiometabolic risk in this situation (15). It has been showed that the MD reduces the overweight, prevent the development, of metabolic syndrome, diabetes and obesity. Inflammatory markers, lipids, and the waist-to-hip ratio are just a few of the cardiovascular disease risk factors that this diet is known to reduce (16). Many nations around the Mediterranean Sea follow the MD as a traditional form of nutrition. The MD model, highlighted for its benefits in the prevention of cardiometabolic diseases, is a reference for nutritional education and a guide for food choices in public health prevention (17). It is distinguished by a profusion of plant-based products; rich in fruits, vegetables and fibers, the preponderance of monounsaturated fatty acids (olive oil) and polyunsaturated fatty acids (omega 3), whole grain cereals, consumption of fatty fish (sardines, mackerel, tuna, etc.), and a limited number of animal-based products. Moderate consumption of red meat and dairy products characterize the diet (18). We carried out this study in women on menopausal transition with abdominal obesity to evaluate the impact of a lifestyle promotion based on the principles of Mediterranean eating patterns on, lipid profile, inflammation, and oxidative stress markers.

Material and Methods

Study population

A prospective randomised study was conducted at the Public Health Establishment Es-Sénia, Oran (west of Algeria). A total of 110 perimenopausal women with abdominal obesity were selected for the study. The midwife and gynecologist both confirmed the diagnosis of perimenopause. The menopause rating scale (MRS) was used to evaluate perimenopausal symptoms. MRS is an instrument which is a validated scale and has been used in research on the etiology of menopausal symptoms to assess their severity (19). Women were included on the basis that have a clear perimenopausal diagnosis and no received nutritional counselling (Table 1). We excluded women on hormone therapy to relieve climacteric syndrome and those taking antioxidant supplements and with renal failure and unstable cardiovascular disease. Before beginning the study, all of the women were informed about the objective of the study and given written consent. Ethical approval was obtained from the Institutional Ethics Committee of the University of Oran 1 Ahmed Ben Bella, Oran, Algeria, (Protocol Code: SDRF-2022-18).

Nutritional intervention

From the 110 women recruited, 90 (48±3 years) were available for the study. Randomisation was performed using the formula $N = (Z\alpha/2+Z\beta)2*(\sigma12 + \sigma22)/(\mu1 - \mu2)2$. After adding 20% for dropouts, the minimum required sample size was 45 women in each group; the intervention group (IG; n = 45) received dietary advice, while the control group (CG; n = 45) continued their usual diet. The intervention group

Table 1. Clinical characteristics of perimenopausal women at baseline.

Variables	All subjects (n=90)
Weight (kg)	78.10 ± 13.79
Height (m)	1.60 ± 0.07
BMI (weight (kg) / height (m^2)	30.43 ± 6,38
Waist circumference (cm)	105.48 ± 12.33
Hip circumference (cm)	111.94 ± 10.73
Waist/hip circumference	0.94 ± 0.072
Menstrual cycle (days)	128.24 ± 139.03
Menstrual duration (days)	5.31 ± 2.56
MRS Score*	24 ± 7
Urea (mmol/L)	6.96 ± 2.35
Creatinine (mmol/mL)	0.82 ± 0.24

Data were expressed as mean ± standard error. *Abbreviations:* BMI: body mass index (weight kg/size m^2). MRS: Menopause Rating Scale. *All women have elevated MRS score, which indicates severe vasomotor symptoms.

received nutritional counselling adapted to the Mediterranean diet for eight weeks. Women were instructed to eat virgin olive oil for seasoning, wholegrain cereals (50 grams of bread at each meal, 250 grams of cereal or carbohydrate once day), fruit (once daily), vegetables (200 grams twice daily), and oil fish (twice a week) in order to meet this goal (16). We asked women to avoid sugary products, all foods with empty calories as well as ultra-processed products. Patients also received recommendations for healthier cooking methods. Women were encouraged to practice regular physical activity at least 30 minutes of walking per day.

Methods

All surveys and biochemical analyses were conducted at the beginning of the nutritional intervention (T0) and eight weeks later (T8).

Biochemical analysis

Antecubital venepuncture was used to take blood samples from all patients at the beginning (T0) and eight weeks (T8) after the start of the nutritional intervention. For the biochemical tests, lithium heparin-filled tubes were utilized. By low-speed centrifugation at 3000X g, at 4°C for 15 minutes, we obtained serum. The serum was taken out, divided up, and kept at -20° C.

- **Lipid profile**

  The colorimetric methods (Kits Biolabo, France) was used to determine the blood glucose, urea, creatinine, triacylglycerols (TG), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C) and the Friedwald formula (20) was used to determine the low-density lipoprotein cholesterol (LDL-C).

  For women, lipid accumulation products (LAP) are [(WC (cm) − 58)*(TG (mmol/l)]. LAP is a measure of central lipid accumulation used to gauge the likelihood of developing metabolic syndrome. The minimal WC values from NHANES III (58 cm for women) are included in the formula (Third National Health and Nutrition Examination Survey) (21).
- **Markers of inflammation**

  Utilizing CRP-Latex, C-reactive protein (CRP) was quantified (Spinreact, Spain). The CRP-latex test uses slide agglutination to detect C-reactive protein (CRP) in human serum in a qualitative and semi-quantitative manner. When combined with CRP-containing samples, latex particles coated with goat IgG cause agglutination.

  Tumor Necrosis Factor Alpha (TNF-α) was determined in duplicate samples with a commercial enzyme-linked immunometric assay kit (ELISA, My BioSource) with a range of 7.81 − 500 pg/mL.
- **Lipid and protein oxidation**

  The NWLSS™ Malondialdehyde (MDA) Assay was used to quantify lipid peroxidation as the concentration of MDA. The NWK-MDA01 assay is based on the reaction of MDA with thiobarbituric acid (TBA); forming an MDA-TBA2 adduct that absorbs strongly at 532 nm.

  Oxidized proteins were estimated by measuring carbonyls concentration according to the method of Levine et al. (1990)

(22) using the 2,4-dinitrophenylhydrazine (DNPH).

- **Antioxidant**

Superoxide dismutase (SOD) is an enzymatic antioxidant which neutralizes superoxide anion, one of the most potent ROS. SOD activity was measured by the method of Marklund. (1974) (23). This method utilizes the inhibition of auto-oxidation of pyrogallol by SOD.

Catalase enzyme activity was measured using the Goth. (1991) [24] method. A stable complex with ammonium molybdate was absorbed at 405 nm.

Thiols and total *glutathione* were determined by a Sedlak and Lindsay. (1968) (25) method, based on the oxidation reaction of -SH groups with 5,5'-dithiobis-2-nitrobenzoic acid (DTNB), thereby releasing thionitrobenzoic acid (TNB) of yellow color, which was absorbed at 412nm.

Statistical analysis

Statistical analysis was performed using SPSS 20.0 (IBM SPSS Statistics, Armonk, NY). Data were expressed as the mean ± SD (standard deviation). Differences between groups were analysed using Mann-Whitney test and Student's t-test and differences within groups at different time points were analysed using Wilcoxson's test. The levels of $p < 0.05$ were considered significant. Data were compared twice: the intervention group compared with the control group (*) and over times (#) T8 compared with T0.

Results

Lipid profile (Table 2)

In the IG compared to the CG, no significant differences were noted in glycemia and TG concentrations. A decrease by (-9%) in blood glucose was noted in the IG compared to T0 ($p<0.01$). A decrease was noted by (-14%) in TG concentrations at T8 compared to T0 ($p<0.001$). In the IG compared to the CG, TC were decreased by (-16%) at T8 ($p<0.01$). These values were also diminished by (-10%) compared to T0 ($p<0.001$). A significant increase in HDL-C concentrations was noted at T8 by (+18%) compared to T0 in the IG ($p<0.05$). However, the LDL-C concentrations remained unchanged in both groups. The TC/HDL-C ratio was diminished by (-21%) in the IG compared to the CG ($p < 0.05$) and by (-23%) at T8 compared to T0 ($p<0.05$).

No difference was noted in LAP values in the IG compared to the CG. While a decrease by (-12%) was noted in LAP values at T8 ($p<0.001$) compared to T0 in the IG.

Table 2. Changes in lipid profile after nutritional intervention.

Variables	Intervention group		Control group	
	T0	T8	T0	T8
Glucose (mmol/L)	5.71 ± 1.49	5.23 ± 0.8##	5.09 ± 1.13	5.24 ± 1.03
TG (mmol/L)	2.24 ± 0.28	1.96 ± 0.26###	1.83 ± 0.67	1.84 ± 0.38
TC (mmol/L)	4.46 ± 1.03	4.05 ± 1.06**###	4.72 ± 1.10	4.73 ± 0.93
HDL-C (mmol/L)	0.94 ± 0.42	1.16 ± 0.41#	0.89 ± 0.40	1.01 ± 0.36
LDL-C (mmol/L)	1.35 ± 0.97	1.20 ± 0.77	1.63 ± 0.84	1.50 ± 0.90
TC/HDL-C	5.08 ± 2.52	4.12 ± 1.51*#	5.83 ± 2.64	5.00 ± 1.81
LAP	105.48 ± 28.9	94.02 ± 27.89###	100.64 ± 34.29	103.02 ± 20

Abbreviations: TG: Triacylglycerols; TC: Total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; LAP: lipid accumulation product. T0: the beginning of study; T8: 8 weeks after the after initiating nutritional intervention. Data are expressed as mean ± standard deviation and were analysed using Wilcoxon's test, Mann-Whitney test and Student's t-test. *IG *vs* CG; #: T8 *vs* T0. *#P<0.05, **##p<0.01, ###***p<0.001.

Inflammatory and pro-oxidant antioxidant markers (Table 3)

No significant variation was observed in CRP concentrations in the IG group compared to the CG group at T8. Nevertheless, in the intervention group, CRP values decreased by (-83%) at T8 compared to T0 ($p<0.05$). In the IG compared to the CG, a significant decrease in TNF-α by (-61%) was noted at T8 ($p<0.01$). TNF-α concentrations were also reduced by (-65%) at T8 ($p<0.05$) compared to T0 in the IG. For pro-oxidant status, results showed that after 8 weeks of nutritional counselling, the level of lipid oxidative products (MDA) was decreased by (-59%) in the IG compared to the CG ($p<0.001$). No significant difference was noted in MDA concentrations at T8 compared to T0. Carbonyls concentrations remained unchanged in both groups. For antioxidant defence, superoxide dismutase activity was increased in the IG compared to the CG after 8 weeks of nutritional counselling. This increase was by (+7%) compared to the CG at T8 ($p<0.05$). Likewise, we noted a significant increase in SOD activity in the IG by (+6%) at T8 ($p<0.01$), compared to T0. A significant increase in catalase activity by (+31%) was noted in the IG compared to the CG at T8 ($p<0.05$). This activity was also increased by (+22%) at T8 ($p<0.05$), compared to T0. Thiol values were enhanced in the IG compared to the CG ($p<0.001$). A significant increase of thiol concentrations by (+10%) was observed in the IG at T8, compared to T0 ($p<0.001$). Total *Glutathione* values remained unchanged in the both groups during all nutritional intervention.

Discussion

This study was undertaken in order to evaluate the effect of nutritional counselling based on the MD pattern on, lipid profile, inflammation and oxidative status. During follow-up, serum biomarkers were assessed at baseline and 8 weeks after initiating intervention. The increase in cardiovascular risk at menopause may be a consequence of oestrogen deprivation or results from a higher prevalence of cardiovascular risk factors such as visceral obesity, insulin resistance, dyslipidemia and endothelial dysfunction which occur with aging (26). During the perimenopause phase, the cardiovascular risk alterations start to occur. When a woman enters menopause, her lipid profile changes, with slightly lower HDL levels and 10-15% higher LDL and triglyceride levels. The latter were confirmed by our results which were unbalanced at baseline and also confirmed by the results conducted by Taleb *et al.* (2016) (11). Because oestrogen and androgen receptors are present in visceral and subcutaneous adipocytes, previous investigations have shown that endogenous sex hormones can affect

Table 3. Changes in inflammatory and pro-antioxidant markers after nutritional intervention.

	Intervention group		Control group	
Variables	**T0**	**T8**	**T0**	**T8**
CRP (mg/L)	3.60 ± 7.64	0.60 ± 1.85#	2.40 ± 5.97	1.50 ± 5.46
TNFα (pg/mL)	43.03 ± 31.75	26.01 ± 21.00**#	35.56 ± 22.27	42.09 ± 20.2
MDA (mmol/L)	9.95 ± 6.50	6.75 ± 2.75***	14.88 ± 6.08	10.77 ± 4.08
Carbonyls (µmol/L)	39.23 ± 22.72	32.86 ± 17.44	42.32 ± 15.45	40.83 ± 9.78
SOD (U/mL)	39.54 ± 2.67	42.47 ± 5.13*##	39.37 ± 2.62	39.31 ± 2.59
Catalase (KU/L)	43.72 ± 21.51	56.74 ± 26.38*#	48.24 ± 23.98	39.06 ± 29.1
Thiols (µmol/L)	25.18 ± 10.29	28.2±12.5*** ###	12.65 ± 7.70	15.65 ± 8.91
Total Glutathion (µmol/L)	3.27 ± 2.16	4.63 ± 2.94	4.00 ± 1.93	3.43 ± 1.06

Abbreviations: CRP: C-reactive protein; TNF-a: Tumor necrosis factor-alpha; MDA: malondialdehyde; SOD: superoxide dismutase; CAT: catalase. T0: the beginning of study; T8: 8 weeks after the after initiating nutritional intervention. Data are expressed as mean ± standard deviation and were analysed using Wilcoxon's test, Mann-Whitney test and Student's t-test. *IG *vs* CG; #: T8 *vs* T0. *#P<0.05, **##p<0.01, ###***p<0.001.

the lipid profile in premenopausal and postmenopausal women (27). In our study, nutritional counselling showed a beneficial effect on the levels of cholesterol, triglycerides, LDL-C, lipid accumulation product and HDL/CT. This beneficial effect was confirmed by a decrease in these parameters in the intervention group. Furthermore, a favourable effect was also noted in HDL-C levels. Our results were similar to those of Silva *et al.* (2021) (28). According to a meta-analysis of more than 50,000 individuals, the MD decreased the risk of metabolic syndrome and shielded against risk markers like waist circumference, lipids, glucose, and blood pressure (29). In menopausal women it is recognized that inflammation is the fundamental mediator of CVD leading to oxidative stress. Loss of oestrogen activates the production of pro-inflammatory cytokines and increases the levels of reactive oxygen species (30). In our study, we found that CRP and TNF-α were higher at baseline. Our results were similar to the result conducted by Silva *et al.* (2021) (28). This rise was explained by the fact that lowering levels of ovarian steroidal hormones at menopausal transition were accompanied by greater levels of the interleukins IL-6, sIL-6, IL-4, IL-2, and tumor necrosis factor (TNF-α), which are all reversible by hormone therapy in postmenopausal women (31). A decrease in CRP and TNF-α was noted in the intervention group at T8. Healthy foods with anti-inflammatory and antioxidant qualities make up MD. Additionally, isoflavones may be advantageous due to their anti-inflammatory and antioxidant characteristics and the ability of the gut bacteria to produce the active metabolite equol from daidzin/daidzein (32). Moreover, MD encourages a high intake of polyphenols and n-3 fatty acids with anti-inflammatory and antioxidant effects as well as a decrease in the consumption of saturated animal fats in favor of unsaturated vegetable fats (33). In addition, it has been noted that oxidative stress, which is defined as an imbalance between the generation of reactive oxygen species (free radicals) and antioxidant defense, is common during the menopause and can result in serious harm (34). Nutritional counselling shown a beneficial effect on reduction of lipid oxidative products levels and the carbonyls concentrations. Moreover, an improvement of in antioxidant enzymes activities as SOD, catalase, total glutathione and thiols. This beneficial effect was confirmed by increase in this parameter at T8 in the intervention group compared to the control group. Extra virgin olive oil, whole grain cereals, nuts, legumes, vegetables, red wine, and fruits all contain phenolic chemicals (polyphenols). These Mediterranean foods may constitute an optimum nutritional pattern during menopause because of their anti-inflammatory and antioxidant qualities (35). Indeed, it has been discovered that the diet and, to a lesser extent, its constituents, lower the risk of cardiovascular disease through a variety of mechanisms, including a decrease in blood pressure, lipids, endothelial dysfunction, glucose, BMI, and waist circumference, as well as increased NO bioavailability, antioxidant properties, and anti-inflammatory effects (16). This study presents some limitations that should be acknowledged. First, the follow-up duration was relatively short (8 weeks), which may not fully capture the long-term effects of adopting a Mediterranean diet and regular physical activity on cardiometabolic markers. Second, adherence to dietary recommendations and physical activity was not objectively monitored, which may introduce inaccuracies in assessing the true level of compliance. Additionally, the sample size was relatively small, potentially limiting the statistical power and the generalizability of the findings. Finally, the recruitment of volunteers from a single health establishment may introduce selection bias, which should be considered when interpreting the results.

Conclusion

In conclusion our results indicate that a lifestyle based on a Mediterranean diet and regular physical activity improves inflammatory status, lipid peroxidation and increases the activity of antioxidant enzymes and helps prevent cardiometabolic complications in perimenopausal women. The combination of these Mediterranean foods could be the optimum dietary pattern for menopause.

Acknowledgements: Our thanks to the volunteers who participated in the study.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Ethics Approval and Consent to Partici pate: The experimental protocol was approved by the Committee for Research on Social Sciences and Humanities Sub jects of Oran (D00L01UN310120200001).

Author Contributions: All authors participated actively in this study. Their involvement covered the design of the research, data collection, analysis, and interpretation of results, as well as drafting and critically revising the manuscript. Each author reviewed the final version and approved it for submission. They collectively accept responsibility for the integrity of the work and agree to address any concerns regarding its accuracy or completeness.

References

1. McElhany K, Aggarwal S, Wood G, Beauchamp J. Protective and harmful social and psychological factors associated with mood and anxiety disorders in perimenopausal women: A narrative review. Maturitas. 2024;190:108118. doi:10.1016/j.maturitas.2024.108118
2. Choi MS, Shin H. Reliability and validity of the menopausal symptom scale. Women Health. 2015;55(1):58–76. doi:10.1080/03630242.2014.972017
3. Senouci A, Benhabib W, Fizi R, Mekki K. Impact of promoting a healthy lifestyle on anthropometric characteristics, climacteric symptoms, sleep quality and quality of life in women during menopausal transition. Curr Womens Health Rev. 2024;20(5):88–97. doi:10.2174/1573404820666230706105736
4. Thurston RC, Aslanidou Vlachos HE, Derby CA,, et al. Menopausal vasomotor symptoms and risk of incident cardiovascular disease events in SWAN. J Am Heart Assoc. 2021;10(3):e017416. doi:10.1161/JAHA.120.017416
5. Mandrup CM, Egelund J, Nyberg M, et al. Effects of high-intensity training on cardiovascular risk factors in premenopausal and postmenopausal women. Am J Obstet Gynecol. 2017;216(4):384.e1–384.e11. doi:10.1016/j.ajog.2016.12.017
6. Muka T, Nano J, Jaspers L, et al. Associations of steroid sex hormones and sex hormone-binding globulin with the risk of type 2 diabetes in women: A population-based cohort study and meta-analysis. Diabetes. 2017;66(3):577–86. doi:10.2337/db16-0473
7. Coll-Risco I, Borges-Cosic M, Acosta-Manzano P, et al. Effects of concurrent exercise on cardiometabolic status during perimenopause: The FLAMENCO Project. Climacteric. 2018;21(6):559–65. doi:10.1080/13697137.2018.1526892
8. Jackson EA, El Khoudary SR, Crawford SL, et al. Hot flash frequency and blood pressure: Data from the Study of Women's Health Across the Nation. J Womens Health (Larchmt). 2016;25(12):1204–09. doi:10.1089/jwh.2015.5670
9. Gray KE, Katon JG, LeBlanc ES, et al. Vasomotor symptom characteristics: Are they risk factors for incident diabetes? Menopause. 2018;25(5):520–30. doi:10.1097/GME.0000000000001033
10. Huang WY, Hsin IL, Chen DR, et al. Circulating interleukin-8and tumor necrosis factor-α are associated with hot flashes in healthy postmenopausal women. PLoS One. 2017;12(8):e0184011. doi:10.1371/journal.pone.0184011
11. Taleb-Belkadi O, Chaib H, Zemour L, et al. Lipid profile, inflammation, and oxidative status in peri- and postmenopausal women. Gynecol Endocrinol. 2016;32(12):982–85. doi:10.1080/09513590.2016.1214257
12. Vincent J, Inassi J. Comparison of oxidative stress between premenopausal and postmenopausal women. Natl J Physiol Pharm Pharmacol. 2020;10(5):359–62. doi:10.5455/njppp.2020.10.02051202002032020
13. Pham-Huy LA, He H, Pham-Huy C. Free radicals, antioxidants in disease and health. Int J Biomed Sci. 2008;4(2):89–96.
14. Oh S, Kim E, Shoda J. Editorial: Lifestyle modification strategies as first line of chronic disease management. Front Physiol. 2023;14:1204581. doi:10.3389/fphys.2023.1204581
15. Franquesa M, Pujol-Busquets G, García-Fernández E, et al. Mediterranean diet and cardiodiabesity: A systematic review through evidence-based answers to key clinical questions. Nutrients. 2019;11(3):655. doi:10.3390/nu11030655
16. Widmer RJ, Flammer AJ, Lerman LO, Lerman A. The Mediterranean diet, its components, and cardiovascular disease. Am J Med. 2015;128(3):229–38. doi:10.1016/j.amjmed.2014.10.014
17. Hidalgo-Mora JJ, García-Vigara A, Sánchez-Sánchez ML, et al. The Mediterranean diet: A historical perspective on food for health. Maturitas. 2020;132:65–69. doi:10.1016/j.maturitas.2019.12.002
18. Dinu M, Pagliai G, Casini A, Sofi F. Mediterranean diet and multiple health outcomes: An umbrella review of meta-analyses of observational studies and randomised trials. Eur J Clin Nutr. 2018;72(1):30–43. doi:10.1038/ejcn.2017.58
19. Heinemann LA, Potthoff P, Schneider HP. International versions of the Menopause Rating Scale (MRS). Health Qual Life Outcomes. 2003;1:28. doi:10.1186/1477-7525-1-28
20. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499–502.
21. Kahn HS. The lipid accumulation product performs better than the body mass index for recognizing cardiovascular risk: A population-based comparison. BMC

CardiovascDisord.2005;5:26.doi:10.1186/1471-2261-5-26. [INCOMPLETE PMCID]
22. Levine RL, Garland D, Oliver CN, et al. Determination of carbonyl content in oxidativelymodified proteins. Methods Enzymol. 1990;186:464–78. doi:10.1016/0076-6879(90)86141-h
23. Marklund S, Marklund G. Involvement of the superoxide anion radical in the autoxidation of pyrogallol and a convenient assay for superoxide dismutase. Eur J Biochem. 1974;47(3):469–74. doi:10.1111/j.1432-1033.1974.tb03714.x
24. Góth L. A simple method for determination of serum catalase activity and revision of reference range. Clin Chim Acta. 1991;196(2–3):143–51. doi:10.1016/0009-8981(91)90067-m
25. Sedlak J, Lindsay RH. Estimation of total, protein-bound, and nonprotein sulfhydryl groups in tissue with Ellman's reagent. Anal Biochem. 1968;25(1):192–205. doi:10.1016/0003-2697(68)90092-4
26. Au A, Feher A, McPhee L, et al. Estrogens, inflammation and cognition. Front Neuroendocrinol. 2016;40:87–100. doi:10.1016/j.yfrne.2016.01.002
27. Marchand GB, Carreau AM, Weisnagel SJ, et al. Increased body fat mass explains the positive association between circulating estradiol and insulin resistance in postmenopausal women. Am J Physiol Endocrinol Metab. 2018;314(5):E448–E456. doi:10.1152/ajpendo.00293.2017
28. Silva TR, Oppermann K, Reis FM, Spritzer PM. Nutrition in menopausal women: A narrative review. Nutrients. 2021;13(7):2149. doi:10.3390/nu13072149
29. Kastorini CM, Milionis HJ, Esposito K, et al. The effect of Mediterranean diet on metabolic syndrome and its components: A meta-analysis of 50 studies and 534,906 individuals. J Am Coll Cardiol. 2011;57(11):1299–313. doi:10.1016/j.jacc.2010.09.073
30. Amrita J, Mahajan M, Bhanwer AJ, et al. Oxidative stress: An effective prognostic tool for an early detection of cardiovascular disease in menopausal women. Biochem Res Int. 2016;2016:6157605. doi:10.1155/2016/6157605
31. McCarthy M, Raval AP. The peri-menopause in a woman's life: A systemic inflammatory phase that enables later neurodegenerative disease. J Neuroinflammation. 2020;17(1):317. doi:10.1186/s12974-020-01998-9
32. Yoshikata R, Myint KZ, Ohta H, Ishigaki Y. Inter-relationship between diet, lifestyle habits, gut microflora, and the equol-producer phenotype: Baseline findings from a placebo-controlled intervention trial. Menopause. 2019;26(3):273–85. doi:10.1097/GME.0000000000001202
33. Thom G, Lean M. Is there an optimal diet for weight management and metabolic health? Gastroenterology. 2017;152(7):1739–51. doi:10.1053/j.gastro.2017.01.056
34. Agacayak E, Basaranoglu S, Tunc SY, et al. Oxidant/antioxidant status, paraoxonase activity, and lipid profile in plasma of ovariectomized rats under the influence of estrogen, estrogen combined with progesterone, and genistein. Drug Des Devel Ther. 2015;9:2975–82. doi:10.2147/DDDT.S82263
35. Pugliese G, Barrea L, Laudisio D, et al. Mediterranean diet as tool to manage obesity in menopause: A narrative review. Nutrition. 2020;79–80:110991. doi:10.1016/j.nut.2020.110991

Correspondence:
Received: 19 September 2025
Accepted: 25 November 25
Dr. Azzeddine Senouci,
Department of Agri-Food, Faculty of Science and Technology, University of Ain Temouchent- Belhadj Bouchaib, Ain Temouchent 46000, Algeria. Thematic Agency of Research in Health Sciences. Oran, Algeria
E-mail : azzeddine.senouci@univ-temouchent.edu.dz