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Effects of a 26 week multicomponent exercise program on cardiovascular and lipid profiles in premenopausal and postmenopausal women

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The present study aimed to examine the effects of a 26-week multicomponent exercise program on lipid and lipoprotein profiles, blood pressure, and anthropometric parameters in healthy, previously sedentary premenopausal and postmenopausal women aged 40–60, as determined by the Baecke physical activity questionnaire. The program, comprising resistance, balance, flexibility, and aerobic exercises performed at 60–80% of maximal heart rate, aimed to identify group-specific responses and establish the program's efficacy in mitigating cardiovascular risks. Seventy-five women were recruited, and 38 completed the intervention. Participants were categorized into premenopausal (n = 18), postmenopausal (n = 10), and control groups (n = 10). Key measures included total cholesterol, low- and high-density lipoprotein cholesterol, triglycerides, glucose, systolic and diastolic blood pressure, and anthropometric variables. Statistical analyses employed the Shapiro-Wilk test, paired t-tests, Wilcoxon tests, and Kruskal-Wallis tests, with α = 0.05. Significant improvements were observed in both groups. Premenopausal women showed superior lipid and glucose reductions ($p < 0.01$, d = 0.79), while postmenopausal women showed greater blood pressure decreases ($p < 0.01$, d > 1.40). Anthropometric changes were significant but varied in magnitude. Premenopausal women improved lipid and glucose profiles; postmenopausal women showed greater reductions in blood pressure and waist circumference. Multicomponent training with aerobic, resistance, and flexibility elements should be adapted to each group's clinical targets.

Keywords Physical fitness, Anthropometry, Health, Blood pressure, Physical activity

Cardiovascular diseases remain the leading cause of death worldwide and represent a substantial burden on public health systems, particularly among aging populations[1]. In women, the transition through menopause marks a critical point in cardiovascular risk stratification, driven largely by the progressive decline in circulating estrogen levels. This hormonal reduction is known to disrupt vascular endothelial function, impair lipid metabolism, increase visceral adiposity, and contribute to elevated blood pressure, all of which are closely linked to atherogenesis and insulin resistance[2–4].

As life expectancy increases globally, a growing proportion of women now spend over one-third of their lives in the postmenopausal phase. During this period, there is a well-documented deterioration in cardiometabolic health, including rises in systolic and diastolic blood pressure, total and LDL cholesterol, and central adiposity, particularly visceral fat accumulation[4–6]. In contrast, premenopausal women—despite showing fewer overt risk factors—may still exhibit subclinical impairments that warrant preventive action, especially among sedentary individuals.

Physical activity is widely recognized as an effective non-pharmacological intervention to mitigate cardiovascular risk in both men and women[7–9]. Aerobic and resistance exercise modalities have been shown to reduce LDL-C and triglycerides, increase HDL-C, improve glycaemic control, and reduce resting blood

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pressure[10–12]. Nevertheless, most available data stem from heterogeneous samples, with few controlled studies designed to directly compare the cardiometabolic responses to exercise in pre- versus postmenopausal women. This is particularly relevant because hormonal status may influence adaptive physiological responses to training stimuli[4,13,14].

Moreover, although many interventions focus exclusively on aerobic or resistance components, multicomponent training—which incorporates aerobic, resistance, flexibility, and balance exercises—has emerged as a comprehensive and functional approach to promoting health in midlife and older populations[13,15,16]. This model addresses multiple physiological systems simultaneously and has demonstrated efficacy in improving not only lipid and blood pressure profiles but also neuromuscular function, balance, and mobility[15–18]. Despite this, its application in comparative designs between different female life stages remains scarce, and there is limited understanding of how menopausal status may modulate training responsiveness.

An additional shortcoming in the literature is the lack of integrated assessments. Many studies isolate single outcomes (e.g., blood pressure or body composition), rather than examining a broader profile of interdependent cardiometabolic variables. Furthermore, methodological limitations such as short intervention durations, lack of structured periodization, or poorly controlled exercise intensity limit the generalizability and practical application of findings[11,13,16].

Recent work suggests that premenopausal women may respond more favourably to exercise-induced lipid and glucose metabolism improvements[4,14], while postmenopausal women may experience greater changes in hemodynamic and anthropometric measures, including waist circumference and BMI[6,11]. These divergent adaptations highlight the need for tailored interventions that account for hormonal, metabolic, and vascular differences. However, few studies have implemented and monitored such targeted protocols in a real-world training environment.

Given these knowledge gaps, the present study aimed to evaluate the differential effects of a 26-week periodized multicomponent exercise program—consisting of aerobic, resistance, flexibility, and balance training—on lipid-lipoprotein profiles, blood pressure, and anthropometric variables in sedentary premenopausal and postmenopausal women aged 40–60 years. This research seeks to clarify whether menopausal status influences physiological responsiveness to exercise and to contribute to the development of age- and hormone-sensitive training guidelines aimed at mitigating cardiovascular risk in middle-aged women.

Materials and methods

Study design

This study was a non-randomized, controlled, quasi-experimental trial with three groups (premenopausal, postmenopausal, and control) evaluated at baseline and post-intervention. It followed the CONSORT guidelines for non-randomized trials where applicable.

Participants

Seventy-five (n = 75) women volunteered to participate in the study and were selected using non-probability convenience sampling. Participants voluntarily enrolled in a university-based health and physical activity program. The present study was designed as a public health intervention involving targeted community engagement, aiming to assess the effects of a structured exercise program in a real-world setting. In brief, the inclusion criteria were; (a) women aged between 40 and 60 years old; (b) healthy, asymptomatic, and non-smoking; (c) medical clearance for participation in physical activity; (d) absence of clinical history related to alcoholism or other uncontrolled metabolic disease; (e) no clinical prescription for hypotensive pharmacological intake, cholesterol reduction, hormonal replacement therapy, or any other intervention capable of inducing alterations in lipid and lipoprotein metabolism; or (f) abnormal blood sample results. The exclusion criteria encompassed the following: (a) non-fulfilment of inclusion criteria; (b) clinical history characterized by symptoms related to cardiovascular disease (CVD), including stroke (CVA) and coronary vascular disease (CVD), renal or hepatic disease, musculoskeletal disorders, or rheumatoid conditions that may impede full participation in the exercise program. Habitual physical activity assessment was carried out using the questionnaire/interview (direct methodology) by Baecke et al.[19], adapted and validated for the Portuguese population[20,21]. This questionnaire aimed to gather information about routine activities in the past year in the domains of household, sports, and leisure activities, allowing to determine those women who were sedentary.

This study was approved by the Research Ethics Committee of the University of Évora (reference: 22137). All methods were performed in accordance with relevant guidelines and regulations, including the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants and were instructed to maintain their regular daily routines and dietary habits. Seventy-five women were initially recruited, and 38 completed the intervention. Only participants who attended 100% of the scheduled sessions over the 26-week period were included in the final analysis to ensure that all individuals received the full training stimulus. The reasons for non-completion included scheduling conflicts due to work or family commitments (n = 19), voluntary withdrawal without specifying reasons (n = 11), minor injuries or health issues unrelated to the intervention (n = 4), and failure to meet the adherence requirement of 100% attendance (n = 3). All withdrawals occurred before the post-intervention assessments. These losses were not associated with any adverse effects of the program and were consistent with real-world challenges in long-term lifestyle interventions. Attendance was systematically recorded at each session, and any participant missing a session was required to complete an equivalent session within the same week. Those who failed to meet this adherence criterion were excluded from the final dataset. Upon completion of the study, a total of 38 women successfully finished the program, distributed across the following groups: pre-menopause (PRE) (n = 18), post-menopause (POST) (n = 10), and control group (CG) (n = 10). The control group was composed of women who met the same inclusion and exclusion criteria as the experimental groups and were similarly classified as sedentary based on the Baecke

physical activity questionnaire. While formal matching procedures were not applied, there were no significant baseline differences in age, physical activity level, or cardiovascular risk factors between the groups. Control group participants were instructed to maintain their habitual daily routines and not to initiate any new structured exercise programs during the 26-week period. Their participation served as a comparative reference to isolate the effects of the multicomponent intervention. Menopausal status was determined through clinical assessment by certified medical professionals within the National Health System. The classification was based on medical records, clinical examinations, and, where applicable, hormonal assessments (e.g., follicle-stimulating hormone and estradiol levels) to ensure accurate identification of reproductive status.

A formal a priori power analysis was not conducted; however, the final sample size was based on feasibility constraints and is consistent with previous intervention studies involving premenopausal and postmenopausal women, which have utilized similar sample sizes[22].

A non-probabilistic convenience sampling method was employed due to the practical constraints of recruiting sedentary premenopausal and postmenopausal women in a real-world public health setting. This approach aligns with similar exercise-based interventions where voluntary participation and community-based implementation limit the use of probabilistic sampling strategies. While this method enhances ecological validity, it may restrict generalizability and introduce selection bias. Participants were stratified by menopausal status (pre/post) upon enrolment.

To ensure full compliance with the inclusion criteria, participants underwent a preliminary health screening conducted by medical professionals affiliated with the National Health System. This included review of clinical history, recent medical diagnostics, and where necessary, complementary tests to confirm asymptomatic status. Additionally, the Baecke physical activity questionnaire, adapted and validated for the Portuguese population, was used to confirm sedentary behaviour at baseline.

Intervention

The physical exercise program, lasting for 26 weeks, was conducted with an uninterrupted weekly frequency of 3 sessions, on non-consecutive days, in the late afternoon (at 7:30 PM), each lasting 60 min.

The exercise program followed a structured periodization model consisting of six progressive training phases over a total duration of 26 weeks. Each phase was designed to gradually increase physiological demands while incorporating strategic recovery and adaptation assessments. The first phase (Adaptation, 3 weeks) introduced participants to the program with low-to-moderate intensity exercises, ensuring initial neuromuscular adjustments and familiarization with movement patterns. The second phase (Load/Development, 5 weeks) aimed to increase intensity slightly to stimulate improvements in general physical fitness. The third phase (Recovery, 2 weeks) served as a planned reduction in training intensity, allowing for the first systematic assessment of physiological responses to the intervention while preparing participants for further load progression. The fourth phase (High-Load, 6 weeks) introduced increased training intensity and volume, with a focus on cardiovascular and musculoskeletal adaptations, consolidating prior fitness improvements. The fifth phase (Recovery, 2 weeks) mirrored the third phase, reinforcing physiological adaptation and fatigue management. Finally, the sixth phase (Final Load Progression, 8 weeks) was subdivided into two sub-phases: Load/Development (5 weeks): Focused on achieving higher adaptation levels than previous load phases, with gradual workload reductions in the final week to assess participants' physiological responses; and Load/Stabilization (3 weeks): Training volume was adjusted based on the individualized responses observed in the previous phase. If positive adaptations were confirmed, volume was slightly increased while maintaining intensity; otherwise, the phase served to stabilize previous adaptations and optimize retention of training effects. Each training block lasted approximately 1 to 1.5 months, consisting of 12 to 18 effective training sessions, ensuring a progressive biological adaptation process aligned with the achievement of increasingly demanding training goals.

The sessions were organized into three main parts: warm-up, main part, and final part. The warm-up lasted approximately 15 min and included a series of low-impact cardiovascular mobilization activities (scoring 12 to 13 points on the Borg Rating of Perceived Exertion Scale)[23], such as walking or dancing. Additionally, this phase incorporated calisthenic exercises, joint-ligament mobilization, and stretching, along with global coordination and static and dynamic balance tasks of increasing complexity. This phase aimed to prepare participants for the demands of the main part of the session, ensuring optimal readiness and reducing injury risk.

The main part of the session was designed to address the specific objectives of each training phase. Activities varied based on the weekly plan, ensuring diversity while following a structured progression. Aerobic Metabolism & Cardiovascular Fitness: The first and last sessions of each week focused on cardiovascular development. These included consistent activities, such as water aerobics in the last session, while the first session integrated transient activities aligned with the program phase (e.g., dance, walking, body combat, and step exercises). Musculoskeletal Fitness: The second weekly session targeted muscular strength and endurance through a functional training circuit. This circuit incorporated a variable number of exercises, intensity, and volume, progressively increasing throughout the program. The exercises engaged major muscle groups and included free weights, ankle weights, toning balls, Swiss balls, medicine balls, elastic bands, steps, and body weight exercises. To ensure progressive adaptation, participants completed a structured preparation phase before the main training workload. This included: five minutes of walking or dancing; and five minutes of stretching. One repetition of the functional circuit with a substantial reduction in resistance to ensure technical proficiency before the main effort. Exercise duration and rest periods were strictly controlled across all training sessions. The internal training load was monitored through the group's average subjective perceived exertion, assessed using Borg's Rating of Perceived Exertion Scale[23]. Additionally, heart rate responses were measured in a random subgroup of participants to validate exertion levels and confirm consistency between perceived effort and physiological response. During aerobic and cardiovascular-focused sessions, exercises were organized into 10-minute high-intensity blocks (scoring 15 to 16 on the Borg scale), interspersed with 3 to 5 min of low-intensity recovery (scoring 10 to 11 on

the Borg scale). Heart rate data were collected using Polar H10 HR sensors (Polar Electro, Kempele, Finland), recorded at the end of the main training phase and during recovery blocks, to compare perceived exertion with physiological markers.

The final part of each session consisted of respiratory and stretching exercises aimed at enhancing flexibility and promoting recovery. Participants performed static stretching: holding each position for 15 to 30 s; Dynamic stretching: repeating each movement 3 to 4 times. This phase was crucial for optimizing muscle relaxation and post-exercise recovery.

In the weekly session dedicated to functional training, control over exercise amplitudes was guided by each subject's subjective perception. The instruction given was that participants should, whenever possible, adhere to the technical recommendations for each exercise but always within the bounds of personal comfort. To minimize potential health risks and ensure participant safety during exercise, all training sessions were conducted under the supervision of certified exercise physiologists with expertise in clinical populations. Medical support was available on-site or on-call through the university's affiliated health center, and participants were monitored throughout for signs of adverse events. Pre-session health checks and real-time monitoring (e.g., perceived exertion and heart rate) were implemented as part of the risk mitigation protocol. Selected variables from the lipid and lipoprotein profile were used to differentiate the effect of exercise intervention between pre-menopausal and post-menopausal women (Table 1).

Outcome measures

Data sample blood

The blood collections (at the beginning and end of the exercise program) were performed at a certified clinic, with the subject seated, and blood was drawn from the anterior cubital vein in the morning, after an overnight fast[24]. Conventional biochemical laboratory analysis methods were employed for the assessment of the plasma parameters under study.

Friedwald equation

The determination of atherogenic coefficients was carried out using the Friedewald equations[25].

$$\mathrm{LDL-C} = \mathrm{CT} - [\mathrm{HDL-C} + 1/5\mathrm{TG}] \quad (1)$$

$$\mathrm{VLDL} = \mathrm{TG}/5 \quad (2)$$

Anthropometric measures

Participants arrived at the research facility after abstaining from exercise, alcohol, or stimulant beverages and having fasted for a minimum of 3 h. Body weight, measured without shoes and in minimal clothing, was recorded with a high-precision digital scale (SECA 861*, SECA, Hamburg, Germany) accurate to 0.01 kg. Height was measured using a stadiometer (SECA 225*, SECA, Hamburg, Germany) with a precision of 0.1 cm, following standardized anthropometric protocols. BMI was calculated as weight (kg) divided by height squared (m^2) [BMI = kg/m^2], a widely accepted index for assessing body composition. Hip and waist circumferences were measured at the maximum diameter over the buttocks and at the midpoint between the lowest rib and iliac crest at the end of a normal expiration, respectively. Both measurements were conducted using a non-elastic measuring tape with a precision of 0.1 cm (SECA 212*, SECA, Hamburg, Germany) while participants maintained an anatomical standing position.

Variable	Abbreviation	Unit
Total cholesterol	TC	mg/dl
Low-density lipoprotein cholesterol	LDL-C	mg/dl
High-density lipoprotein cholesterol	HDL-C	mg/dl
Triglycerides	TG	mg/dl
Castelli risk index I	TC/HDL-C	a.u.
Castelli risk index II	LDL-C/HDL-C	a.u.
Very low-density lipoprotein cholesterol	VLDL-C	mg/dl
Glucose	G	mg/dl
Systolic blood pressure	SBP	mmHg
Diastolic blood pressure	DBP	mmHg
Heart rate	HR	bpm
Body mass index	BMI	kg/m^2
Waist circumference	WC	cm
Hip circumference	HP	cm
Waist circumference/ Hip circumference	WC/HP	a.u.

Table 1. Selected lipid and lipoprotein profile variables. mg = milligrams; dl = decilitre; a.u.=arbitrary units; mmHg = millimetres of mercury; bpm = Beats per minute; kg = Kilogram; m = meter; cm = centimetres.

Blood pressure

Blood pressure was assessed following a 10 to 20-minute period of rest using a previously validated automatic device (Omron M6 Comfort, model HEM-7360-E, Omron Healthcare Co., Kyoto, Japan), adhering to established guidelines[26]. Three consecutive measurements were taken at intervals of 1 to 2 min in the arm that initially exhibits the highest reading. The result is the average of these two measurements.

Daily physical activity

The determination of habitual daily physical activity habits was carried out through the use of the questionnaire/interview, adapted and validated for the Portuguese population[27]. This questionnaire aimed to gather information about routine activities in the past year in the domains of household, sports, and leisure activities.

Randomization

Due to logistical constraints and the nature of the community-based intervention, participants were allocated to groups based on menopausal status and availability, without randomization.

Statistical analysis

For descriptive analysis, variables were expressed as mean ± standard deviation or median [IQR] depending on the normality of distribution, as determined by the Shapiro-Wilk test. For parametric comparisons (e.g., paired t-tests), the assumption of homogeneity of variances was assessed using Levene's test. No significant violations were observed under parametric conditions, thus supporting the appropriateness of t-test applications. In cases where this assumption was not met or when distributions deviated from normality, non-parametric alternatives (Wilcoxon or Kruskal-Wallis) were applied. Intragroup comparisons employed paired t-tests or Wilcoxon signed-rank tests, depending on normality. Intergroup differences were assessed using Kruskal-Wallis tests, followed by Dunn's post hoc tests with Bonferroni correction for multiple comparisons. Effect sizes were calculated for all comparisons to complement p-values. For parametric tests (e.g., paired t-tests), Cohen's d was used. For nonparametric tests (e.g., Wilcoxon), effect size r was computed using the formula $r = Z/\sqrt{n}$. In Table 3, these are all presented under the column heading 'Cohen's d/r' for consistency. The Tukey post-hoc statistical test was employed to determine which variables exhibited differences among the groups. Statistical significance was set at $\alpha = 0.05$. Analyses were performed using SPSS 24.0 (IBM Corp., Armonk, NY, USA).

Results

A total of 38 women aged 40 to 60 years completed the 26-week exercise intervention, having met the inclusion criterion of 100% adherence to scheduled sessions. Participants were stratified into three groups: premenopausal ($n = 18$), postmenopausal ($n = 10$), and control ($n = 10$). All individuals were classified as sedentary at baseline based on the Baecke physical activity questionnaire, and no significant comorbidities were present. Baseline clinical evaluations indicated that only the premenopausal group exhibited total cholesterol and LDL-C values below recommended thresholds (TC < 190 mg/dL; LDL-C < 130 mg/dL), while the postmenopausal and control groups presented borderline or elevated values. All groups had HDL-C levels above 45 mg/dL and fell within the cardiovascular risk limits for Castelli indices I and II. No baseline differences in BMI or anthropometric parameters were observed between the groups. Menopausal status was confirmed by certified clinicians using clinical criteria and, where applicable, biochemical markers such as FSH and estradiol. These baseline data confirm the clinical comparability of the groups prior to intervention.

To ensure accurate interpretation of non-parametric outcomes, variables analysed using Wilcoxon or Kruskal-Wallis tests are presented as medians and interquartile ranges [IQR] in Table 2, while parametric outcomes are shown as means ± standard deviations. Regarding total cholesterol levels, all groups except the pre-menopausal women's group exhibited values exceeding the recommended threshold (TC < 190 mg/dL). Both the premenopausal women's group and the control group presented values within the defined cut-off range (LDL-C < 130 mg/dL), whereas the postmenopausal group exhibited values exceeding this threshold. In relation to the LDL-C/HDL-C ratio, which clearly establishes cardiovascular risk through the atherogenic index, all groups fall within the confidence interval (LDL-C/HDL-C < 2.5) (Table 2).

In pre-test, statistically significant differences were found among the analysed groups, specifically in the variable glucose ($\chi2 = 17.737$; $p < 0.000$). More specifically, significant differences were observed between the post-menopausal group and the control group (z=-13.30; $p = 0.000$) and between the pre-menopausal group and the control group (z=-11.14; $p < 0.000$). In post-test, statistically significant differences were also found among the analysed groups, particularly in the variable's glucose ($\chi2 = 11.56$; $p = 0.03$; $\varepsilon^2=0.273$), systolic blood pressure ($\chi2 = 12.18$; $p = 0.02$; $\varepsilon^2=0.291$), and diastolic blood pressure ($\chi2 = 7.69$; $p = 0.021$; $\varepsilon^2=0.163$). Regarding glucose, differences were found between the pre-menopausal group and the control group (z=-17.077; $p = 0.01$). In relation to systolic blood pressure, differences were observed between the pre-menopausal group and the control group (z=-22.266; $p < 0.000$). As for diastolic blood pressure, differences were noted between the post-menopausal group and the control group (z=-12.700; $p = 0.044$) and between the pre-menopausal group and the control group (z = 15.922; $p = 0.003$) (Table 3).

Concerning the post-menopausal women's group, statistically significant differences were found in the variables: systolic blood pressure (t = 3.736; $p = 0.005$), diastolic blood pressure (t = 3.914; $p = 0.004$), body mass index (BMI) (t = 4.610; $p = 0.001$), waist circumference (t = 3.709; $p = 0.005$), and hip circumference (t = 6.278; $p = 0.000$). In the pre-menopausal women's group, statistically significant differences were observed in the variables: glucose (t = 3.266; $p = 0.005$), systolic blood pressure (z=-3.311; $p = 0.001$), diastolic blood pressure (t = 4.213; $p = 0.005$), BMI (t = 3.131; $p = 0.006$), and hip circumference (t = 4.734; $p = 0.000$). Regarding the control group, only one variable exhibited statistically significant differences, specifically hip circumference (t=-2.355, $p = 0.043$) (Table 3).

	Post-menopausal		Pre-menopausal		Control	
	Pre-test	Post-test	Pre-test	Post-test	Pre-test	Post-test
TC (mg/dl)	201.20 ± 29.02	215.40 ± 51.66	187.44 ± 47.33	184.61 ± 24.42	201.20 ± 36.07	206.60 ± 33.93
LDL-C (mg/dl)	131.50 ± 27.62	133.20 ± 41.69	115.28 ± 20.08	111.18 ± 21.97	120.30 ± 36.94	130.00 ± 38.41
HDL-C (mg/dl)	-	55.60 ± 8.25	57.39 ± 10.13	56.06 ± 10.71	58.20 ± 12.00	56.70 ± 12.97
HDL-C* (mg/dl)	52.58 [5.51]	56.86[12.58]	-	-	-	-
TG* (mg/dl)	83.20 [33.36]	91.51 [53.97]	70.44 [29.24]	81.95 [51.63]	107.77[86.53]	107.82[89.11]
CRI-I (a.u.)	3.82 ± 0.53	3.93 ± 1.06	-	-	3.63 ± 1.08	3.83 ± 1.10
CRI-I (a.u.)*	-	-	3.32 [0.79]	3.32 [1.11]	-	-
CRI-II (a.u.)	2.50 ± 0.51	2.44 ± 0.82	-	-	2.18 ± 0.85	2.42 ± 0.90
CRI-II (a.u.)*	-	-	2.14 [0.78]	2.17 [1.05]	-	-
VLDL-C (mg/dl)*	15.59 [3.31]	17.12 [12.27]	13.73 [5.77]	15.79 [10.87]	18.61 [26.27]	25.53 [10.79]
G (mg/dl)	76.40 ± 7.37	72.80 ± 8.80	78.56 ± 6.47	70.72 ± 12.43	89.70 ± 3.37	87.80 ± 8.27
SBP (mmHg)	155.90 ± 24.90	128.40 ± 11.93	-	-	142.30 ± 15.70	142.10 ± 18.10
SBP (mmHg)*	-	-	131.55[21.17]	120.70[14.13]	-	-
DBP (mmHg)	95.50 ± 11.58	82.00 ± 5.48	87.94 ± 8.53	78.78 ± 9.32	92.60 ± 13.72	94.70 ± 17.52
HR (bpm)	72.60 ± 12.87	74.30 ± 9.65	77.78 ± 8.63	79.44 ± 8.42	73.10 ± 10.72	75.90 ± 9.37
BMI (kg/m^2)	25.82 ± 3.26	25.68 ± 3.05	25.82 ± 3.81	25.69 ± 3.74	28.43 ± 6.48	28.30 ± 6.56
WC (cm)	90.50 ± 6.58	88.25 ± 6.50	90.75 ± 9.27	89.47 ± 10.27	93.50 ± 15.39	93.50 ± 15.10
HP (cm)	101.05 ± 5.88	97.65 ± 5.79	105.25 ± 7.68	102.94 ± 8.41	106.95 ± 15.62	107.70 ± 15.85
WC/HP (a.u.)	0.90 ± 0.04	0.90 ± 0.05	-	-	0.87 ± 0.05	0.87 ± 0.04
WC/HP (a.u.)*			0.84[0.09]	0.86[0.10]		

Table 2. Descriptive and comparative analysis of selected variables presented as means ± standard deviations for parametric data and medians [interquartile range] for non-parametric data. TC = Total Cholesterol; LDL-C = Low-density lipoprotein Cholesterol; HDL-C = High-density lipoprotein Cholesterol; TG = Triglycerides; CRI = Castelli Risk Index; VLDL-C = Very Low-Density Lipoprotein Cholesterol; G = Glucose; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; HR = Heart Rate; BMI = Body Mass Index; WC = Waist Circumference; HP = Hip Circumference; mg = milligrams; dl = decilitre; a.u.=arbitrary units; mmHg = millimetres of mercury; bpm = Beats per minute; kg = Kilogram; m = meter; cm = centimetres; *=Variables analysed using non-parametric tests and are presented as median [interquartile ranges].

Table 4 presents the Spearman correlation coefficients between changes in anthropometric and cardiovascular variables. The strength of correlations was interpreted according to standard thresholds for Spearman's rho: weak ($\rho < 0.30$), moderate ($\rho = 0.30–0.49$), and strong ($\rho \geq 0.50$) associations. In the premenopausal group, a significant positive correlation was observed between ΔDBP and ΔBMI ($r = 0.552$, $p < 0.05$), as well as between ΔWC and ΔCRI-I ($r = 0.471$, $p < 0.05$), and between ΔWC and ΔSBP ($r = 0.555$, $p < 0.05$). In the postmenopausal group, a positive correlation was identified between ΔDBP and ΔHP ($r = 0.543$), although it did not reach statistical significance. When analysing the combined group (PRE + POST), significant correlations included a negative association between ΔWC/HP and ΔHDL-C ($r = -0.472$, $p < 0.05$), a positive correlation between ΔDBP and ΔBMI ($r = 0.482$, $p < 0.01$), and between ΔDBP and ΔSBP ($r = 0.533$, $p < 0.01$).

Discussion

The present study investigated the differential effects of a 26-week periodized multicomponent exercise program—comprising aerobic, resistance, flexibility, and balance components—on lipid-lipoprotein profiles, blood pressure, and anthropometric markers in sedentary premenopausal and postmenopausal women aged 40 to 60 years. The findings indicate that premenopausal participants exhibited a more favorable physiological response to the intervention, with marked improvements across multiple lipid and lipoprotein parameters. Although postmenopausal women also experienced beneficial changes, these adaptations were comparatively modest, suggesting that hormonal status may modulate the magnitude of cardiometabolic responsiveness to exercise in this population.

Magnitude and direction of the lipid and lipoprotein response

The lipid and lipoprotein responses to the 26-week multicomponent exercise intervention revealed differential adaptations between premenopausal and postmenopausal women. While both groups benefitted from the program, the premenopausal participants demonstrated greater improvements in markers associated with lipid metabolism, whereas postmenopausal women showed more modest or inconsistent responses.

TC declined slightly in the premenopausal group but increased in postmenopausal participants, likely influenced by elevated TG and HDL-C levels. Although these trends did not reach statistical significance, the reduction in TC among premenopausal women is physiologically relevant and consistent with aerobic exercise-induced lipid modulation reported in prior studies[28,29]. Conversely, the increase observed in postmenopausal

	Post-menopausal			Pre-menopausal			Control		
	t/z	p	Cohen's d/r	t/z	p	Cohen's d/r	t/z	p	Cohen's d/r
TC (mg/dl)	-0.860[t]	0.412	0.3389[d]	0.516[t]	0.613	0.075[d]	-1.197[t]	0.262	0.154[d]
LDL-C (mg/dl)	-0.127[t]	0.902	0.048[d]	-0.054[t]	0.958	0.195[d]	-1.680[t]	0.127	0.257[d]
HDL-C (mg/dl)	-1.379[z]	0.168	-0.436[r]	0.772[t]	0.451	0.128[r]	0.594[t]	0.567	-0.581[d]
TG (mg/dl)	-0.153[z]	0.878	-0.048[r]	-1.044[z]	0.296	-0.246[r]	-1.837[z]	0.066	-0.581[r]
CRI-I (a.u.)	-0.370[t]	0.720	0.131[d]	-0.414[z]	0.679	-0.098[r]	-1.351[t]	0.210	0.183[d]
CRI-II (a.u.)	0.226[t]	0.826	0.088[d]	-0.402[z]	0.687	-0.095[r]	-1.662[t]	0.131	0.274[d]
VLDL-C (mg/dl)	-0.153[z]	0.878	-0.048[r]	-1.044[z]	0.296	-0.246[r]	-1.837[z]	0.066	-0.581[r]
G[a,b] (mg/dl)	1.192[t]	0.264	0.444[d]	3.266[t]	0.005*	0.791[d]	1.075[t]	0.310	0.301[d]
SBP[b] (mmHg)	3.736[t]	0.005*	1.409[d]	-3.311[z]	0.001*	-0.78[r]	0.83[t]	0.936	0.012[d]
DBP[b] (mmHg)	3.914[t]	0.004*	1.490[d]	4.213[t]	0.005*	1.025[d]	-0.922[t]	0.381	0.133[d]
HR (bpm)	-0.355[t]	0.731	0.149[d]	-0.670[t]	0.512	0.195[d]	-1.993[t]	0.077	0.278[d]
BMI (kg/m^2)	4.610[t]	0.001*	0.044[d]	3.131[t]	0.006*	0.034[d]	-0.067[t]	0.948	0.020[d]
WC (cm)	3.709[t]	0.005*	0.338[d]	1.132[t]	0.273	0.131[d]	0.0[t]	1.00	0
HP (cm)	6.278[t]	0.000*	0.583[d]	4.734[t]	0.000*	0.287[d]	-2.355[t]	0.043*	0.048[d]
WC/HP (a.u.)	-1.360[t]	0.207	0	-0.833[z]	0.405	-0.196[r]	0.721[t]	0.489	0

Table 3. Analysis of variance for the set of parameters studied in the three groups (postmenopausal, premenopausal, and control group) at the two evaluation time points (pre and post-test). TC = Total Cholesterol; LDL-C = Low-density lipoprotein Cholesterol; HDL-C = High-density lipoprotein Cholesterol; TG = Triglycerides; CRI = Castelli Risk Index; VLDL-C = Very Low-Density Lipoprotein Cholesterol; G = Glucose; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; HR = Heart Rate; BMI = Body Mass Index; WC = Waist Circumference; HP = Hip Circumference; mg = milligrams; dl = decilitre; a.u.=arbitrary units; mmHg = millimetres of mercury; bpm = Beats per minute; kg = Kilogram; m = meter; cm = centimetres; t = Inferential analysis using repeated measures t-test; z = Inferential analysis based on the non-parametric Wilcoxon test; p = Between group-subject effect; *indicates significant changes; d = Cohen's d for parametric comparisons; r = effect size for Wilcoxon tests; a = Kruskal Wallis differences between groups in pre-test; b = Kruskal Wallis differences between groups in post-test.

women aligns with reports suggesting a blunted lipid response in this population, potentially due to hormonal alterations post-menopause[4,6,28].

LDL-C values decreased marginally in the premenopausal group, which, despite lacking statistical significance, may have clinical relevance given that even a 1% reduction in LDL-C is associated with a 2–3% decrease in coronary heart disease risk[14]. These findings contrast with literature indicating minimal responsiveness of LDL-C to exercise in isolation[6], suggesting that a combined modality approach—as applied here—may yield cumulative benefits, particularly in estrogen-replete women.

HDL-C improvements were evident only in the postmenopausal group. This improvement is consistent with literature reporting HDL-C as the most exercise-sensitive lipid marker, particularly in response to interventions of sufficient duration[12,30]. This is particularly notable given the intervention's inclusion of moderate-to-vigorous aerobic work and strength training at 60–80% HRmax—both known to enhance HDL-C concentrations via increased lipoprotein lipase activity and HDL particle maturation[31]. In contrast, premenopausal women experienced a modest reduction in HDL-C, likely attributable to already elevated baseline values, suggesting a ceiling effect rather than an adverse adaptation.

TG increased across all groups, though the experimental groups exhibited substantially attenuated rises compared to controls. This pattern may reflect transient metabolic fluxes related to increased fatty acid mobilization during sustained moderate-intensity exercise[32], or suggest that a longer intervention may be required to elicit reductions. These findings challenge prior research indicating TG sensitivity to training[33], highlighting possible threshold effects or group-specific metabolic profiles.

The Castelli indices I and II, representing the ratios TC/HDL-C and LDL-C/HDL-C, respectively, exhibited mixed results. While both indices increased in the control group—suggesting elevated cardiovascular risk—the postmenopausal group showed modest reductions in Castelli II due to concurrent increases in HDL-C. These differential effects further support the idea that improvements in lipid ratios may occur independently of absolute lipid concentration changes and may serve as more sensitive markers of cardiovascular adaptation to exercise[34].

VLDL-C showed uniform reductions across all groups. While this might initially appear paradoxical given concurrent TG increases, it likely reflects a shift in lipoprotein particle transport and metabolism during sustained physical activity. VLDL particles are triglyceride-rich and contribute to atherogenesis upon conversion to LDL-C, thus their reduction may carry prognostic value for long-term vascular health[35].

Glucose regulation improved significantly in the premenopausal group, reflecting enhanced insulin sensitivity likely driven by concurrent reductions in adiposity. These effects are supported by previous findings linking

		Δ HDL-C	Δ CRI-I	Δ SBP	Δ DBP
Δ BMI	Post	0.244	-0.261	0.298	0.354
	Pre	-0.142	0.084	0.686**	0.552*
	Post/Pre	-0.061	-0.054	-0.533**	0.482**
Δ WC	Post	0.122	-0.006	0.219	0.222
	Pre	-0.484*	0.471*	0.555*	0.327
	Post/Pre	-0.311	0.255	0-398*	0.222
Δ HP	Post	0.166	-0.018	0.419	0.543
	Pre	-0.035	0.123	0.487*	0.370
	Post/Pre	-0.003	-0.030	0.304	0.341
Δ WC/HP	Post	0.052	-0.037	-0.015	-0.613
	Pre	-0.748**	0.540*	0.173	0.000
	Post/Pre	-0.472*	0.343	0.160	-0.110

Table 4. Correlation coefficients (Spearman's test) and their respective statistical significance, regarding the rates of change (Δ) from pre to post-test, between the anthropometric parameters and the remaining variables, for the total experimental group and for each of the individual experimental groups. HDL-C = High-density lipoprotein Cholesterol; CRI = Castelli Risk Index; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; BMI = Body Mass Index; WC = Waist Circumference; HP = Hip Circumference; p - statistical significance; * - statistically significant differences ($p < 0.05$); ** - statistically significant differences ($p < 0.01$). Correlation strength was interpreted using the following thresholds: $|\rho| < 0.30$ = weak; 0.30–0.49 = moderate; ≥ 0.50 = strong.

reduced visceral fat to improved glucose uptake and metabolic flexibility[36,37]. While postmenopausal women did not exhibit statistically significant changes, a downward trend was noted, which may become more pronounced with longer interventions or higher intensity training.

Taken together, these findings reinforce the value of structured multicomponent training in modulating cardiovascular risk markers. Notably, the intervention's moderate-to-vigorous intensity (60–80% HRmax), verified by heart rate monitoring and subjective exertion ratings, was sufficient to induce lipid and glycaemic adaptations in both groups—albeit to differing extents. The more robust changes in premenopausal women likely reflect preserved estrogenic influence on lipid metabolism and vascular responsiveness, while the relative attenuation in postmenopausal participants may be mitigated by the addition of longer duration or intensified protocols. From a clinical perspective, the intervention appeared to stabilize or slow the deterioration of key lipid variables in postmenopausal women, while promoting favorable shifts in the premenopausal group. These divergent trajectories highlight the importance of tailoring exercise prescriptions to hormonal status, with emphasis on lipid and glucose regulation in younger women, and cardiometabolic risk containment in older cohorts.

Magnitude and direction of the tension response

The multicomponent exercise intervention elicited significant reductions in both SBP and DBP across the experimental groups, aligning with consistent evidence that regular physical activity leads to blood pressure improvements, particularly in midlife and older women[11]. These effects are of clinical significance, as even modest SBP reductions (~ 5 mmHg) can yield a 10% decrease in coronary disease risk[38], and a 5–10 mmHg drop is associated with meaningful reductions in cardiovascular morbidity and mortality in hypertensive individuals[39]. In our study, postmenopausal women experienced more pronounced reductions in both SBP and DBP, which may reflect higher baseline values and a greater physiological margin for improvement. These results reinforce the idea that postmenopausal women, due to hormonal changes such as estrogen deficiency, may have a heightened cardiovascular risk profile[6], making them particularly responsive to interventions targeting vascular health. The structured exercise program was conducted at intensities ranging from 60 to 80% of maximal heart rate and included aerobic, resistance, flexibility, and balance components. While aerobic and resistance modalities are traditionally emphasized in blood pressure regulation, the inclusion of flexibility and balance training may have contributed synergistically, especially by reducing stress and enhancing autonomic function[8]. Although these components were not isolated in our analysis, they should be acknowledged as potential contributors to the favourable hemodynamic outcomes. In contrast, the control group—characterized by continued sedentary behaviour—exhibited an increase in SBP and DBP, underscoring the adverse consequences of physical inactivity and further reinforcing the protective role of supervised exercise. From a practical standpoint, these findings underscore the utility of moderate-to-vigorous multicomponent training in the primary prevention of hypertension. Weight loss, observed in both experimental groups, likely mediated part of this response, as reductions in body mass and waist circumference are known to correlate with improvements in vascular function[40]. This is supported by the significant correlations found in our study between changes in DBP and BMI, particularly among premenopausal participants. Overall, the observed blood pressure reductions in both groups—notably greater among postmenopausal women—support the incorporation of individualized, periodized exercise interventions in public health strategies aimed at mitigating cardiovascular risk among women during the menopausal transition.

Magnitude and direction of the association between anthropometric parameters

The multicomponent training program yielded clinically meaningful reductions in anthropometric parameters, particularly BMI, WC, HC, WHR, with differential responses between premenopausal and postmenopausal women. BMI significantly decreased in both experimental groups, a result consistent with numerous studies demonstrating the impact of physical exercise on adiposity[18,40]. This reduction is physiologically relevant, as elevated BMI is a well-established predictor of metabolic syndrome and cardiovascular disease risk[40]. Notably, the control group showed no significant changes, emphasizing the role of exercise in achieving favourable anthropometric adaptations. Although both groups experienced similar BMI reductions, postmenopausal women may have benefitted more due to their higher baseline values, aligning with prior findings[10].

WC decreased significantly in the postmenopausal group, while a non-significant reduction was observed in premenopausal women. Given that abdominal adiposity has stronger predictive power for cardiovascular events than BMI[41] this change in WC represents a meaningful improvement in cardiometabolic health. Importantly, the reduction in WC is likely linked to the observed BMI changes and may reflect a shift in fat distribution toward lower-risk profiles. Our findings support recent evidence that consistent exercise at moderate-to-vigorous intensities improves abdominal fat parameters, especially in older adults[42].

HC decreased significantly in both experimental groups and, unexpectedly, also in the control group. Although higher HC has been associated with cardioprotective effects[43], its interpretation must consider the accompanying changes in WC and WHR. In our study, reductions in HC were associated with improved cardiovascular markers, suggesting that the benefits of overall fat reduction may outweigh potential drawbacks of HC loss, particularly when not accompanied by increases in WC.

Waist-to-hip ratio, a more robust marker of cardiovascular risk than BMI alone[44], improved slightly in premenopausal women and remained stable in postmenopausal women, whereas it worsened in the control group. These shifts likely stem from the opposing trends in WC and HC and support the protective role of physical activity against central adiposity. Moreover, significant correlations between WHR and HDL-C observed in the premenopausal group further highlight the physiological link between body composition and lipid metabolism. Taken together, the results suggest that the exercise-induced reductions in BMI and WC—particularly among postmenopausal women—may be attributed to greater visceral fat mobilization, consistent with their higher baseline risk. This aligns with evidence suggesting that fat loss, especially from the abdominal region, enhances insulin sensitivity and lipid profiles, reinforcing the preventive potential of regular structured exercise[36]. From a mechanistic perspective, the moderate-to-vigorous intensity (60–80% HRmax) of the intervention likely played a central role in stimulating energy expenditure, promoting hormonal adaptations (e.g., increased irisin), and facilitating fat oxidation[45]. Although these markers were not measured directly, previous work confirms the role of irisin and similar myokines in mediating beneficial changes in body composition during aerobic and resistance training[46]. Given the statistically significant and directionally consistent reductions in BMI and WC, particularly among postmenopausal women, these anthropometric shifts should be interpreted as early indicators of reduced cardiovascular risk. The preservation of WHR or its modest improvement—alongside decreased SBP and DBP—suggests a multifaceted physiological benefit arising from structured, supervised multicomponent training. These effects may be especially relevant in clinical or community settings aiming to prevent the onset of metabolic syndrome or related complications in aging women.

While the present multicomponent program integrated aerobic, resistance, flexibility, and balance elements, the current manuscript primarily emphasized aerobic and resistance components when interpreting changes in lipid profiles and hemodynamic variables. However, it is important to recognize that balance and flexibility training—although lower in intensity—may confer additional cardiometabolic benefits, particularly in populations with heightened cardiovascular risk. Previous studies have indicated that flexibility-oriented modalities, such as yoga and Pilates, can positively influence lipid metabolism, reduce blood pressure, and improve autonomic function, potentially via reductions in stress and enhancements in endothelial function and circulation[8,9]. These effects, although not isolated in our analyses, may have contributed to the observed improvements in postmenopausal women, particularly regarding blood pressure and waist circumference. Moreover, given that balance and flexibility exercises are often more accessible and better tolerated by older adults, future research should explore their independent and combined effects on cardiometabolic markers to support a more comprehensive and individualized intervention model.

This study presents several limitations that warrant consideration. The use of a non-randomized convenience sample may limit the generalizability of the findings and introduces a potential risk of selection bias. Although participants were stratified by menopausal status, random allocation procedures were not implemented, which may influence internal validity. In addition, possible confounding factors—such as dietary intake, sleep quality, and psychosocial stress—were not systematically monitored. While participants were encouraged to maintain consistent lifestyle habits, unmeasured behavioural variables may have influenced the intervention's outcomes. Biochemical evaluations were limited to pre- and post-intervention measures and focused on standard cardiometabolic markers (e.g., lipid profile, glucose), in line with the clinical protocols of the National Health System. However, more sensitive indicators of metabolic function—such as markers of insulin resistance (e.g., HOMA-IR), systemic inflammation (e.g., CRP, IL-6), and vascular function—were not assessed. This may have limited our ability to detect subclinical or early physiological adaptations to exercise. Furthermore, although validated and standardized instruments were employed, inherent measurement variability and the timing of outcome collection (restricted to two time points) must be acknowledged. Another limitation of the present study pertains to the lack of intermediate assessments throughout the 26-week intervention. Although baseline and post-intervention data were collected, the absence of periodic evaluations limits the capacity to monitor the temporal evolution of physiological adaptations, distinguish transient from stable changes, and adapt the training protocol in response to unexpected trends (e.g., stagnation or adverse responses). Intermediate testing would have allowed for a more nuanced understanding of the mechanisms underlying training adaptations and

the optimization of individualized training progression. Future studies should consider incorporating periodic monitoring to enhance both the interpretability and the responsiveness of multicomponent interventions, particularly in long-duration public health applications. Finally, the modest sample size, particularly in the postmenopausal and control groups, may have reduced the power to detect smaller yet clinically relevant effects. Future investigations employing larger, randomized samples and expanded biomarker panels are recommended to enhance both mechanistic insight and external validity.

Conclusions

This study demonstrated that a 26-week periodized multicomponent exercise program induced distinct and clinically relevant cardiometabolic and anthropometric adaptations in sedentary premenopausal and postmenopausal women. Specifically, premenopausal women experienced greater improvements in lipid metabolism, including reductions in total cholesterol and LDL-C, as well as enhanced glucose regulation, underscoring the sensitivity of this group to exercise-induced metabolic adaptations. In contrast, postmenopausal women exhibited more pronounced improvements in hemodynamic and anthropometric measures, notably reductions in systolic and diastolic blood pressure, waist circumference, and body mass index—parameters closely associated with cardiovascular risk in this population. These findings support the application of structured, periodized multicomponent training programs—which include aerobic, resistance, flexibility, and balance components distributed across progressive intensity phases—as a targeted strategy for mitigating age- and hormone-related health risks in middle-aged women. Given the divergent responses observed between menopausal groups, the use of differentiated programming is recommended: emphasizing metabolic and lipid-targeted aerobic and resistance work for premenopausal women, and blood pressure- and anthropometry-focused training (e.g., moderate-intensity aerobic and circuit-based resistance exercise) for postmenopausal women. Although limited by sample size and the absence of mechanistic biomarkers (e.g., inflammation or insulin sensitivity markers), this study underscores the clinical utility of well-structured physical activity as a non-pharmacological intervention for cardiovascular risk reduction in sedentary women across different reproductive stages.

Data availability

Data are available from the corresponding author (Bruno Figueira) upon reasonable request and with permission of the University de Évora.

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Author contributions

B.F. conceived the idea and designed the study with A.R. B.F., J.B., A.R. and An.R. did the statistical analyses and drafted the relevant manuscript sections. All the authors drafted the manuscript and the supplemental text. All authors provided several rounds of critical feedback and specialised expertise, and contributed to the study design during subsequent revisions. B.F. is the guarantor, is responsible for the overall content and accepts full responsibility for the work and/or the conduct of the study, had access to the data and controlled the decision to publish.

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Declarations

Competing interests
The authors declare no competing interests.

Ethics approval and consent to participate
This study was approved by the University of Évora Ethics Committee (ref. 22137). All procedures were conducted in accordance with the Declaration of Helsinki. Risk mitigation strategies included supervised training by qualified personnel and medical monitoring protocols to ensure participant safety during physical exertion. Participants gave informed consent to participate in the study before taking part.

Additional information
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