

Original Article



Effects of *Garcinia Cambogia* and Aerobic Exercise on Metabolic and Physical Fitness Indicators in Obesity Class I: A Randomized Controlled Trial

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Abstract

Introduction: Aerobic exercise (AE) and medicinal herbs may synergistically improve metabolic regulation and physical fitness. Therefore, this study examined the effects of an 8-week aerobic training combined with *garcinia cambogia* (GC) supplementation on metabolic and fitness outcomes in women with class I obesity.

Methods: This randomized controlled trial included 80 class I obese women (BMI > 30) aged 35–45 years employed at Iran Khodro Factory. Participants were randomly assigned to control, AE, GC, or combined intervention groups. Physical fitness and biochemical variables were assessed pre- and post-intervention. Moreover, exercise was performed three times per week, and hydroxycitric acid was supplemented daily at the recommended dose. Data were analyzed using one-way ANOVA and paired t-test.

Results: Significant reductions were observed in serum aspartate transferase (MD: -3.7 U/L; 95% CI: -6.8 to -0.6; $P=0.021$), alanine aminotransferase (MD: -4.0 U/L; 95% CI: -6.2 to -1.8; $P=0.015$), triglycerides (MD: -13.3 mg/dL; 95% CI: -18.2 to -8.4; $P=0.001$), total cholesterol (MD: -15.5 mg/dL; 95% CI: -22.1 to -8.9; $P=0.012$), very-low-density lipoprotein (MD: -5.8 mg/dL; 95% CI: -8.5 to -3.1; $P=0.001$), and fasting blood glucose (MD: -16.4 mg/dL; 95% CI: -22.5 to -10.3; $P=0.001$) in the intervention groups compared with the control group. Conversely, high-density lipoprotein cholesterol (MD: +8.4 mg/dL; 95% CI: 5.2 to 11.6; $P=0.004$) and peak oxygen consumption (MD: +9.8 mL/kg/min; 95% CI: 7.2 to 12.4; $P=0.001$) substantially increased following the interventions.

Conclusion: The combination of AE and GC supplementation effectively improved lipid metabolism, liver enzyme profiles, and aerobic capacity in obese class I women.

Keywords: *Garcinia cambogia*, Exercise, Lipids, Liver, Physical fitness

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Introduction

Obesity is recognized as a major global public health challenge, which is associated with an increased risk of chronic metabolic conditions, such as type 2 diabetes, cardiovascular diseases, and non-alcoholic fatty liver disease. These conditions not only reduce individuals' quality of life but also impose a serious economic burden on healthcare systems (1).

Obesity is frequently accompanied by adverse changes in metabolic markers, including elevated fasting blood glucose (FBG); dyslipidemia characterized by increased total cholesterol (TC), low-density lipoprotein (LDL), and triglycerides (TG), and decreased high-density lipoprotein (HDL); and elevated liver enzymes, such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST). These markers are the critical indicators of metabolic health and liver function, highlighting the importance of strategies aimed at improving such markers (2).

In recent decades, non-pharmacological interventions, including herbal supplements and physical activity, have

received growing attention as approaches to manage obesity and enhance metabolic health. For instance, *Garcinia cambogia* (GC), a tropical plant containing the active compound hydroxycitric acid (HCA), has been investigated for its potential effects on weight management and metabolic improvement. According to preliminary studies, HCA may influence lipid and glucose metabolism by mechanisms such as inhibiting fat synthesis and promoting fat oxidation. However, results remain inconsistent, and the standalone efficacy of GC is still uncertain (3). Similarly, some evidence suggests that this supplement may help regulate blood glucose and improve insulin sensitivity, though further research is needed to confirm these effects.

Aerobic exercise (AE), on the other hand, is a well-established non-pharmacological intervention known to improve glucose metabolism, reduce body fat, and enhance liver function. More precisely, regular physical activities may positively influence metabolic and hepatic markers by increasing energy expenditure, reducing systemic inflammation, and enhancing lipid metabolism.

The magnitude of these effects, however, depends on exercise intensity, duration, and modality (4).

Given the potential for both GC and AE to modulate distinct metabolic pathways, it is hypothesized that combining these interventions may produce complementary or synergistic effects. In other words, such a combination could potentially improve metabolic markers, promote weight reduction, and enhance physical fitness. Nonetheless, information regarding their combined effects, particularly in obese class I women, remains limited (5). Due to physiological and hormonal differences, including variations in fat metabolism and hormonal responses to exercise and supplements, women may respond differently to these interventions compared to men, highlighting the need for targeted research in this population.

Furthermore, considering the high prevalence of non-alcoholic fatty liver disease among obesity class I individuals, assessing the impact of these interventions on liver enzymes (e.g., ALT and AST) is particularly important, as these enzymes serve as the sensitive indicators of liver health (6).

Accordingly, this study aims to investigate the combined effects of GC supplementation and AE on FBG, lipid profile (TC, LDL, HDL, and TG), and liver enzyme levels (ALT and AST) in obese class I women. More precisely, the research seeks to determine whether this combined approach can improve metabolic and physical fitness indicators more effectively than either intervention alone. It is anticipated that the findings will provide valuable insights into the efficacy and safety of this combined strategy, supporting the development of effective non-pharmacological interventions to control obesity and improve metabolic health in obese class I women. Ultimately, these outcomes may contribute to enhancing the quality of life while reducing the risk of chronic diseases and alleviating the burden of obesity-related conditions in this population.

The scientific gap in this research is the synergistic efficacy of the simultaneous combination of AE and GC supplementation on a wide range of health parameters. It should be noted that previous studies have mainly focused on the individual effects of each of these interventions while not being able to comprehensively evaluate their combined effects on key fitness indicators (e.g., peak oxygen consumption [$\text{VO}_{2\text{peak}}$]) and metabolic and liver markers (e.g., fasting blood sugar [FBS], HDL, and ALT/AST enzymes) in a specific and at-risk population (obesity class I women). Another gap is our limited understanding of how the combination of these two factors and the contribution of each factor affect the combined non-pharmacological interventions for managing excess weight and improving health profiles.

Materials and Methods

This randomized controlled trial with a pretest–posttest design was conducted on the female employees of Iran Khodro Factory. The inclusion criteria were age range

of 35–45 years, body mass index (BMI) $\geq 30 \text{ kg/m}^2$, no history of cardiovascular, metabolic, or musculoskeletal disorders, lack of smoking, no medication use, and no dietary supplements in the previous two months 80 participants. First, the research process was explained to volunteers in an introductory session, and 200 subjects who met the inclusion criteria were randomly selected as the study sample. Nonetheless, participants were excluded if they had any musculoskeletal injuries or physical disabilities that could limit participation in exercise training, or if they had diagnosed chronic diseases (e.g., cardiovascular, metabolic, or endocrine disorders) that could interfere with the study outcomes. Likewise, the other exclusion criteria included the use of medications affecting metabolism or lipid profile, participation in regular structured exercise programs during the previous three months, and unwillingness or inability to continue participation or comply with the study protocol. Then, demographic information, medical history, and physical activity status were collected using a standardized questionnaire. Moreover, written informed consent was obtained from all participants prior to enrollment. Finally, baseline measurements were conducted after a 12-hour overnight fast at 8 am.

Sample Size Calculation

The sample size was calculated a priori using G*Power software (version 3.1) based on the primary outcome variable, namely, $\text{VO}_{2\text{peak}}$. According to previous studies and expected changes in $\text{VO}_{2\text{peak}}$, mean values of approximately $36.0 \pm 4.0 \text{ mL/kg/min}$ at baseline and an expected improvement to approximately $41.0 \pm 5.0 \text{ mL/kg/min}$ following the intervention were considered for the sample size estimation. Furthermore, a one-way analysis of variance (ANOVA) (fixed effects, omnibus test) was selected for comparison among four independent groups. Additionally, the significance level and the statistical power ($1-\beta$) were set at $\alpha = 0.05$ and 0.80, respectively. Based on these assumptions, the required total sample size was estimated to be 68 participants (17 participants per group). Considering a potential dropout rate of approximately 15%, the final sample size was then increased to 80 participants (20 participants per group). Therefore, the study was adequately powered to detect clinically and statistically meaningful differences in $\text{VO}_{2\text{peak}}$ and related physical performance outcomes.

Randomization and Allocation Concealment

Using a computer-generated randomization sequence, the participants were randomly assigned to four groups in a 1:1:1:1 ratio, including control, AE, GC, and AE+GC. To ensure unbiased group allocation, the randomization sequence was generated using a reproducible random seed and then implemented through the Fisher–Yates shuffling algorithm.

In addition, allocation concealment was ensured using sealed, opaque, sequentially numbered envelopes that were prepared by an independent researcher who was not

involved in participant recruitment, baseline assessment, intervention administration, or data analysis. After the completion of baseline measurements, participants were assigned to their respective groups by opening the envelopes in strict sequential order.

Blinding

Due to the nature of the AE intervention, participant blinding was not feasible. However, the outcome assessors and statistical analysts were blinded to group assignments throughout the data collection and statistical analysis processes in order to minimize potential measurement and analytical bias.

Participant Retention and Intervention Adherence

Overall, 200 volunteers were screened for eligibility; however, 120 individuals were excluded due to not meeting the inclusion criteria or declining to participate. Accordingly, 80 participants were randomized into control (n=20), AE (n=20), GC (n=20), and AE+GC (n=20) groups. During the 8-week intervention, three participants withdrew due to personal reasons unrelated to the study protocol (one from GC group and two from the AE+GC group). Therefore, 77 participants completed the study and were included in the final analyses. It should be mentioned that adherence to the intervention was monitored using weekly attendance records for exercise sessions and capsule counts for supplementation. The overall compliance rate was 92%, and no intervention-related adverse events were reported (Figure 1).

Blood samples were collected from the antecubital vein under fasting conditions and processed by centrifugation at 3,500 rpm for 10 minutes. The obtained serum/plasma samples were then stored at -80°C until biochemical analyses. Subsequently, serum TG, HDL, and AST were measured using Bionic laboratory kits, and FBG was determined by the enzymatic method using a Bionic AutoAnalyzer 5000. Furthermore, quality control procedures and duplicate measurements were performed to ensure analytical accuracy.

Likewise, leg explosive power and $\text{VO}_{2\text{peak}}$ were assessed using the Sargent vertical jump test and the Cooper treadmill test, respectively. Further, exercise intensity was individually prescribed using the Karvonen equation.

Participants in the GC and AE+GC groups received 1500 mg/day of GC extract standardized to 60% HCA for 8 weeks, while the control and exercise groups received identical placebo capsules. Supplement adherence was then assessed by capsule counts, and the overall compliance rate was 92%.

Given the nature of the exercise intervention, participants were not blinded to exercise allocation; however, blinding was maintained for supplementation allocation, and outcome assessors were unaware of supplement assignment. To maintain similar nutrient intake, all participants followed a standardized dietary plan with alternative food options, and the use of additional

supplements, medications, or structured exercise programs was prohibited during the study. Baseline BMI values confirmed obesity status in all groups, with no significant between-group differences (control: 32.8 ± 2.1 , exercise: 33.1 ± 1.9 , exercise+GC: 32.5 ± 2.3 , and GC: $33.0 \pm 2.0 \text{ kg/m}^2$).

Aerobic Exercise Program

The protocol consisted of 8 weeks of progressive treadmill running, with 3 sessions per week (at least 48 hours apart for recovery), totaling 24 sessions. In addition, each session lasted 45–60 minutes and was structured as follows:

Warm-up: 5–10 minutes of light walking (4–5 km/h, 0% incline) + dynamic stretches (arm circles and lunges).

Main Session: Continuous net running time on a motorized treadmill (Technogym Run 600, digital display for precise distance/time tracking; distances measured on treadmill, not field running, for controlled intensity).

Cool-Down: 5–10 minutes of walking (3–4 km/h, 0% incline) + static stretches for legs and back.

Intensity Parameters: Based on pretest $\text{VO}_{2\text{peak}}$ (mean 28 mL/kg/min), intensity was prescribed as $\% \text{VO}_{2\text{max}}$, equated to speed (km/h) and incline (%) via American College of Sports Medicine running equation ($\text{VO}_2 = 3.5 + 0.2 \times \text{speed} + 0.9 \times \text{speed} \times \text{incline} \%$). Individual adjustments used heart rate monitoring (Polar H10; target 70–90% HR_{max}). Progressive overload increased distance (50–100 m/session), time (1–2 min/session), and intensity (5% every 2 weeks, capped at 86–90% to avoid overexertion).

Adherence: Participants (AE and AE+GC groups) required $\geq 80\%$ attendance (24 sessions). They were supervised by a trainer with weekly checklists and the Strava app logging. Mean adherence: 92% (SD = 5%); total

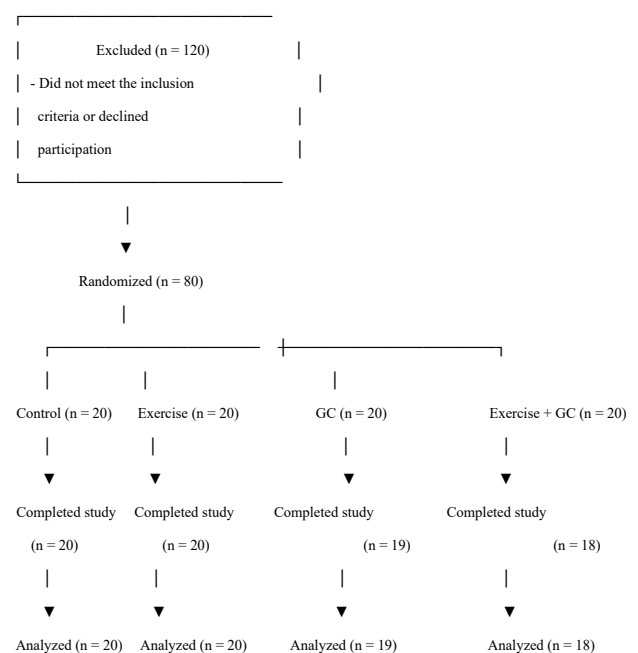


Figure 1. CONSORT Diagram Showing Recruitment, Randomization, and Completion of Participants
Note: GC: *Garcinia cambogia*; CONSORT: Consolidated Standards of Reporting Trials

absences: 12/160 sessions (7.5%, mainly work-related; no > 2 consecutive per participant). Low adherers (<80%) were retained but covaried in ANOVA. No related injuries were reported (7, 8).

It should be mentioned that the exercise protocol was adjusted to the subjects' baseline fitness level and health status by conducting several pilot tests (Table 1).

Garcinia Cambogia

Participants in the GC and AE + GC groups received 1500 mg/day of GC extract, standardized to 60% HCA (Nature's Bounty Laboratories, USA; batch No. NB-GC-2247). The extract was administered as two 750 mg capsules daily, one taken 30 minutes before breakfast and one 30 minutes before dinner for a total of 8 consecutive weeks.

Compliance and Monitoring

Participant adherence was monitored using weekly supplementation logs and capsule counts, with an average compliance rate of 92%. To minimize bias while ensuring safety, participants were instructed not to consume any other herbal or dietary supplements during the intervention period. Additionally, they were advised to maintain their usual diet and physical activity levels, except for the prescribed exercise program. No adverse events or side effects were reported during the intervention, and regular weekly check-ins ensured timely reporting of any health-related issues.

Rationale for Dose and Duration

The chosen dose of 1500 mg/day and 8-week duration were based on previous clinical studies demonstrating the efficacy and safety of GC supplementation in obesity class I and obese adults, particularly in modulating lipid metabolism, glucose homeostasis, and body composition. Moreover, the timing of supplementation before meals was intended to maximize the absorption of HCA and enhance its effects on appetite regulation and fat metabolism (9).

In addition, serum levels of the research indicators were measured using commercially available bionic laboratory kits. Furthermore, TG (serial No. 142979), HDL cholesterol (HDL-C; serial No. 141766), and AST (serial No. 141421) were determined using different kits, according to the manufacturer's instructions.

Further, TC was enzymatically measured using standard laboratory procedures. However, low-density

lipoprotein cholesterol (LDL-C) and very low-density lipoprotein cholesterol (VLDL-C) were not computed by direct enzymatic assays; instead, they were calculated from the measured lipid parameters using the Friedewald equation. Specifically, LDL-C and VLDL-C were determined as TC-HDL-C (TG/5) and TG/5, respectively. It is noteworthy that these values were applied only for samples with TG levels below 400 mg/dL in accordance with standard methodological guidelines.

Likewise, FBS was estimated using the enzymatic method with the Bionic Autoanalyzer 5000 (automated biochemistry autoanalyzer; serial No. 31413 Bionic, Iran).

All measurements were performed in duplicate to ensure accuracy, following the manufacturer's protocols for sample handling, reagent preparation, and incubation times. Additionally, calibration curves and internal quality control samples were included in each assay run in order to validate the precision and reliability of the results. Blood samples were collected in the morning after a 12-hour overnight fast, allowed to clot for 30 minutes, and then centrifuged at 3,000 rpm for 10 minutes to separate serum, which was stored at -80°C until analysis. To minimize bias in the measurements, laboratory personnel were blinded to group allocation.

Data Analysis

For statistical analysis and comparison of groups, one-way ANOVA and paired T-test were performed for between-group and within-group comparisons, respectively, after confirming the normal distribution of the data and homogeneity. The interaction determined if the changes in the dependent variables (e.g., FBS and HDL) over time were significantly different across the various treatment groups ($P < 0.05$).

Results

Statistical descriptions, including demographics and characteristics of the subjects, were collected and recorded (Table 2).

One-way ANOVA revealed significant between-group differences in body weight ($P = 0.003$) and BMI ($P = 0.001$) following the intervention period, whereas no significant differences were observed at baseline for body weight ($P = 0.742$) and BMI ($P = 0.688$). Likewise, paired t-test analyses demonstrated sharp reductions in body weight in the exercise ($P = 0.004$), exercise + GC ($P = 0.001$), and GC ($P = 0.018$) groups. Similarly, BMI significantly

Table 1. Incremental Aerobic Exercise Program

Week	Mean Distance (m/Session)	Mean Net Time (Minute/Session)	Intensity (%VO _{2max})	Sample Speed (km/h)	Sample Incline (%)
1	2700	21	65–70	8.0–8.5	1.0–1.5
2	2850	22.5	71–75	8.5–9.0	1.5–2.0
3	2975	25	76–80	9.0–9.5	2.0–2.5
4	3150	27	81–85	9.5–10.0	2.5–3.0
5	3350	28.5	86–90	10.0–10.5	2.5–3.0
6	3550	30	86–90	10.5–11.0	2.5–3.0
7	3750	31.5	86–90	11.0–11.5	2.0–2.5

decreased in the exercise ($P=0.002$), exercise+GC ($P=0.001$), and GC ($P=0.009$) groups. Additionally, the highest reductions in body weight and BMI were noted in the combined intervention group. Physiological variables (VO_{2peak} , Sargent Jump, Handgrip Strength, Medicine Ball Throw, and Standing Long Jump) were analyzed by one-way repeated-measures ANOVA (Table 3).

One-way ANOVA (Figure 2, Table 3) confirmed a substantial between-group difference only in VO_{2peak} following the intervention period ($P=0.001$). Conversely, no significant between-group differences were observed in Sargent jump ($P=0.058$), handgrip strength ($P=0.121$), medicine ball throw ($P=0.119$), or standing long jump ($P=0.314$). Paired t-test results indicated remarkable within-group improvements in VO_{2peak} in the exercise

($P=0.001$), exercise+GC ($P=0.001$), and GC ($P=0.032$) groups. For the Sargent jump, significant improvements were found in the exercise ($P=0.041$) and exercise+GC ($P=0.001$) groups, whereas no marked change was noticed in the GC group ($P=0.921$). Handgrip strength significantly enhanced in the exercise ($P=0.038$), exercise+GC ($P=0.041$), and GC ($P=0.012$) groups. Similarly, medicine ball throw showed significant increases in all intervention groups (exercise: $P=0.001$; exercise+GC: $P=0.001$; GC: $P=0.028$). Additionally, standing long jump considerably improved in all intervention groups (exercise: $P=0.002$; exercise+GC: $P=0.001$; GC: $P=0.044$).

Based on the analysis of the lipid profile, significant between-group differences were found in TG, LDL-C,

Table 2. Changes in Body Weight and BMI Following the Interventions: Descriptive Statistics and Between-Group Comparisons (Mean $\pm$ SD)

Variable	Group	Pre (Mean $\pm$ SD)	Post (Mean $\pm$ SD)	Change (%)	95% CI for Change (Exe + GC)	P (Within Con)	P (Within Exe)	P (Within Exe + GC)	P (Within GC)	P (Between Pre)	P (Between Post)
Weight (kg)	Con	82.1 $\pm$ 6.2	81.5 $\pm$ 5.9	-0.7	[-4.5, -0.3]	0.360	—	—	—	0.742	0.003*
	Exe	83.4 $\pm$ 5.8	80.9 $\pm$ 5.4	-3.0		—	0.004*	—	—		
	Exe+GC	84.2 $\pm$ 6.1	79.1 $\pm$ 5.7	-6.0		—	—	0.001*	—		
	GC	82.7 $\pm$ 5.5	80.8 $\pm$ 5.2	-2.3		—	—	—	0.018*		
BMI (kg/m ²)	Con	31.2 $\pm$ 1.2	30.9 $\pm$ 1.1	-1.0	[-9.2, -2.1]	0.310	—	—	—	0.688	0.001*
	Exe	31.8 $\pm$ 1.4	30.6 $\pm$ 1.3	-3.7		—	0.002*	—	—		
	Exe+GC	32.1 $\pm$ 1.6	29.8 $\pm$ 1.5	-7.2		—	—	0.001*	—		
	GC	31.5 $\pm$ 1.3	30.5 $\pm$ 1.2	-3.1		—	—	—	0.009*		

Note. BMI: Body mass index; SD: Standard deviation. Con: Control; Exe: Exercise; Exe+GC: Exercise+Garcinia cambogia; GC: Garcinia cambogia. $P<0.05$.

Table 3. Between-Group Comparisons of Physiological Variables Following the Interventions: Descriptive Statistics and One-Way ANOVA Results (Mean $\pm$ SD)

Variable	Group	Pre (Mean $\pm$ SD)	Post (Mean $\pm$ SD)	Change (%)	95% CI for Change	P (Within Con)	P (Within Exe)	P (Within Exe + GC)	P (Within GC)	P (Between Pre)	P (Between Post)
VO_{2peak} (mL/kg/ min)	Con	35.7 $\pm$ 5.1	35.3 $\pm$ 6.2	-1.1	[7.2, 12.4]	0.684	—	—	—	0.684	0.001*
	Exe	36.5 $\pm$ 3.7	41.5 $\pm$ 5.5	+13.7		—	0.001*	—	—		
	Exe+GC	36.8 $\pm$ 4.4	41.5 $\pm$ 4.5	+9.7		—	—	0.001*	—		
	GC	36.6 $\pm$ 2.9	38.9 $\pm$ 6.7	+6.3		—	—	—	0.032*		
Sargent jump (cm)	Con	22 $\pm$ 4	22 $\pm$ 3	0.0	[-5.2, 2.6]	0.772	—	—	—	0.772	0.058
	Exe	22 $\pm$ 2	24 $\pm$ 5	+9.1		—	0.041*	—	—		
	Exe+GC	22 $\pm$ 3	27 $\pm$ 5	+22.7		—	—	0.001*	—		
	GC	21 $\pm$ 2	21 $\pm$ 3	0.0		—	—	—	0.921		
Handgrip strength (kg)	Con	30 $\pm$ 3	30 $\pm$ 2	0.0	[1.2, 2.8]	0.655	—	—	—	0.655	0.121
	Exe	32 $\pm$ 2	33 $\pm$ 2	+3.1		—	0.041*	—	—		
	Exe+GC	31 $\pm$ 1	32 $\pm$ 2	+3.2		—	—	0.012*	—		
	GC	29 $\pm$ 3	31 $\pm$ 1	+6.9		—	—	—	0.038*		
Medicine ball throw (m)	Con	4.32 $\pm$ 0.18	4.41 $\pm$ 0.20	+2.2	[0.82, 1.24]	0.701	—	—	—	0.701	0.119
	Exe	4.35 $\pm$ 0.21	5.01 $\pm$ 0.24	+15.2		—	0.001*	—	—		
	Exe+GC	4.38 $\pm$ 0.17	5.42 $\pm$ 0.26	+23.7		—	—	0.001*	—		
	GC	4.30 $\pm$ 0.19	4.68 $\pm$ 0.22	+8.8		—	—	—	0.028*		
Standing long jump (m)	Con	1.63 $\pm$ 0.05	1.75 $\pm$ 0.06	+7.4	[0.18, 0.39]	0.689	—	—	—	0.689	0.314
	Exe	1.66 $\pm$ 0.07	1.86 $\pm$ 0.08	+12.0		—	0.002*	—	—		
	Exe+GC	1.68 $\pm$ 0.06	1.97 $\pm$ 0.07	+17.3		—	—	0.001*	—		
	GC	1.68 $\pm$ 0.06	1.78 $\pm$ 0.07	+5.9		—	—	—	0.044*		

Note. SD: Standard deviation; ANOVA: Analysis of variance. Con: Control; Exe: Exercise; Exe+GC: Exercise+Garcinia cambogia; GC: Garcinia cambogia. $P<0.05$.

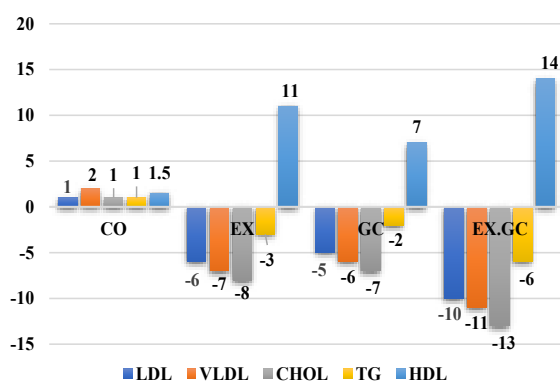


Figure 2. Comparison of Lipid Profile Changes After Completing the 8-Week Protocol in the 4 Experimental and Control Groups

Note. LDL: Low-density lipoprotein; VLDL: Very low-density lipoprotein; CHOL: Cholesterol; TG: Triglyceride; HDL: High-density lipoprotein; Con: Control; Exe: Exercise; Exe+GC: Exercise+ *Garcinia cambogia*; GC: *Garcinia cambogia*

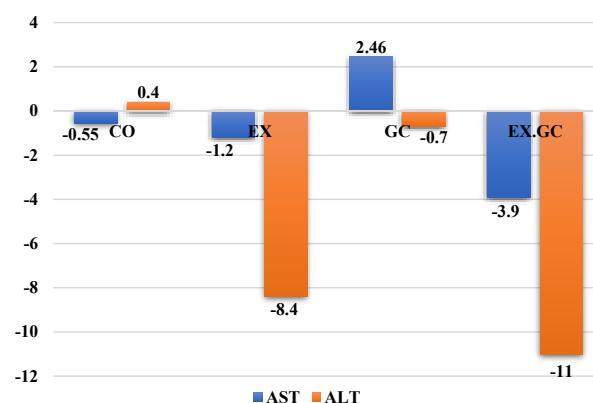


Figure 3. Comparison of Liver Enzyme Changes After Completing the 8-Week Protocol in the 4 Experimental and Control Groups

Note. ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; Con: Control; Exe: Exercise; Exe+GC: Exercise+ *Garcinia cambogia*; GC: *Garcinia cambogia*

VLDL-C, TC, and HDL-C following the intervention period ($P < 0.05$). The greatest reduction in TG was observed in the exercise+GC group. However, TC showed the most pronounced improvement in this group. Similarly, HDL-C levels notably increased in the exercise and exercise+GC groups, indicating the beneficial effects of exercise-based interventions on HDL-C. Overall, the exercise+GC group represented the most favorable changes across lipid profile variables compared with the control group.

Biochemical parameters related to liver health and blood sugar metabolism, including ALT and AST enzymes as well as FBS, were examined as a health profile of obesity class I women. Details of these percentage changes, time and group effects, as well as the effect sizes of these interventions, underwent assessment (Table 5, Figure 3).

Based on the results of one-way ANOVA, there were no significant differences among the groups at baseline for ALT ($P = 0.712$), AST ($P = 0.734$), or FBS ($P = 0.689$). However, significant between-group differences were observed in ALT ($P = 0.050$), AST ($P = 0.050$), and FBS ($P = 0.001$) after the intervention period.

Within-group analyses revealed significant reductions in ALT levels in the exercise ($P = 0.001$), exercise+GC ($P = 0.001$), and GC ($P = 0.022$) groups. In addition, ALT levels decreased by 14.6%, 20.4%, and 7.4% in the exercise, exercise+GC, and GC groups, respectively, whereas the control group showed only a 2.4% reduction.

Similarly, AST levels substantially decreased in the exercise ($P = 0.002$), exercise+GC ($P = 0.001$), and GC ($P = 0.031$) groups. The percentage reduction in AST was 14.55% in the exercise group, 16.23% in the exercise+GC group, and 6.20% in the GC group compared with a 2.59% decline in the control group.

FBS levels also displayed significant reductions in the exercise ($P = 0.001$), exercise+GC ($P = 0.001$), and GC ($P = 0.004$) groups. The reductions were 10.09%, 10.21%, and 7.73% in the exercise, exercise+GC, and GC groups, respectively, whereas the control group exhibited a 1.45% reduction.

Based on the results, the greatest improvements in ALT

and AST were observed in the combined exercise+GC group. The 95% confidence intervals for the changes in the combined intervention group were -6.2 to -1.8 U/L for ALT, -6.8 to -0.6 U/L for AST, and -22.5 to -10.3 mg/dL for FBS, indicating that the combined intervention exerted greater beneficial effects on liver enzymes and glycemic control compared with the single interventions.

Discussion

Our findings demonstrated reductions in LDL, VLDL, cholesterol, and TG levels across all intervention groups, which is consistent with the results of another study (10). These improvements are likely attributable to AE, which can reduce circulating LDL and VLDL levels by enhancing lipid oxidation and increasing lipoprotein lipase (LPL) activity. The observed decrease in VLDL suggests an enhancement in lipid metabolism resulting from the effects of exercise and supplementation, which corroborates the findings of van Zwol et al (11). In addition, AE may contribute to improved lipid profiles by increasing insulin sensitivity and upregulating enzymes involved in fat oxidation, which is in line with the findings of Chen et al (12). Similarly, reductions in TG levels reported by Mogharnasi et al and Fakhri et al (13, 14) support the current findings. Variations across studies may arise from differences in intervention protocols, assessment methodologies, participant demographics (e.g., age and gender), and obesity levels.

The decline in plasma TG may be related to enhanced TG catabolism via increased LPL activity, which facilitates TG hydrolysis and promotes uptake into peripheral tissues (15, 16).

HDL influences glycemic regulation following exercise. More precisely, elevated HDL may promote glucose uptake in skeletal muscle, activate AMP-activated protein kinase signaling, and stimulate insulin secretion from pancreatic β -cells (17). Mechanisms underlying exercise-induced HDL increases include reduced hepatic HDL catabolism and enhanced apolipoprotein A1 synthesis, which are both associated with weight loss.

Hajinia et al and Hoseini et al found results similar to

Table 4. Changes in Lipid Profile Parameters Following the Interventions: Descriptive Statistics, Within-Group and Between-Group Comparisons (Mean $\pm$ SD)

Variable	Group	Pre (Mean $\pm$ SD)	Post (Mean $\pm$ SD)	Change (%)	95% CI for Change	P (Within Con)	P (Within Exe)	P (Within Exe + GC)	P (Within GC)	P (Between Pre)	P (Between Post)
TG (mg/dL)	Con	112.5 $\pm$ 5.3	113.0 $\pm$ 5.6	+0.45	[-18.2, -8.4]	0.701	—	—	—	0.701	0.001*
	Exe	116.0 $\pm$ 6.3	105.1 $\pm$ 6.7	-9.41		—	0.001*	—	—		
	Exe + GC	114.5 $\pm$ 7.0	100.7 $\pm$ 5.8	-12.08		—	—	0.001*	—		
	GC	115.2 $\pm$ 6.5	108.1 $\pm$ 7.1	-6.16		—	—	—	0.012*		
HDL (mg/dL)	Con	41.4 $\pm$ 3.2	40.9 $\pm$ 3.6	-1.28	[5.2, 11.6]	0.688	—	—	—	0.688	0.001*
	Exe	42.1 $\pm$ 4.0	48.1 $\pm$ 3.9	+14.26		—	0.001*	—	—		
	Exe + GC	41.6 $\pm$ 3.0	47.5 $\pm$ 6.2	+14.05		—	—	0.041*	—		
	GC	42.2 $\pm$ 3.1	43.0 $\pm$ 3.1	+1.99		—	—	—	0.041*		
LDL (mg/dL)	Con	120.5 $\pm$ 12.3	119.8 $\pm$ 11.9	-0.6	[-18.7, -12.3]	0.732	—	—	—	0.732	0.050*
	Exe	122.1 $\pm$ 13.2	110.4 $\pm$ 10.8	-9.7		—	0.001*	—	—		
	Exe + GC	124.7 $\pm$ 14.0	102.5 $\pm$ 9.2	-17.8		—	—	0.033*	—		
	GC	121.3 $\pm$ 12.8	115.6 $\pm$ 11.4	-4.7		—	—	—	0.015*		
CHOL (mg/dL)	Con	180.2 $\pm$ 18.5	178.9 $\pm$ 17.8	-0.7	[-22.1, -8.9]	0.745	—	—	—	0.745	0.050*
	Exe	182.4 $\pm$ 19.2	165.7 $\pm$ 16.3	-9.1		—	0.001*	—	—		
	Exe + GC	185.6 $\pm$ 20.1	152.3 $\pm$ 14.5	-17.9		—	—	0.028*	—		
	GC	181.8 $\pm$ 18.9	172.4 $\pm$ 17.2	-5.2		—	—	—	0.003*		
VLDL (mg/dL)	Con	33.0 $\pm$ 5.1	32.6 $\pm$ 4.9	-1.2	[-8.5, -3.1]	0.709	—	—	—	0.709	0.001*
	Exe	33.6 $\pm$ 5.3	28.5 $\pm$ 4.1	-15.2		—	0.001*	—	—		
	Exe + GC	34.2 $\pm$ 5.4	26.1 $\pm$ 3.8	-23.7		—	—	0.001*	—		
	GC	33.3 $\pm$ 5.2	29.7 $\pm$ 4.4	-10.8		—	—	—	0.015*		

Note. BMI: Body mass index; SD: Standard deviation; LDL: Low-density lipoprotein; VLDL: Very low-density lipoprotein; CHOL: Cholesterol; TG: Triglyceride; HDL: High-density lipoprotein; Con: Control; Exe: Exercise; Exe + GC: Exercise + *Garcinia cambogia*; GC: *Garcinia cambogia*. $P < 0.05$.

Table 5. Changes in ALT, AST, and Fasting Blood Glucose Following the Interventions: Descriptive Statistics and Within-Group and Between-Group Comparisons (Mean $\pm$ SD)

Variable	Group	Pretest (Mean $\pm$ SD)	Posttest (Mean $\pm$ SD)	Change (%)	95% CI for Change	P (Within Con)	P (Within Exe)	P (within Exe + GC)	P (Within GC)	P (Between Pre)	P (Between Post)
ALT (U/L)	Con	25.4 $\pm$ 3.2	24.8 $\pm$ 3.0	-2.4	[-6.2, -1.8]	0.712	—	—	—	0.712	0.050*
	Exe	26.1 $\pm$ 3.4	22.3 $\pm$ 2.6	-14.6		—	0.001*	—	—		
	Exe + GC	27.0 $\pm$ 3.5	21.5 $\pm$ 2.5	-20.4		—	—	0.001*	—		
	GC	25.8 $\pm$ 3.3	23.9 $\pm$ 2.8	-7.4		—	—	—	0.022*		
FBS (mg/dL)	Con	151.7 $\pm$ 5.6	149.5 $\pm$ 10.7	-1.45	[-22.5, -10.3]	0.689	—	—	—	0.689	0.001*
	Exe	151.6 $\pm$ 6.6	136.3 $\pm$ 6.2	-10.09		—	0.001*	—	—		
	Exe + GC	158.6 $\pm$ 11.3	142.4 $\pm$ 16.1	-10.21		—	—	0.004*	—		
	GC	159.1 $\pm$ 0.9	146.8 $\pm$ 8.7	-7.73		—	—	—	0.031*		
AST (U/L)	Con	21.2 $\pm$ 2.2	20.7 $\pm$ 2.1	-2.59	[-6.8, -0.6]	0.734	—	—	—	0.734	0.050*
	Exe	22.0 $\pm$ 2.8	18.8 $\pm$ 1.9	-14.55		—	0.001*	—	—		
	Exe + GC	22.8 $\pm$ 3.6	19.1 $\pm$ 2.7	-16.23		—	—	0.001*	—		
	GC	24.2 $\pm$ 2.2	22.7 $\pm$ 3.4	-6.20		—	—	—	0.031*		

Note. CI: Confidence interval; SD: Standard deviation; ALT: Alanine aminotransferase; FBS: Fasting blood sugar; AST: Aspartate aminotransferase; Con: Control; Exe: Exercise; Exe + GC: Exercise + *Garcinia cambogia*; GC: *Garcinia cambogia*. $P < 0.05$.

our findings (18, 19). In contrast, Amirkhani et al found that TG levels significantly decreased after eight weeks of combined exercise and curcumin, but no notable changes were observed in VLDL-C, HDL-C, LDL-C, or TC (20).

The hypoglycemic effects of AE and GC supplementation may partially result from reductions in serum free fatty acids, likely due to increased oxidation and utilization of these substrates (21, 22). GC is known to suppress appetite and inhibit lipogenesis, thereby contributing to weight loss without stimulating the central nervous system (23).

Oral administration reduces food intake and inhibits de novo fatty acid synthesis in the liver and adipose tissue, resulting in decreased fat accumulation in animal models (24). Nonetheless, Brandt et al concluded that long-term HCA treatment increased hepatic de novo lipogenesis and liver fat content in rats, despite reductions in body weight (25), and that HCA inhibits lipogenesis when cytoplasmic acetyl-CoA is available (26). Likewise, HCA may inhibit ATP-citrate lyase, thus limiting fatty acid and cholesterol synthesis, which is likely due to the activation of multiple

metabolic pathways influenced by both HCA and AE (27). Possible underlying mechanisms include enhanced insulin sensitivity, stimulation of muscle glucose uptake, activation of glucose transporter type 4 transporters, and inhibition of lipase activity, collectively improving carbohydrate metabolism (28).

AE enhances lipoprotein metabolism by increasing LPL activity, promoting VLDL catabolism and HDL synthesis, as well as facilitating reverse cholesterol transport (29). The effects of exercise on lipid metabolism and the metabolic properties of GC have been confirmed by Manore et al (30).

Moreover, AE and GC supplementation may reduce AST and ALT by improving lipid metabolism and liver function. More precisely, these interventions decrease oxidative stress and inflammation, enhance liver cell function and LPL activity, and facilitate tissue repair (31, 32). In addition, AE improves insulin sensitivity and increases fat oxidation capacity, and GC inhibits lipogenesis pathways, thereby enhancing metabolic status and liver function.

The decrease in blood sugar may be due to increased glucose transporter type 4 expression, enhanced insulin receptor signaling, increased glycogen synthase and hexokinase activity, muscle tissue adaptations (increased capillaries and mitochondria), improved glucose uptake, better serum and muscle free fatty acid utilization, and changes in adipokines, such as adiponectin and leptin (33, 34). These findings align with the results of Shenoy et al (35), but contrast with those of Yavari et al, possibly due to differences in participant characteristics and exercise intensity (36).

The increase in VO_{2peak} in the exercise and combined groups may result from increased plasma volume, reduced hematocrit, improved cardiac stroke volume, enhanced muscle mitochondrial density, angiogenesis, capillary density, myoglobin content, fat oxidation, decreased glycolysis, and elevated Krebs cycle and electron transport enzyme activity (37, 38). Furthermore, the increase in VO_{2peak} in beginners and those with class I obesity and low initial fitness levels may be due to central and peripheral adaptations. Central adaptations include increased cardiac stroke volume, improved cardiac output, and increased cardiovascular efficiency. Similarly, peripheral adaptations encompass increased skeletal muscle capillary density, improved mitochondrial oxidative capacity, and increased aerobic enzyme activity, which all contribute to increased oxygen consumption. However, these findings should be interpreted with the limitations of the indirect measurement method in mind. GC may enhance these effects by improving fat metabolism, reducing body fat, and optimizing energy utilization, supporting more efficient oxygen uptake.

The lack of changes in leg explosive power (vertical jump and Sargent test) can be explained by the nature of aerobic training and GC, which primarily enhances cardiovascular endurance and fat metabolism rather than

fast-twitch muscle fiber performance (39, 40). Ayán-Pérez et al reported similar findings (41).

Resistance and plyometric training have greater impacts on jump performance and muscle strength, which is in conformity with the results of Liu et al (42). Hand strength was not directly targeted, and GC could primarily affect weight management and metabolism rather than neuromuscular adaptations (43, 44).

Conclusion

Combining GC supplementation with AE has been shown to have a positive synergistic effect on metabolic markers and improve aerobic capacity. Accordingly, the simultaneous use of this natural supplement with a regular AE program can be recommended as an effective strategy for improving the metabolic and physical health of obese class I women.

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During the preparation of this manuscript, the authors employed ChatGPT for language editing, grammatical editing, and summarization. It should be noted that all content generated with the assistance of these tools was carefully reviewed, validated, and revised by the authors in order to ensure accuracy, originality, and scholarly rigor. Moreover, they assume full responsibility for the integrity and final content of the published articles.

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Competing Interests

The authors declare they have no conflict of interests related to this study.

Ethical Approval

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Islamic Azad University, Varamin-Pishva Branch (approval code: IR.IAU.SRB.REC.1403.002). In addition, written informed consent was obtained from all participants before their inclusion in the study. This trial was also registered with the Iranian Registry of Clinical Trials under registration number IRCT20171210037809N9 (registration date: 2025-05-16, 1404/02/26).

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