Physical Education Theory and Methodology
Теорія та методика фізичного виховання

ISSN 1993-7989 (print)
ISSN 1993-7997 (online)
ISSN-L 1993-7989

Original Scientific Article

Combination Training Improves Leptin Regulation, Reduces Body Fat, and Increases Skeletal Muscle Mass in Obese Females

Dwi Cahyo Kartiko[1ABDE], Purwo Sri Rejeki[2ABDE], Dono Indarto[3ABDE], Irmantara Subagio[1BD], Sasminta Christina Yuli Hartati[1BD], Bekir Erhan Orhan[4CD], Yuni Nurwati[1BD] and Adi Pranoto[1ABCD]

[1]Universitas Negeri Surabaya
[2]Universitas Airlangga
[3]Universitas Sebelas Maret
[4]Istanbul Aydın University

Authors' Contribution: A – Study design; B – Data collection; C – Statistical analysis; D – Manuscript Preparation; E – Funds Collection

Corresponding Author: Dwi Cahyo Kartiko, e-mail: dwicahyo@unesa.ac.id
Accepted for Publication: February 13, 2026
Published: March 30, 2026

DOI: 10.17309/tmfv.2026.2.16

Abstract

Background. Leptin dysregulation and unfavorable body composition are central features of obesity in women. Although combined aerobic and resistance training is commonly prescribed to improve adiposity and muscle mass, evidence integrating changes in serum leptin concentration with detailed body composition outcomes in this population remains limited.

Objectives. The present randomized controlled trial aimed to examine the effects of an eight-week combined training intervention on leptin concentration, fat mass, and skeletal muscle mass in obese women.

Materials and Methods. Thirty obese women were allocated to either a combined training group or a non-exercising control group. The intervention consisted of moderate-intensity aerobic and resistance exercise performed five times per week for eight weeks. Body composition included body fat percentages (BFP), fat mass (FM), Free Fat Mass (FFM) and skeletal muscle mass (SMM) was assessed using bioelectrical impedance analysis, and serum leptin concentration was determined by enzyme-linked immunosorbent assay. Pre–post and between–group differences were analyzed using parametric statistical tests (independent sample t test and paired t test) with a significance threshold of $p < 0.05$.

Results. Participants who completed the combined training program exhibited significant reductions in serum leptin concentration and adiposity indices (specifically BFP and FM), alongside significant increases in FFM and SMM, compared to the control group ($p < 0.05$).

Conclusions. Using short-term moderate-intensity combined aerobic and resistance training was associated with favorable changes in leptin concentration. These findings support the potential role of structured combination training as an exercise-based approach for improving metabolic and musculoskeletal profiles within the limits of the present study.

Keywords: body composition, combination training, leptin, muscle mass, obesity.

Introduction

Obesity remains a major public health challenge worldwide, with a rapidly increasing prevalence among adult populations, particularly women, and is closely linked to a broad range of metabolic and cardiovascular complications (GBD 2021 Adult BMI Collaborators, 2025; Martin et al., 2025). According to data from Survei Kesehatan Indonesia (SKI) 2023, the prevalence of obesity in Indonesian adults (aged 18 years and older) rose from 21.8% in 2018 to 23.4% in 2023 (Kepmenkes 2025). Beyond excessive fat accumulation, obesity is characterized by dysregulation of adipose tissue function, including changes in the secretion of adipokines like leptin, which are essential for controlling hunger, energy balance, and metabolic homeostasis (Sakers et al., 2022; Klein et al., 2022). In individuals with obesity, chronically elevated leptin levels are often accompanied by impaired leptin signaling, contributing to persistent energy imbalance and metabolic dysfunction (Lee et al., 2022; Fahed et al., 2022). At the same time, obesity is frequently associated with reduced skeletal muscle mass and function, a condition increasingly recognized as sarcopenic obesity, which further exacerbates metabolic risk and limits functional capacity, particularly in women (Donini et al., 2022; Petermann-Rocha et al., 2022; Yuan & Larsson, 2023). Although clinical and public health guidelines consistently emphasize physical activity as a cornerstone of obesity management (Rock et al., 2022; American Diabetes Association Professional Practice Committee, 2025), there remains a gap between theoretical recommendations and empirical evidence regarding how specific exercise modalities simultaneously influence leptin regulation, fat mass reduction, and muscle mass preservation in obese female populations.

Combined aerobic and resistance training has been proposed as an effective exercise strategy to address the multifaceted nature of obesity by targeting both adiposity and muscle tissue. Previous intervention studies and systematic reviews have shown that combination training can improve body composition, reduce body fat, and favorably modify adipokine profiles in individuals with overweight and obesity (Rejeki et al., 2023; Oh et al., 2023; Sugiharto et al., 2024; Hejazi et al., 2023; Tan et al., 2024). However, existing evidence remains heterogeneous with respect to training protocols, participant characteristics, and outcome measures, and relatively few randomized controlled trials have focused specifically on obese women while concurrently assessing leptin levels alongside detailed body composition indicators. Moreover, many studies emphasize weight loss or inflammatory outcomes without adequately addressing the combined dynamics of leptin regulation, fat mass reduction, and skeletal muscle mass accretion within a single intervention framework (González-Jurado et al., 2020; Gil-Cosano et al., 2024; Hintze et al., 2021). As a result, the specific contribution of moderate-intensity combination training to integrated metabolic and musculoskeletal adaptations in obese females remains insufficiently clarified.

From a theoretical perspective, combining aerobic and resistance exercise offers a complementary stimulus that may promote favorable changes in energy balance while preserving or increasing muscle mass, thereby addressing key physiological challenges associated with obesity. Rather than focusing on isolated outcomes, an integrated approach that simultaneously evaluates leptin concentrations, body fat, and skeletal muscle mass may provide a more comprehensive understanding of exercise-induced adaptations relevant to obesity management.

Various previous intervention studies have shown that physical exercise, both aerobic and resistance training, can reduce circulating leptin levels (González-Jurado et al., 2020; Hintze et al., 2021; Li et al.,2022; Gil-Cosano et al., 2024; Sugiharto et al., 2024). However, most of these studies only assessed the effects of exercise on leptin regulation and body composition changes separately, without evaluating the physiological adaptations of both simultaneously in an integrated exercise program. Furthermore, these studies used diverse populations ranging from children to obese adults so that evidence in the Indonesian population, especially in obese women is still limited. To the best our knowledge, research related to the combination of aerobic and resistance training is still limited. One of the studies that identified related topic in Indonesia was by Rejeki et al. (2023) which showed that 4 weeks of combined training improved body composition and adipokine levels in obese women. Accordingly, this randomized controlled trial investigated whether an eight-week moderate-intensity aerobic–resistance exercise regimen could modulate serum leptin levels alongside changes in adiposity and lean tissue indices in women living with obesity. We hypothesized that combination training would lead to a significant reduction in leptin concentration and body fat, accompanied by an increase in skeletal muscle mass, compared with a non-exercising control group.

Materials and Methods

Study Design

This study used a pretest–posttest control-group study and a true experimental design, with participants being obese adults females. Purposive sampling was used to choose participants based on the inclusion criteria as follows: BMI of 28 kg/m² or higher, sedentary lifestyle for at least 6 months, non-smoker, and no history of metabolic, cardiovascular, endocrine, or orthopedic disorders. Before being enrolled as participants, eligible individuals gave written informed permission. The intervention was conducted over an 8-week period. This study was approved by the Health Research Ethics Committee, Faculty of Medicine, Universitas Airlangga (Approval No. 203/EC/KEPK/FKUA/2025).

Sample Size and Power Calculation

G*Power was used to determine the sample size (Version 3.1.9.7). A paired mean difference model was selected based on expected pre–post change in leptin and body composition parameters. The minimum sample size required was 27 people with a medium effect size (Cohen's d = 0.50), α = 0.05, and statistical power ($1-\beta$) = 0.80. Accounting for an estimated 10–15% attrition, a total of 30 subjects were targeted for enrollment.

Randomization and Allocation

After baseline assessments were peformed, individuals were randomized using computer-generated basic randomization sequence to either the intervention or control group. To ensure allocation concealment, sealed opaque envelopes were prepared by in indepencent researcher who was not involved in testing or analysis. Moreover, those assessing outcome and laboratory staff were blinded to the group allocations.

Training Protocol

The intervention group performed combination training five sessions per week. Each session began with moderate intensity aerobic activity on a motorized treadmill (60-70% HRmax) followed by resistance training with Cybex fitness equipment that targeted the main muscle groups in the upper and lower bodies. The resistance was set at about 60-70% of their one-repetition maximum, two to 4-6 sets per exercise, 10-12 repetitions per set, with 60-90 seconds of rest between sets. The average duration of each session was around 60 minutes, and their exercise intensity was monitored using heart rate. Participants in the control group, on the other hand, received instructions to carry on with their regular activities without adding any organized exercise.

Diet and Physical Activity Control

Before participated in the intervention programs, the researcher thoroughly explained about the recommended diet during the research. The recommendation diet was based on the balanced nutritional guidelines, including limiting the consumption of foods high in sugar, salt, and fat. To avoid contamination effects, participants were prohibited from engaging in structured exercise outside the study protocol. Light daily activity was tracked using smartphone-based pedometer step counts and weekly activity checklists to verify compliance.

Biochemical and Body Composition Analysis

Blood samples (10-12 hours after fasting) were collected at baseline and after intervention. Serum leptin concentration was analyzed using enzyme-linked immunosorbent assay (ELISA) in accordance with manufacturer instructions Human LEP (Leptin) ELISA Kit (Cat.No.: E-EL-H6017; Elabscience, Inc. USA) with sensitivity = 9.38 pg/mL; detection range = 15.63-1000 pg/mL; inter-assay variability =< 10%. Body composition variables including body fat percentage, fat mass (FM), free fat mass (FFM), and skeletal muscle mass were assessed using TANITA DC-360 bioelectrical impedance analysis (TANITA Corp. Inc., ArlingtonHeights, IL, USA).

Statistical Analysis

By using the Shapiro-Wilk test, the normality of the data distribution was evaluated. The normality of data distribution was assessed using the Shapiro–Wilk test. We used independent sample t-test and to assess differences within group, and to evaluate differences within the group, we used paired sample t-test. Correlation between changes in leptin concentration and body composition were also analyzed using the Pearson's correlation. Additionally, statistical significance was set at $p < 0.05$. Effect size (ES) estimates were calculated using Cohen's to quantify the magnitude of observed effects. According to Cohen's classification, effect sizes (Cohen's d) were interpreted as small (d = 0.2), medium (d = 0.5), large (d = 0.8), and very large (d ≥ 1.3) to evaluate the magnitude of changes following the exercise intervention (Sullivan & Feinn, 2012).

Results

Participant Flow and Baseline Characteristics

Thirty people in all were recruited and randomly assigned into training group and control group with each group consists of 15 participants. All participants completed the 8-week intervention and post-testing (attrition = 0%). Age, blood pressure, resting heart rate, oxygen saturation, body temperature, fasting blood glucose, hemoglobin, height, weight, and body mass index were not significantly difference between the groups at baseline. It shows that the charactericties of participants in the training and control group is similar ($p > 0.05$) (Table 1).

Table 1. Baseline Characteristics of Participants

Characteristics	Control (n = 15)	Training (n = 15)	p-value
Age (yrs)	25.53 ± 1.85	26.33 ± 1.92	0.254
SBP (mmHg)	116.53 ± 3.16	116.07 ± 3.06	0.684
DBP (mmHg)	72.87 ± 5.14	72.93 ± 4.27	0.969
RHR (bpm)	75.93 ± 4.51	74.53 ± 2.45	0.302
SpO2 (%)	97.73 ± 0.88	98.27 ± 0.89	0.110
BT (°C)	36.41 ± 0.31	36.31 ± 0.27	0.316
FBG (mg/dL)	97.53 ± 7.59	98.20 ± 4.69	0.775
Hb (g/dL)	14.14 ± 1.48	13.97 ± 1.35	0.749
Height (m)	1.55 ± 0.07	1.56 ± 0.06	0.788
Weight (kg)	76.14 ± 7.57	77.99 ± 9.95	0.572
BMI (kg/m^2)	31.70 ± 2.75	32.20 ± 3.34	0.659

Note: SBP: Systolic blood pressure, DBP: Diastolic blood pressure, RHR: Resting heart rate, SpO2: Oxygen saturation, BT: Body temperature, FBG: Fasting blood glucose, Hb: Hemoglobin, BMI: Body mass index. All data are presented as mean ± SD. The p-value was obtained using the independent samples t-test.

Effects on Serum Leptin

In comparison to baseline, the training group showed a significantly decrease in leptin concentration after eight week intervention. While there was no significant change in leptin level of the control group (p=0.399), the decreasing of leptin level in training group after intervention showed significantly reduction (p=0.001; ES: 2.163) (Figure 1).

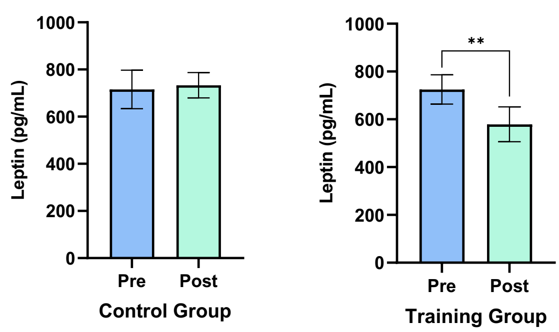

Fig. 1. Changes in Leptin Concentrations After an 8-Week Intervention. Note: (**) Significant at pre ($p < 0.001$). The p-value was obtained using the paired samples t-test

Effects on Body Composition

Based on the Figure 2, it can be seen that in the intervention group, combination training led to increase free fat mass (FFM) and decrease body fat percentage. Within-group improvements were statistically significant (body fat percentage p = 0.001, effect size (ES) = 0.683; fat mass p = 0.001, ES = 0.663; free fat mass p = 0.001, ES = 0.645; skeletal

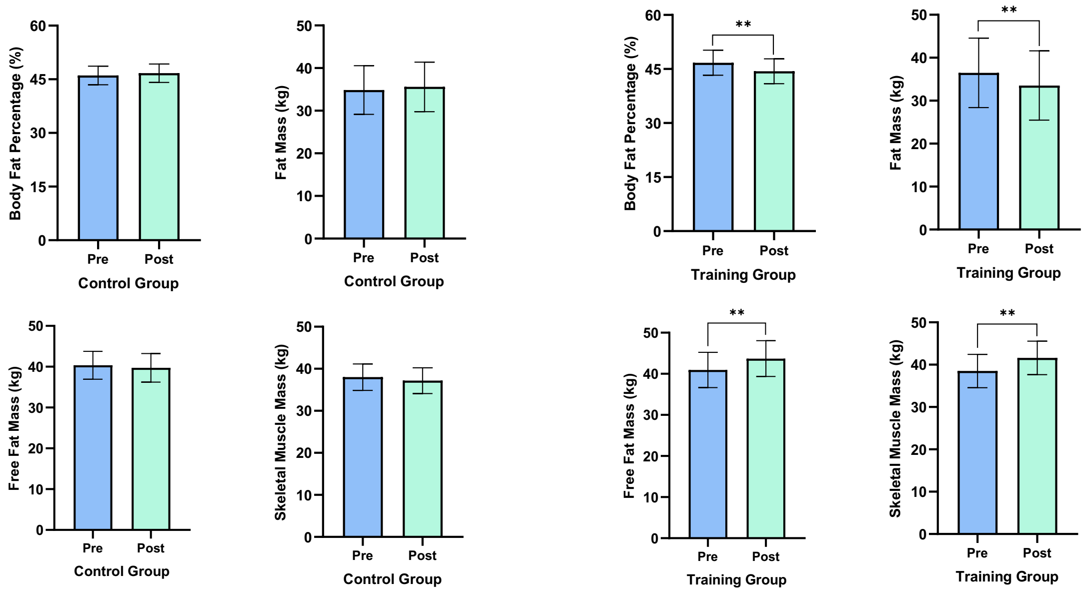

Fig. 2. Changes in Body Composition After an 8-Week Intervention.
Note: (**) Significant at pre (p < 0.001). All data are presented as mean ± standard deviation (SD). The p-value was obtained using the paired samples t-test

muscle mass p = 0.001, ES = 0.785), whereas the control group did not exhibit significant changes (p > 0.05).

Between-Group Differences

Between-group comparisons confirmed greater improvements in leptin and body composition in the training group compared to control group (all key outcomes p < 0.05) (Table 2). Table 3 showed the association between changes in leptin levels and the body composition. As shown in Table 2, the training program significantly reduced leptin level by -19.27±14.24% (95%CI: 13.28-31.91, p<0.001, ES: 1.814). Moreover, combined training also significantly reduced the body composition namely BFP by -5.01±3.81% (95%CI: 4.31-8.52, p<0.001, ES: 2.283), and FM by -8.36±2.09% (95%CI, 9.34-11.72, p<0.001, ES: 2.535), alongside significant

Table 2. Between-Group Differences in Leptin and Body Composition Improvements

Parameters	Control (n = 15)	Training (n = 15)	95% CI		p-value	ES
			Lower	Upper		
Pre-Leptin (pg/mL)	715.09 ± 81.66	724.91 ± 61.28	-63.81	44.19	0.713	0.135
Post-Leptin (pg/mL)	732.99 ± 53.79	579.11 ± 72.99††	105.92	201.83	0.001	2.400
Δ-Leptin (pg/mL)	17.89 ± 79.69	-145.79 ± 110.48††	91.64	235.73	0.001	1.699
Change-Leptin (%)	3.33 ± 10.36	-19.27 ± 14.24††	13.28	31.91	0.001	1.814
Pre-BFP (%)	46.06 ± 2.59	46.75 ± 3.46	-2.98	1.61	0.544	0.224
Post-BFP (%)	46.70 ± 2.55	44.39 ± 3.45†	0.05	4.58	0.047	0.763
Δ-BFP (%)	0.64 ± 0.49	-2.36 ± 1.70††	2.06	3.94	0.001	2.396
Change-BFP (%)	1.41 ± 1.11	-5.01 ± 3.81††	4.31	8.52	0.001	2.283
Pre-FM (kg)	34.84 ± 5.71	36.47 ± 8.09	-6.86	3.61	0.530	0.232
Post-FM (kg)	35.59 ± 5.82	33.53 ± 8.07	-3.19	7.32	0.429	0.292
Δ-FM (kg)	0.75 ± 0.31	-2.93 ± 0.49††	3.38	3.99	0.001	1.871
Change-FM (%)	2.17 ± 0.84	-8.36 ± 2.09††	9.34	11.72	0.001	2.535
Pre-FFM (kg)	40.37 ± 3.42	40.92 ± 4.29	-3.46	2.35	0.699	0.142
Post-FFM (kg)	39.73 ± 3.52	43.71 ± 4.35†	-6.94	-1.03	0.010	1.007
Δ-FFM (kg)	-0.64 ± 0.46	2.79 ± 0.35††	-3.73	-3.12	0.001	1.456
Change-FFM (%)	-1.61 ± 1.18	6.87 ± 1.09††	-9.33	-7.63	0.001	2.186
Pre-SMM (kg)	38.01 ± 3.14	38.51 ± 3.94	-3.16	2.16	0.704	0.141
Post-SMM (kg)	37.17 ± 3.08	41.61 ± 3.95††	-7.09	-1.79	0.001	1.253
Δ-SMM (kg)	-0.84 ± 0.37	3.10 ± 0.34††	-4.21	-3.68	0.001	1.648
Change-SMM (%)	-2.21 ± 0.95	7.51 ± 0.99††	-10.44	-8.99	0.001	2.231

Note: (†) Significant at control group (p < 0.05). (††) Significant at control group (p < 0.001). ES: Effect Size. Δ: Delta (Post - Pre). All data are presented as mean ± standard deviation (SD). The p-value was obtained using the independent samples t-test.

increases in FFM by 6.87±1.09% (95%CI: -9.33-(-7.63), p<0.001, ES: 2.186) and skeletal muscle mass by 7.51±0.99% (95%CI: -10.44-(-8.99), p<0.001, ES: 2.231), compared with the control group ($p < 0.05$).

On the other hand, correlation analysis showed that changes in leptin concentration before and after training program significantly associated with changes in all body composition parameters namely BFP (r:0.554), FM (r: 0.659), FFM (r: -0.661), and SMM (r: -0.601) (Table 3). The r value of BFP, FM, and FFM showed positive value and for SMM showed negative value. It means that the decrease in leptin concentration significantly inline with reduction of the BFM, FM, and FFM; meanwhile, reduction in leptin concentration significantly increase the SMM.

Table 3. Shows the correlations between leptin concentrations and body fat and skeletal muscle mass.

Parameter	Δ-Leptin (pg/mL)	
	r	p-value
Δ-BFP (%)	0.554	0.001
Δ-FM (kg)	0.659	0.001
Δ-FFM (kg)	-0.661	0.001
Δ-SMM (kg)	-0.601	0.001

Note: BFP: body fat percentage, FM: fat mass, FFM: fat free mass, SMM: skeletal muscle mass. The p-value was obtained using the Pearson's correlation test.

Discussion

This study evaluated changes in leptin levels, body composition, and skeletal muscle mass in obese women with ages ranging from 25.53 ± 1.85 in the control group and 26.33 ± 1.92 in the intervention group. In the present study, we observed that an eight-week moderate-intensity combined aerobic–resistance training program significantly reduced serum leptin concentrations by almost 19.5% (-19.27±14.24%, 95%CI: 13.28-31.91, p<0.001, ES: 1.814), reduced body fat percentage by 5% (-5.01±3.81% , 95%CI: 4.31-8.52, p<0.001, ES: 2.283), and FM by 8% (-8.36±2.09%, 95%CI, 9.34-11.72, p<0.001, ES: 2.535), while increasing FFM by 6.87±1.09% (95%CI: -9.33-(-7.63), p<0.001, ES: 2.186) and SMM by 7.51±0.99% (95%CI: -10.44-(-8.99), p<0.001, ES: 2.231), in obese women compared with the control group (Table 2). These findings are consistent with previous randomized and controlled studies reporting favorable effects of concurrent training on body composition and adipokine profiles in obese populations (Rejeki et al., 2023; Oh et al., 2023; Sugiharto et al., 2024). Providing aerobic and resistance trainingfor 8 weeks with a frequency 5 times a week for 60 minutes in this study also can improve leptin levels and body composition. Leptin levels before intervention in both groups were not significantly different. After intervention program, there was a significant difference in leptin levels with the training group showing lower leptin levels than the control group. Changes in leptin concentration also significantly associated with body composition (Table 3).

Obesity is characterized by the body mass index (BMI) ≥25kg/m2 by criteria from the World Health Organization (WHO). Obese individuals have an excessive accumulation of adipose tissue, which functions as an endocrine organ through the secretion of adipokines, which play a role in regulating food intake, lipid and glucose metabolism, insulin action, and energy balance is shown in obesity people. Adipokine dysfunction contributes to the development of obesity, primarily through impaired regulation of leptin and adiponectin, key regulators of metabolic homeostasis (Park & Shimokawa 2024). Leptin, a product of the obesity gene, plays a role in weight control by regulating appetite and energy expenditure and also influences neuroendocrine function (Landecho et al., 2019). In obesity, hyperleptinemia occurs due to leptin resistance triggered by excessive energy intake and low physical activity, resulting in persistently high circulating leptin levels. This condition not only refrects metabolic disturbances but also plays a role in the pathogenesis of cardiometabolic complications, inflammation, and malignancy. Therefore, intervention like combination program that can reduce hyperleptinemia have the potential to reduce obesity-related morbidity (Rostas et al., 2017). Combination training in this study significantly reduced leptin levels in training groups. It has been suggested that exercise might increase energy expenditure and inhibit leptin secretion (Murawska-Ciałowicz et al., 2022).

Leptin, glucagon-like peptide 1 (GLP-1), ghrelin, peptide YY (PYY), glucose dependent insulinotropric polypeptide (GIP), and cholecystokinin (CCK) are among the hormones that regulate appetite and satiety behaviors. All the mentioned hormones above are released and produced by intestine and adipocytes which interact with hypothalamus to regulate eating and satiety behaviors (Zouhal et al., 2019). Exercise inhibits the production of ghrelin via attaching to the its receptor namely G-protein coupled receptor 81 (GPCR81) in gastric cell (Vanderheyden et al., 2020). On the other hand, exercise also can lower leptin levels and increase leptin sensitivity in the brain. After doing exercise, the hypothalamusbecomes more sensitive to leptin, which improves its capability to control hunger and energy balance. It also reduces inflammation, increases insulin sensitivity, and stimulates neurogenesis and synaptic plasticity (Augusto-Oliveira et al., 2023; de Assis et al., 2023). Leptin starts to initiate signaling pathways after binding to its hypothalamic receptor, long isoform receptor (ObRb) with the most notable being the JAK2/STAT3 pathway, which leads to the transcription of genes such as POMC, suppressor of cytokine signaling 3 (SOCS3), and brain-derived neurotrophic factor (BDNF) that promote satiety and reduce food intake (Kwon et al., 2016; Liu et al., 2021).

In this study, body composition such as FM, FFM, and SMM significantly improved by intervention of combination training. Exercise activates the cardiovascular, respiratory, endocrine, and metabolic systems, triggering key signaling pathways such as adenosine monophosphate (AMP)-activated protein kinase (AMPK), calcium/calmodulin-dependent protein kinase (CaMK), protein kinase A (PKA), protein kinase C (PKC), and mammalian target of rapamycin (mTOR) (Hoffman et al., 2015). Specifically, AMPK activates peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α) to sense energy defisit and promote mitochondrial biogenesis (Hoffman et al., 2015; Gowans et al., 2013). Another crucial kinase, namely CaMK-II promotes lipid oxidation and increases glucose absorption via glucose transporter-4 (GLUT-4), supports lipid oxidation

(Yong & Song 2024). Resistance training typically activates the phosphaidylinositol 3-kinase (PI3K)- protein kinase B(Akt)-mTOR (PAM) signaling pathway, which regulates synthesis and degradation of protein, resulting in higher lean body mass and muscle hypertrophy (Plaza-Diaz et al., 2022). Aerobic exercise, on the other hand, mainly stimulates the AMPK-MAPK-PGC-1 α pathway, which leads to increased mitochondrial biogenesis, improved oxidative metabolism, a change in fiber phenotype from rapid glycolytic to more oxidative, and angiogenesis (Ashcroft et al., 2024). The physiological changes that affect body composition are highlighted by these molecular process. Together, aerobic and resistance training can contributeto long-term improvements not only body composition but also health and performance.

Comparable benefits have been reported across pediatric, adolescent, and adult populations presenting with excess body weight, although the extent of these adaptations appears to depend on exercise modality, training dose, and individual characteristics (González-Jurado et al., 2020; Gil-Cosano et al., 2024; Hintze et al., 2021; Headid & Park, 2021). In addition, evidence from broader exercise interventions suggests that combination training is consistently associated with favorable adaptations in muscle mass and functional capacity across different age groups (Wu et al., 2021). Taken together, the present results reinforce the notion that combination training is an effective exercise modality for simultaneously targeting excess adiposity and muscle mass deficits, which are central features of obesity in women.

Although the present study did not directly assess molecular or inflammatory pathways, the observed reductions in leptin levels and fat mass, alongside increases in skeletal muscle mass, may be explained by previously described exercise-induced adaptations. Prior studies suggest that combined aerobic and resistance training can modulate adipokine secretion, reduce low-grade inflammation, and enhance metabolic flexibility through coordinated adaptations in adipose and skeletal muscle tissue (Makarewicz et al., 2022; Sanchis et al., 2024). Exercise-induced myokines and exerkines have also been proposed to facilitate crosstalk between muscle and adipose tissue, thereby influencing leptin sensitivity and energy balance (Chow et al., 2022). In addition, changes in endocrine mediators such as insulin-like growth factor-1 and fibroblast growth factor-21 have been reported following concurrent training interventions, which may support muscle hypertrophy and lipid metabolism (Colleluori et al., 2025; Fiorenza et al., 2024). Broader systemic hormonal and metabolic adaptations to regular exercise, including age- and lifestyle-related endocrine responses, have also been suggested to contribute to favorable changes in body composition, with exercise intensity and prescription playing an important role in shaping these responses (Pataky et al., 2021; Hansen et al., 2022). These mechanisms should be interpreted as plausible explanations rather than direct causal pathways, given that they were not directly measured in the present study.

From a practical perspective, the present findings support the application of moderate-intensity combination training as a non-pharmacological strategy for improving metabolic and musculoskeletal health in obese women. Compared with single-modality exercise, concurrent training offers a time-efficient approach that addresses both fat reduction and muscle preservation, which is particularly relevant for female populations at risk of sarcopenic obesity (Donini et al., 2022). The benefits of combined exercise modalities have also been demonstrated in other clinical and functional contexts, including improvements in muscle strength, balance, and overall functional performance (Sadeghi et al., 2021), as well as in structured rehabilitation settings following disease-related deconditioning (Jimeno-Almazán et al., 2022). The main contribution of this study lies in its integrated evaluation of leptin regulation, detailed body composition parameters, and skeletal muscle mass within a randomized controlled design. By focusing on obese women and employing a structured training protocol, this study extends previous evidence and provides novel empirical support for the role of combination training in comprehensive obesity management.

It is important to recognize that there were several limitations of this present findings. First, the results' generalizability may be limited by the comparatively small sample size and brief intervention period. Second, bioelectrical impedance analysis was used to measure body composition instead of imaging-based techniques, which may introduce measurement variability. Third, dietary intake was monitored but not strictly controlled, and additional biomarkers related to inflammation, insulin sensitivity, or muscle metabolism were not assessed. These limitations should be considered when extrapolating the findings beyond the study context.

Further research should prioritize the inclusion of more heterogeneous cohorts, prolonged training durations, and multidimensional assessments of metabolic, inflammatory, and hormonal pathways to better elucidate the biological processes underlying exercise-induced alterations in leptin regulation and body composition. The integration of molecular, transcriptomic, or gut microbiota analyses may further clarify the pathways through which combination training exerts its effects (Pillon et al., 2020; Quiroga et al., 2020; Brunelli et al., 2024; Estébanez et al., 2023). Additionally, comparative trials examining exercise training alongside pharmacological interventions for obesity may help to define optimal multimodal treatment strategies (Melson et al., 2025; Jastreboff et al., 2025).

Notwithstanding these limitations, the present results suggest that combined aerobic–resistance exercise promotes favorable short-term changes in leptin homeostasis alongside improvements in body composition profiles among women with obesity. Beyond metabolic outcomes, previous research indicates that regular exercise—particularly when combining aerobic and resistance components—can also support broader systemic and neurophysiological health benefits, including cognitive and neurotrophic adaptations (Ruiz-González et al., 2021; Kao et al., 2025; Dhahbi et al., 2025). The findings align with broader evidence supporting exercise as a cornerstone of obesity management and metabolic disease prevention (Rahmati et al., 2025; Batrakoulis et al., 2022). Given the increasing burden of obesity-related comorbidities, including cardiovascular and hepatic complications (Packer et al., 2025; Younossi et al., 2025; European Association for the Study of the Liver, 2025), the present results justify the inclusion of structured combination training programs as part of evidence-based lifestyle interventions.

Conclusion

The findings of this randomized controlled study indicate that eight weeks of moderate-intensity combined aerobic–resistance training elicit favorable changes in leptin levels alongside improvements in body composition profiles in women living with obesity. Compared with a non-exercising control group, participants who completed the combination training intervention exhibited reduced leptin levels and adiposity alongside increased muscle mass, indicating favorable short-term adaptations in metabolic and musculoskeletal health. Taken together, the present evidence underscores the relevance of combined aerobic–resistance exercise as a non-pharmacological approach to addressing fundamental physiological aspects of obesity, especially in female populations at risk of unfavorable body composition profiles. However, the results should be interpreted within the context of the study's scope, including the relatively small sample size, short intervention duration, and the use of indirect body composition assessment methods. Overall, the present study provides evidence that moderate-intensity combined aerobic–resistance training may contribute to improved leptin regulation and body composition in obese women. While the findings strengthen existing evidence on the benefits of concurrent exercise, further large-scale and long-term studies incorporating comprehensive metabolic assessments are warranted to confirm these effects and clarify their underlying mechanisms.

Conflict of Interest

The authors declare that they have no conflicts of interest.

Funding

This research was funded by Universitas Negeri Surabaya under the Indonesian Collaborative Research (RKI) scheme based on contract number 622/UN38/HK/2025 and B/52736/UN38.III.1/LK.04.00/2025.

Data Availability Statement

Data are available from the corresponding author upon reasonable request (dwicahyo@unesa.ac.id)

AI Transparency Statement

The authors used AI-assisted tools for language editing only and take full responsibility for the content.

References

GBD 2021 Adult BMI Collaborators (2025). Global, regional, and national prevalence of adult overweight and obesity, 1990-2021, with forecasts to 2050: a forecasting study for the Global Burden of Disease Study 2021. *Lancet (London, England), 405*(10481), 813-838. https://doi.org/10.1016/S0140-6736(25)00355-1

Martin, S.S., Aday, A.W., Allen, N.B., Almarzooq, Z.I., Anderson, C.A. M., Arora, P., Avery, C.L., Baker-Smith, C.M., Bansal, N., Beaton, A.Z., Commodore-Mensah, Y., Currie, M.E., Elkind, M.S. V., Fan, W., Generoso, G., Gibbs, B.B., Heard, D.G., Hiremath, S., Johansen, M.C., Kazi, D.S., … American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Committee (2025). 2025 Heart Disease and Stroke Statistics: A Report of US and Global Data From the American Heart Association. *Circulation, 151*(8), e41-e660. https://doi.org/10.1161/CIR.0000000000001303

Decree of the Minister of Health (Kepmenkes). (2025). *National Guidelines for Clinical Services for the Management of Adult Obesity.* Jakarta: Minister of Health of the Republic of Indonesia.

Sakers, A., De Siqueira, M.K., Seale, P., & Villanueva, C.J. (2022). Adipose-tissue plasticity in health and disease. *Cell, 185*(3), 419-446. https://doi.org/10.1016/j.cell.2021.12.016

Klein, S., Gastaldelli, A., Yki-Järvinen, H., & Scherer, P.E. (2022). Why does obesity cause diabetes?. *Cell metabolism, 34*(1), 11-20. https://doi.org/10.1016/j.cmet.2021.12.012

Lee, S.H., Park, S.Y., & Choi, C.S. (2022). Insulin Resistance: From Mechanisms to Therapeutic Strategies. *Diabetes & metabolism journal, 46*(1), 15-37. https://doi.org/10.4093/dmj.2021.0280

Fahed, G., Aoun, L., Bou Zerdan, M., Allam, S., Bou Zerdan, M., Bouferraa, Y., & Assi, H.I. (2022). Metabolic Syndrome: Updates on Pathophysiology and Management in 2021. *International journal of molecular sciences, 23*(2), 786. https://doi.org/10.3390/ijms23020786

Donini, L.M., Busetto, L., Bischoff, S.C., Cederholm, T., Ballesteros-Pomar, M.D., Batsis, J.A., Bauer, J.M., Boirie, Y., Cruz-Jentoft, A.J., Dicker, D., Frara, S., Frühbeck, G., Genton, L., Gepner, Y., Giustina, A., Gonzalez, M.C., Han, H.S., Heymsfield, S.B., Higashiguchi, T., Laviano, A., … Barazzoni, R. (2022). Definition and Diagnostic Criteria for Sarcopenic Obesity: ESPEN and EASO Consensus Statement. *Obesity facts, 15*(3), 321-335. https://doi.org/10.1159/000521241

Petermann-Rocha, F., Balntzi, V., Gray, S.R., Lara, J., Ho, F.K., Pell, J.P., & Celis-Morales, C. (2022). Global prevalence of sarcopenia and severe sarcopenia: a systematic review and meta-analysis. *Journal of cachexia, sarcopenia and muscle, 13*(1), 86-99. https://doi.org/10.1002/jcsm.12783

Yuan, S., & Larsson, S.C. (2023). Epidemiology of sarcopenia: Prevalence, risk factors, and consequences. Metabolism: clinical and experimental, 144, 155533. https://doi.org/10.1016/j.metabol.2023.155533

Rock, C.L., Thomson, C.A., Sullivan, K.R., Howe, C.L., Kushi, L.H., Caan, B.J., Neuhouser, M.L., Bandera, E.V., Wang, Y., Robien, K., Basen-Engquist, K.M., Brown, J.C., Courneya, K.S., Crane, T.E., Garcia, D.O., Grant, B.L., Hamilton, K.K., Hartman, S.J., Kenfield, S.A., Martinez, M.E., … McCullough, M.L. (2022). American Cancer Society nutrition and physical activity guideline for cancer survivors. *CA: a cancer journal for clinicians, 72*(3), 230-262. https://doi.org/10.3322/caac.21719

American Diabetes Association Professional Practice Committee (2025). 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2025. *Diabetes care, 48*(1 Suppl 1), S27-S49. https://doi.org/10.2337/dc25-S002

Rejeki, P.S., Pranoto, A., Rahmanto, I., Izzatunnisa, N., Yosika, G.F., Hernaningsih, Y., Wungu, C.D. K., & Halim, S. (2023). The Positive Effect of Four-Week Combined

Aerobic-Resistance Training on Body Composition and Adipokine Levels in Obese Females. *Sports (Basel, Switzerland), 11*(4), 90. https://doi.org/10.3390/sports11040090

Oh, D.H., & Lee, J.K. (2023). Effect of Different Intensities of Aerobic Exercise Combined with Resistance Exercise on Body Fat, Lipid Profiles, and Adipokines in Middle-Aged Women with Obesity. *International journal of environmental research and public health, 20*(5), 3991. https://doi.org/10.3390/ijerph20053991

Sugiharto, Rejeki, P.S., Lesmana, R., Merawati, D., Susanto, H., Taufiq, A., Widyatama, F.S., Amelia, D., & Pranoto, A. (2024). Effect of combined aerobic-resistance training on regulating adipokine levels among obese females. *Comparative Exercise Physiology, 20*(3), 231-242. https://doi.org/10.1163/17552559-20231010

Hejazi, K., & Wong, A. (2023). Effects of exercise training on inflammatory and cardiometabolic health markers in overweight and obese adults: a systematic review and meta-analysis of randomized controlled trials. *The Journal of sports medicine and physical fitness, 63*(2), 345-359. https://doi.org/10.23736/S0022-4707.22.14103-4

Tan, L., Yan, W., Zhang, B., Zhao, Z., Lipowski, M., & Ossowski, Z. (2024). Comparative efficacy of different exercise types on inflammatory markers in women with overweight and obesity: A systematic review and network meta-analysis of randomized controlled trials. *Journal of science and medicine in sport, 27*(7), 458-465. https://doi.org/10.1016/j.jsams.2024.03.007

González-Jurado, J.A., Suárez-Carmona, W., López, S., & Sánchez-Oliver, A.J. (2020). Changes in Lipoinflammation Markers in People with Obesity after a Concurrent Training Program: A Comparison between Men and Women. *International journal of environmental research and public health, 17*(17), 6168. https://doi.org/10.3390/ijerph17176168

Gil-Cosano, J.J., Plaza-Florido, A., Gracia-Marco, L., Migueles, J.H., Cadenas-Sanchez, C., Olvera-Rojas, M., Ubago-Guisado, E., Labayen, I., Lucia, A., & Ortega, F.B. (2024). Effects of combined aerobic and resistance training on the inflammatory profile of children with overweight/obesity: A randomized clinical trial. *Pediatric obesity, 19*(10), e13152. https://doi.org/10.1111/ijpo.13152

Hintze, L.J., Kravchychyn, A.C. P., Ferreira, Y.A. M., Campos, R.M. D.S., Dantas, A.D. A., Masquio, D.C. L., Caranti, D.A., Thivel, D., & Dâmaso, A.R. (2021). Semi-intensive and Intensive Interdisciplinary Treatments Have Similar Effects on Metabolic Syndrome and Selected Inflammatory Markers in Adolescents with Obesity. *Journal of obesity & metabolic syndrome, 30*(4), 386-395. https://doi.org/10.7570/jomes21049

Li, C., Lyu, S., & Zhang, J. (2022). Effects of Aerobic Exercise on the Serum Leptin Level and Heart Rate Variability in the Obese Girl Children. *Computational intelligence and neuroscience,* 2022, 2298994. https://doi.org/10.1155/2022/2298994

Sullivan, G.M., & Feinn, R. (2012). Using Effect Size-or Why the P Value Is Not Enough. *Journal of graduate medical education, 4*(3), 279-282. https://doi.org/10.4300/JGME-D-12-00156.1

Park, S., & Shimokawa, I. (2024). Influence of Adipokines on Metabolic Dysfunction and Aging. *Biomedicines, 12*(4), 873. https://doi.org/10.3390/biomedicines12040873

Landecho, M.F., Tuero, C., Valentí, V., Bilbao, I., de la Higuera, M., & Frühbeck, G. (2019). Relevance of Leptin and Other Adipokines in Obesity-Associated Cardiovascular Risk. *Nutrients, 11*(11), 2664. https://doi.org/10.3390/nu11112664

Rostás, I., Pótó, L., Mátrai, P., Hegyi, P., Tenk, J., Garami, A., Illés, A., Solymár, M., Pétervári, E., Szűcs, Á., Párniczky, A., Pécsi, D., Rumbus, Z., Zsiborás, C., Füredi, N., & Balaskó, M. (2017). In middle-aged and old obese patients, training intervention reduces leptin level: A meta-analysis. *PloS one, 12*(8), e0182801. https://doi.org/10.1371/journal.pone.0182801

Murawska-Ciałowicz, E., Kaczmarek, A., Kałwa, M., & Oniszczuk, A. (2022). Influence of Training and Single Exercise on Leptin Level and Metabolism in Obese Overweight and Normal-Weight Women of Different Age. *International journal of environmental research and public health, 19*(19), 12168. https://doi.org/10.3390/ijerph191912168

Zouhal, H., Sellami, M., Saeidi, A., Slimani, M., Abbassi-Daloii, A., Khodamoradi, A., El Hage, R., Hackney, A.C., & Ben Abderrahman, A. (2019). Effect of physical exercise and training on gastrointestinal hormones in populations with different weight statuses. *Nutrition reviews, 77*(7), 455-477. https://doi.org/10.1093/nutrit/nuz005

Vanderheyden, L.W., McKie, G.L., Howe, G.J., & Hazell, T.J. (2020). Greater lactate accumulation following an acute bout of high-intensity exercise in males suppresses acylated ghrelin and appetite postexercise. *Journal of applied physiology (Bethesda, Md. : 1985), 128*(5), 1321-1328. https://doi.org/10.1152/japplphysiol.00081.2020

Augusto-Oliveira, M., Arrifano, G.P., Leal-Nazaré, C.G., Santos-Sacramento, L., Lopes-Araújo, A., Royes, L.F. F., & Crespo-Lopez, M.E. (2023). Exercise Reshapes the Brain: Molecular, Cellular, and Structural Changes Associated with Cognitive Improvements. *Molecular neurobiology, 60*(12), 6950-6974. https://doi.org/10.1007/s12035-023-03492-8

de Assis, G.G., & Murawska-Ciałowicz, E. (2023). Exercise and Weight Management: The Role of Leptin-A Systematic Review and Update of Clinical Data from 2000-2022. *Journal of clinical medicine, 12*(13), 4490. https://doi.org/10.3390/jcm12134490

Kwon, O., Kim, K.W., & Kim, M.S. (2016). Leptin signalling pathways in hypothalamic neurons. *Cellular and molecular life sciences : CMLS, 73*(7), 1457-1477. https://doi.org/10.1007/s00018-016-2133-1

Liu, H., Du, T., Li, C., & Yang, G. (2021). STAT3 phosphorylation in central leptin resistance. *Nutrition & metabolism, 18*(1), 39. https://doi.org/10.1186/s12986-021-00569-w

Hoffman, N.J., Parker, B.L., Chaudhuri, R., Fisher-Wellman, K.H., Kleinert, M., Humphrey, S.J., Yang, P., Holliday, M., Trefely, S., Fazakerley, D.J., Stöckli, J., Burchfield, J.G., Jensen, T.E., Jothi, R., Kiens, B., Wojtaszewski, J.F., Richter, E.A., & James, D.E. (2015). Global Phosphoproteomic Analysis of Human Skeletal Muscle Reveals a Network of Exercise-Regulated Kinases and AMPK Substrates. *Cell metabolism, 22*(5), 922-935. https://doi.org/10.1016/j.cmet.2015.09.001

Gowans, G.J., Hawley, S.A., Ross, F.A., & Hardie, D.G. (2013). AMP is a true physiological regulator of AMP-activated protein kinase by both allosteric activation and enhancing net phosphorylation. *Cell metabolism, 18*(4), 556-566. https://doi.org/10.1016/j.cmet.2013.08.019

Yong, J., & Song, J. (2024). CaMKII activity and metabolic imbalance-related neurological diseases: Focus on vascular dysfunction, synaptic plasticity, amyloid beta accumulation, and lipid metabolism. *Biomedecine & pharmacotherapie,* 175, 116688. https://doi.org/10.1016/j.biopha.2024.116688

Plaza-Diaz, J., Izquierdo, D., Torres-Martos, Á., Baig, A.T., Aguilera, C.M., & Ruiz-Ojeda, F.J. (2022). Impact of Physical Activity and Exercise on the Epigenome in Skeletal Muscle and Effects on Systemic Metabolism. *Biomedicines, 10*(1), 126. https://doi.org/10.3390/biomedicines10010126

Ashcroft, S.P., Stocks, B., Egan, B., & Zierath, J.R. (2024). Exercise induces tissue-specific adaptations to enhance cardiometabolic health. *Cell metabolism, 36*(2), 278-300. https://doi.org/10.1016/j.cmet.2023.12.008

Headid Iii, R.J., & Park, S.Y. (2021). The impacts of exercise on pediatric obesity. *Clinical and experimental pediatrics, 64*(5), 196-207. https://doi.org/10.3345/cep.2020.00997

Wu, P.Y., Huang, K.S., Chen, K.M., Chou, C.P., & Tu, Y.K. (2021). Exercise, Nutrition, and Combined Exercise and Nutrition in Older Adults with Sarcopenia: A Systematic Review and Network Meta-analysis. *Maturitas,* 145, 38-48. https://doi.org/10.1016/j.maturitas.2020.12.009

Makarewicz, A., Jamka, M., Geltz, J., Śmidowicz, A., Kokot, M., Kaczmarek, N., Mądry, E., & Walkowiak, J. (2022). Comparison of the Effect of Endurance, Strength, and Endurance-Strength Training on Inflammatory Markers and Adipokines Levels in Overweight and Obese Adults: Systematic Review and Meta-Analysis of Randomised Trials. *Healthcare (Basel, Switzerland), 10*(6), 1098. https://doi.org/10.3390/healthcare10061098

Sanchis, P., Ezequiel-Rodriguez, A., Sánchez-Oliver, A.J., Suarez-Carmona, W., Lopez-Martín, S., García-Muriana, F.J., & González-Jurado, J.A. (2024). Changes in the Expression of Inflammatory Genes Induced by Chronic Exercise in the Adipose Tissue: Differences by Sex. *Sports (Basel, Switzerland), 12*(7), 184. https://doi.org/10.3390/sports12070184

Chow, L.S., Gerszten, R.E., Taylor, J.M., Pedersen, B.K., van Praag, H., Trappe, S., Febbraio, M.A., Galis, Z.S., Gao, Y., Haus, J.M., Lanza, I.R., Lavie, C.J., Lee, C.H., Lucia, A., Moro, C., Pandey, A., Robbins, J.M., Stanford, K.I., Thackray, A.E., Villeda, S., … Snyder, M.P. (2022). Exerkines in health, resilience and disease. *Nature reviews. Endocrinology, 18*(5), 273-289. https://doi.org/10.1038/s41574-022-00641-2

Colleluori, G., Viola, V., Bathina, S., Armamento-Villareal, R., Qualls, C., Giordano, A., & Villareal, D.T. (2025). Effect of aerobic or resistance exercise, or both on insulin secretion, ciliary neurotrophic factor, and insulin-like growth factor-1 in dieting older adults with obesity. *Clinical nutrition (Edinburgh, Scotland),* 51, 50-62. https://doi.org/10.1016/j.clnu.2025.05.016

Fiorenza, M., Checa, A., Sandsdal, R.M., Jensen, S.B. K., Juhl, C.R., Noer, M.H., Bogh, N.P., Lundgren, J.R., Janus, C., Stallknecht, B.M., Holst, J.J., Madsbad, S., Wheelock, C.E., & Torekov, S.S. (2024). Weight-loss maintenance is accompanied by interconnected alterations in circulating FGF21-adiponectin-leptin and bioactive sphingolipids. Cell reports. *Medicine, 5*(7), 101629. https://doi.org/10.1016/j.xcrm.2024.101629

Pataky, M.W., Young, W.F., & Nair, K.S. (2021). Hormonal and Metabolic Changes of Aging and the Influence of Lifestyle Modifications. *Mayo Clinic proceedings, 96*(3), 788-814. https://doi.org/10.1016/j.mayocp.2020.07.033

Hansen, D., Abreu, A., Ambrosetti, M., Cornelissen, V., Gevaert, A., Kemps, H., Laukkanen, J.A., Pedretti, R., Simonenko, M., Wilhelm, M., Davos, C.H., Doehner, W., Iliou, M.C., Kränkel, N., Völler, H., & Piepoli, M. (2022). Exercise intensity assessment and prescription in cardiovascular rehabilitation and beyond: why and how: a position statement from the Secondary Prevention and Rehabilitation Section of the European Association of Preventive Cardiology. *European journal of preventive cardiology, 29*(1), 230-245. https://doi.org/10.1093/eurjpc/zwab007

Sadeghi, H., Jehu, D.A., Daneshjoo, A., Shakoor, E., Razeghi, M., Amani, A., Hakim, M.N., & Yusof, A. (2021). Effects of 8 Weeks of Balance Training, Virtual Reality Training, and Combined Exercise on Lower Limb Muscle Strength, Balance, and Functional Mobility Among Older Men: A Randomized Controlled Trial. *Sports health, 13*(6), 606-612. https://doi.org/10.1177/1941738120986803

Jimeno-Almazán, A., Franco-López, F., Buendía-Romero, Á., Martínez-Cava, A., Sánchez-Agar, J.A., Sánchez-Alcaraz Martínez, B.J., Courel-Ibáñez, J., & Pallarés, J.G. (2022). Rehabilitation for post-COVID-19 condition through a supervised exercise intervention: A randomized controlled trial. *Scandinavian journal of medicine & science in sports, 32*(12), 1791-1801. https://doi.org/10.1111/sms.14240

Pillon, N.J., Gabriel, B.M., Dollet, L., Smith, J.A. B., Sardón Puig, L., Botella, J., Bishop, D.J., Krook, A., & Zierath, J.R. (2020). Transcriptomic profiling of skeletal muscle adaptations to exercise and inactivity. *Nature communications, 11*(1), 470. https://doi.org/10.1038/s41467-019-13869-w

Quiroga, R., Nistal, E., Estébanez, B., Porras, D., Juárez-Fernández, M., Martínez-Flórez, S., García-Mediavilla, M.V., de Paz, J.A., González-Gallego, J., Sánchez-Campos, S., & Cuevas, M.J. (2020). Exercise training modulates the gut microbiota profile and impairs inflammatory signaling pathways in obese children. *Experimental & molecular medicine, 52*(7), 1048-1061. https://doi.org/10.1038/s12276-020-0459-0

Brunelli, D.T., Bonfante, I.L. P., Boldrini, V.O., Scolfaro, P.G., Duft, R.G., Mateus, K., Fatori, R.F., Chacon-Mikahil, M.P. T., Farias, A.S., Teixeira, A.M., & Cavaglieri, C.R. (2024). Combined Training Improves Gene Expression Related to Immunosenescence in Obese Type 2 Diabetic Individuals. *Research quarterly for exercise and sport, 95*(3), 730-739. https://doi.org/10.1080/02701367.2023.2299716

Estébanez, B., Amaro-Gahete, F.J., Gil-González, C., González-Gallego, J., Cuevas, M.J., & Jiménez-Pavón, D. (2023). Influence of 12-Week Concurrent Training on Exosome Cargo and Its Relationship with Cardiometabolic Health Parameters in Men with Obesity. *Nutrients, 15*(13), 3069. https://doi.org/10.3390/nu15133069

Melson, E., Ashraf, U., Papamargaritis, D., & Davies, M.J. (2025). What is the pipeline for future medications for obesity?. *International journal of obesity (2005), 49*(3), 433-451. https://doi.org/10.1038/s41366-024-01473-y

Jastreboff, A.M., le Roux, C.W., Stefanski, A., Aronne, L.J., Halpern, B., Wharton, S., Wilding, J.P. H., Perreault, L., Zhang, S., Battula, R., Bunck, M.C., Ahmad, N.N., Jouravskaya, I., & SURMOUNT-1 Investigators (2025). Tirzepatide for Obesity Treatment and Diabetes Prevention. *The New England journal of medicine, 392*(10), 958-971. https://doi.org/10.1056/NEJMoa2410819

Ruiz-González, D., Hernández-Martínez, A., Valenzuela, P.L., Morales, J.S., & Soriano-Maldonado, A. (2021). Effects of physical exercise on plasma brain-derived neurotrophic factor in neurodegenerative disorders: A systematic review and meta-analysis of randomized controlled trials. *Neuroscience and biobehavioral reviews,* 128, 394-405. https://doi.org/10.1016/j.neubiorev.2021.05.025

Kao, S.C., Chen, F.T., Moreau, D., Drollette, E.S., Amireault, S., Chu, C.H., & Chang, Y.K. (2025). Acute effects of exercise engagement on neurocognitive function: a systematic review and meta-analysis on P3 amplitude and latency. *International Review of Sport and Exercise Psychology, 18*(1), 111-153. https://doi.org/10.1080/1750984X.2022.2155488

Dhahbi, W., Briki, W., Heissel, A., Schega, L., Dergaa, I., Guelmami, N., Omri, A.E., & Chaabene, H. (2025). Physical Activity to Counter Age-Related Cognitive Decline: Benefits of Aerobic, Resistance, and Combined Training-A Narrative Review. *Sports medicine - open, 11*(1), 56. https://doi.org/10.1186/s40798-025-00857-2

Rahmati, M., Lee, H., Lee, H., Park, J., Vithran, D.T. A., Li, Y., Kazemi, A., Boyer, L., Fond, G., Smith, L., Veronese, N., Soysal, P., Dragioti, E., Cortese, S., Kang, J., Yon, D.K., & Solmi, M. (2025). Associations Between Exercise Training, Physical Activity, Sedentary Behaviour and Mortality: An Umbrella Review of Meta-Analyses. *Journal of cachexia, sarcopenia and muscle, 16*(2), e13772. https://doi.org/10.1002/jcsm.13772

Batrakoulis, A., Jamurtas, A.Z., Metsios, G.S., Perivoliotis, K., Liguori, G., Feito, Y., Riebe, D., Thompson, W.R., Angelopoulos, T.J., Krustrup, P., Mohr, M., Draganidis, D., Poulios, A., & Fatouros, I.G. (2022). Comparative Efficacy of 5 Exercise Types on Cardiometabolic Health in Overweight and Obese Adults: A Systematic Review and Network Meta-Analysis of 81 Randomized Controlled Trials. Circulation. *Cardiovascular quality and outcomes, 15*(6), e008243. https://doi.org/10.1161/CIRCOUTCOMES.121.008243

Packer, M., Zile, M.R., Kramer, C.M., Baum, S.J., Litwin, S.E., Menon, V., Ge, J., Weerakkody, G.J., Ou, Y., Bunck, M.C., Hurt, K.C., Murakami, M., Borlaug, B.A., & SUMMIT Trial Study Group (2025). Tirzepatide for Heart Failure with Preserved Ejection Fraction and Obesity. *The New England journal of medicine, 392*(5), 427-437. https://doi.org/10.1056/NEJMoa2410027

Younossi, Z.M., Kalligeros, M., & Henry, L. (2025). Epidemiology of metabolic dysfunction-associated steatotic liver disease. *Clinical and molecular hepatology, 31*(Suppl), S32-S50. https://doi.org/10.3350/cmh.2024.0431

European Association for the Study of the Liver (2025). EASL Clinical Practice Guidelines on the management of hepatocellular carcinoma. *Journal of hepatology, 82*(2), 315-374. https://doi.org/10.1016/j.jhep.2024.08.028

Комбіновані тренування покращують регуляцію лептину, знижують вміст жиру в організмі та збільшують масу скелетних м’язів у жінок із ожирінням

Дві Кахйо Картіко[1ABDE], Пурво Срі Реджекі[2ABDE], Доно Індарто[3ABDE], Ірмантара Субагіо[1BD], Сасмінта Крістіна Юлі Хартаті[1BD], Бекір Ерхан Орхан[4CD], Юні Нурваті[1BD], Аді Праното[1ABCD]

[1]Державний університет Сурабаї
[2]Університет Ейрлангга
[3]Університет Себелас Марет
[4]Університет Айдин

Авторський вклад: A – дизайн дослідження; B – збір даних; C – статаналіз; D – підготовка рукопису; E – збір коштів

Реферат. Стаття: 11 с., 3 табл., 2 рис., 62 джерела.

Обґрунтування. Порушення регуляції лептину та несприятливий склад тіла є ключовими ознаками ожиріння у жінок. Попри те, що для покращення показників кількості жиру в організмі (адипозності) та м’язової маси зазвичай призначають комбіновані аеробно-силові тренування, дані щодо взаємозв’язку між змінами концентрації лептину в сироватці крові та детальними параметрами складу тіла у цієї категорії осіб залишаються обмеженими.

Цілі. Це рандомізоване контрольоване дослідження мало на меті вивчити вплив восьмитижневої програми комбінованих тренувань на концентрацію лептину, жирову масу та масу скелетних м’язів у жінок із ожирінням.

Матеріали та методи. Тридцять жінок із ожирінням були розподілені на групу з використанням методу комбінованих тренувань або контрольну групу без фізичних навантажень. Програма інтервенції включала аеробні та силові вправи помірної інтенсивності, які виконувалися п'ять разів на тиждень протягом восьми тижнів. Склад тіла, зокрема відсоток жиру в організмі (ВЖО), жирову маса (ЖМ), безжирову масу (БЖМ) та масу скелетних м'язів (МСМ), оцінювали за допомогою біоімпедансного аналізу (БІА). Концентрацію лептину в сироватці крові визначали методом імуноферментного аналізу (ІФА). Аналіз відмінностей у показниках перед та після інтервенції, а також між групами проведено за допомогою параметричних статистичних тестів (t-критерій для незалежних вибірок та t-критерій для парних вибірок) із порогом значущості $p < 0.05$.

Результати. В учасниць, які пройшли програму комбінованих тренувань, спостерігалося значуще зниження концентрації лептину в сироватці крові та показників адипозності (зокрема ВЖО та ЖМ), а також суттєве збільшення БЖМ та МСМ порівняно з контрольною групою ($p < 0.05$).

Висновки. Використання методу короткострокового комбінованого аеробно-силового тренування помірної інтенсивності асоційовано з позитивними змінами у показниках концентрації лептину. Отримані результати підтверджують потенційну роль структурованих комбінованих тренувань як дієвого підходу на основі фізичних вправ для покращення метаболічного та опорно-рухового профілів у межах даного дослідження.

Ключові слова: склад тіла, комбіновані тренування, лептин, м'язова маса, ожиріння.

Information about the Authors:

Kartiko, Dwi Cahyo: dwicahyo@unesa.ac.id; https://orcid.org/0000-0002-6727-0879; Department of Physical Education, Health and Recreation, Faculty of Sport and Health Science, Universitas Negeri Surabaya, Jl Raya Kampus Kampus, Lidah Wetan, Kec. Lakarsantri, Surabaya, East Java 60213, Indonesia.

Rejeki, Purwo Sri: purwo-s-r@fk.unair.ac.id; https://orcid.org/0000-0002-6285-4058; Physiology Division, Department of Medical Physiology and Biochemistry, Faculty of Medicine, Universitas Airlangga, Jl. Prof. DR. Moestopo No.47, Pacar Kembang, Kec. Tambaksari, Surabaya, East Java 60132, Indonesia.

Indarto, Dono: dono@staff.uns.ac.id; https://orcid.org/0000-0001-7420-5816; Department of Physiology, Faculty of Medicine, Universitas Sebelas Maret, Jalan Kolonel Sutarto, Jebres, Surakarta City, Central Java 57126, Indonesia.

Subagio, Irmantara: irmantarasubagio@unesa.ac.id; https://orcid.org/0000-0003-0265-7842; Department of Sports Coaching Education, Faculty of Sport and Health Science, Universitas Negeri Surabaya, Jl Raya Kampus Kampus, Lidah Wetan, Kec. Lakarsantri, Surabaya, East Java 60213, Indonesia.

Hartati, Sasminta Christina Yuli: sasmintahartati@unesa.ac.id; https://orcid.org/0009-0004-5698-9185; Department of Physical Education, Health and Recreation, Faculty of Sport and Health Science, Universitas Negeri Surabaya, Jl Raya Kampus Kampus, Lidah Wetan, Kec. Lakarsantri, Surabaya, East Java 60213, Indonesia.

Orhan, Bekir Erhan: bekirerhanorhan@aydin.edu.tr; https://orcid.org/0000-0002-3149-6630; Faculty of Sports Sciences, Istanbul Aydın University, Küçükçekmece, Istanbul 34295, Turkey.

Nurwati, Yuni: yuninurwati@unesa.ac.id; https://orcid.org/0009-0002-3229-5897; Department of Nutrition, Faculty of Sport and Health Science, Universitas Negeri Surabaya, Jl Raya Kampus Kampus, Lidah Wetan, Kec. Lakarsantri, Surabaya, East Java 60213, Indonesia.

Pranoto, Adi: adipranoto@unesa.ac.id; https://orcid.org/0000-0003-4080-9245; Department of Sports Coaching Education, Faculty of Sport and Health Science, Universitas Negeri Surabaya, Jl Raya Kampus Kampus, Lidah Wetan, Kec. Lakarsantri, Surabaya, East Java 60213, Indonesia.

Cite this article as: Kartiko, D. C., Rejeki, P. S., Indarto, D., Subagio, I., Hartati, S. C. Y., Orhan, B. E., Nurwati, Y., & Pranoto, A. (2026). Combination Training Improves Leptin Regulation, Reduces Body Fat, and Increases Skeletal Muscle Mass in Obese Females. *Physical Education Theory and Methodology, 26*(2), 380-390. https://doi.org/10.17309/tmfv.2026.2.16

Received: 13.11.2025. Accepted: 13.02.2026. Published: 30.03.2026