Shahiddoust and Monazzami
BMC Sports Science, Medicine and Rehabilitation (2025) 17:73
https://doi.org/10.1186/s13102-025-01123-4

BMC Sports Science, Medicine and Rehabilitation

RESEARCH

Open Access

Exercise-induced changes in insulin sensitivity, atherogenic index of plasma, and CTRP1/CTRP3 levels: the role of combined and high-intensity interval training in overweight and obese women

Fatemeh Shahiddoust[1] and Amir Abbas Monazzami[1*]

Abstract

Background Obesity, defined as excessive body fat accumulation, is closely linked to an increased risk of metabolic disorders, cardiovascular diseases, and insulin resistance. This study investigates the effects of combined training (CT) and high-intensity interval training (HIIT) on insulin sensitivity, atherogenic index of plasma (AIP), and serum levels of C1q/TNF-related proteins (CTRP1 and CTRP3) in overweight and obese women.

Methods Thirty-three overweight and obese women (aged 18–50 years) were randomly divided into three groups: control (CON, $n = 10$), HIIT($n = 9$), and combined training (CT, $n = 10$). The HIIT protocol consisted of intervals performed at 100% of maximum aerobic velocity (MAV) and rest intervals at 50% of MAV, with a 30-s work-to-rest ratio. The CT sessions included RT followed by AT. RT comprised seven exercises performed in three sets of 10–16 repetitions at 60–75% of one-repetition maximum (1RM). AT involved running for 15–30 min at 60–75% of heart rate reserve. Subjects trained three times per week. Body composition, biochemical, and functional assessments were conducted 48 h before and after the interventions.

Results Body mass index 1.3% and 2% ($p = 0.001$); TG 1.7%, 1.2% ($p = 0.001$);LDL 0.93%,0.83% ($p = 0.012$); HOMA-IR 9.5%,11.7% ($p = 0.018$); AST 4.2%,11.7% ($p = 0.001$); ALT 9.3%,10.9% ($p = 0.001$); 1RM 2.5%, 14.2% ($p = 0.001$); and maximum oxygen consumption 8%,2.4% ($p = 0.001$) showed significant improvements in both the HIIT and CT groups,resectively. Additionally, serum levels of CTRP 10.47%,0.34% ($p = 0.007$); and CTRP3 1.51%,1.53% ($p = 0.011$) significantly decreased in the HIIT and CT groups,resectively.

Conclusions The results suggest that HIIT and CT are effective strategies for improving body composition, lipid profile, glycemic control, liver enzyme levels, and functional capacity. Moreover, both exercise modalities were associated with reduced serum levels of the adipokines CTRP1 and CTRP3, highlighting a potential link between these biomarkers and improvements in body composition, lipid profile, glycemic control, and liver enzyme levels.

Trial registration Registered retrospectively in the Iranian Registry of Clinical Trials (IRCT20241207063967 N1) on 18/01/2025. Access at https://https://irct.behdasht.gov.ir/trial/80615.

Keywords Body composition, Obesity, Combined training, HIIT, CTRP3, CTRP1

*Correspondence:
Amir Abbas Monazzami
Monazzami.amirabbas@gmail.com

Background

Obesity and overweight have long been recognized as significant public health issues across various societies, with widespread impacts on individuals' physical and social health [1]. According to the World Health Organization, obesity and overweight are defined as an abnormal or excessive accumulation of body fat that poses serious health risks. The body mass index (BMI) is one of the most commonly used diagnostic criteria for assessing obesity and overweight. Specifically, a BMI between 25 and 29.9 kg/m^2 indicates overweight, while values over 30 kg/m^2 signify obesity [2, 3]. In recent decades, obesity has become one of the primary health concerns worldwide. The prevalence of this condition has increased rapidly, with global statistics indicating that over 1.9 billion people worldwide are affected by overweight, and approximately 650 million are classified as obese [3]. This issue has spread not only in developed countries but also in developing nations, with projections suggesting that by 2030, around 38% of the global adult population will be overweight, and 20% will be obese [4]. Obesity and overweight are associated with an increased risk of several diseases, including cardiovascular diseases (CVD), hypertension (HTN), type 2 diabetes (T2D), and several types of cancer [4]. These conditions can significantly diminish individuals'quality of life and lead to increased healthcare costs [5]. One of the novel biological markers known as the Atherogenic Index of Plasma (AIP), calculated from the ratio of triglyceride (TG) to high-density lipoprotein (HDL) cholesterol, has been recognized as an important predictor of cardiovascular and metabolic diseases. This index has a positive correlation with obesity and BMI and an inverse relationship with physical activity levels [6, 7].

One of the complex mechanisms through which obesity impacts health is through the dysregulation of adipokine secretion and the establishment of chronic low-grade inflammation [8]. Adipokines are bioactive proteins secreted by adipose tissue that play crucial roles in the regulation of metabolic processes [9]. Among the most significant adipokines are leptin and adiponectin, whose alterations in obese individuals disrupt glucose homeostasis, increase insulin resistance (IR), and ultimately lead to the development of T2D [10]. Additionally, C1Q/TNF-related proteins (CTRPs), a group of 15 proteins, similar to adiponectin, are involved in the regulation of glucose and fatty acid metabolism. CTRP1 and CTRP3 are notable members of this protein family that have important effects on metabolic processes such as IR, dyslipidemia, and reducing the risk of CVD. Previous studies have shown that CTRP1 activates the protein kinase B/Akt and p44/42-MAPK signaling pathways, leading to glucose uptake in muscle cells and enhanced insulin sensitivity (IS). Furthermore, recombinant CTRP1 injections have been found to reduce glucose levels in mice [11]. Additionally, circulating CTRP1 levels have been correlated with metabolic components such as BMI, homeostasis model assessment of IR (HOMA-IR), and fasting blood glucose (FBS). Previous studies have determined that circulating CTRP1 levels are elevated in patients with T2D, metabolic syndrome (MetS), CVD, HTN, and non-alcoholic fatty liver disease [11–13]. In humans, circulating levels of CTRP3 have shown a positive correlation with adiponectin, but a negative correlation with waist circumference, blood pressure, FBS, TG, and cholesterol. Furthermore, CTRP3 regulates glucose metabolism independently of insulin [14, 15]. This adipokine activates AMPK and enhances insulin signaling along with IS. Additionally, CTRP3 reduces the secretion of inflammatory cytokines from adipose tissue cells. Moreover, CTRP3 exerts protective effects on lipid metabolism and the cardiovascular system [14]. CTRP3 levels in serum are approximately 10 times lower than systemic adiponectin levels, and its concentration decreases further in obesity. These data suggest that CTRP3 is a potential target for the treatment of patients with MetS [16].

The unique roles of CTRP1 and CTRP3 in metabolic regulation distinguish them from other adipokines like leptin and adiponectin. While leptin and adiponectin primarily influence energy balance and systemic insulin sensitivity, CTRP1 and CTRP3 are more directly involved in the regulation of glucose and lipid metabolism at the cellular level. Moreover, the dysregulation of CTRP1 and CTRP3 in obesity and metabolic disorders highlights their potential as biomarkers and therapeutic targets for improving metabolic health [17, 18].

Given the adverse effects of obesity on public health and the complexities of the underlying mechanisms, various approaches have been proposed for the prevention and treatment of this issue. One of the most significant and effective approaches involves lifestyle interventions, including proper nutrition and physical activity [4]. Exercise activities, particularly aerobic training (AT) and resistance training (RT), play vital roles in weight reduction, improving cardiovascular health, and regulating metabolic processes. AT, which include activities such as walking and cycling, are recognized as a low-cost and accessible method for most individuals in the community. These types of exercises aid in reducing body fat and improving individuals'metabolic status by increasing energy expenditure during workout sessions [19]. RT is an activity aimed at increasing strength and muscle mass; improving metabolic components (such as enhancing dyslipidemia, HTN, and IR) with lower energy expenditure is among the benefits of this type of exercise [19]. Combining RT and AT in a training program

is referred to as combined training (CT) [20]. Findings indicate that CT is more effective than AT or RT alone for controlling body weight, body fat percentage (BFP), and reducing the risk of CVD [21]. In this context, Ho et al. (2012) observed greater improvements in body composition and cardiovascular fitness components in the CT group following 12 weeks of aerobic, resistance, or CT, conducted five days a week in overweight and obese men and women [22]. In recent years, another type of exercise known as high-intensity interval training (HIIT) has become a popular alternative to AT. HIIT consists of short bursts of intense exercise interspersed with recovery periods. The popularity of this type of training is attributed to its time efficiency [23]. Emerging evidence suggests that HIIT may lead to greater reductions in body fat compared to low to moderate-intensity AT. Furthermore, these exercises effectively reduce visceral and abdominal fat, which are the most dangerous types of fat [24]. In this regard, Tong et al. (2018) noted a significant reduction in BFP, and android and gynoid fat in both groups after 12 weeks of sprint interval training and traditional HIIT, with no significant inter-group differences; however, the reduction in visceral fat was better in the traditional HIIT group [25]. Among exercise modalities, combined training (CT), which integrates aerobic and resistance exercises, and high-intensity interval training (HIIT) have gained attention for their efficacy in improving insulin sensitivity, lipid profiles, and body composition [26, 27]. Exercise-induced changes in CTRP1 and CTRP3 levels may provide insights into the metabolic adaptations observed following physical training. For instance, reductions in CTRP1 levels post-exercise could reflect improvements in insulin sensitivity, while increases in CTRP3 may contribute to favorable lipid profile modifications [27]. Furthermore, the atherogenic index of plasma (AIP), a marker of cardiovascular risk, has been shown to correlate with adipokine levels, suggesting a potential link between CTRPs and atherogenic risk in overweight and obese populations [28]. On the other hand, the comparative effects of these training modalities on CTRP1 and CTRP3 levels, as well as their relationship with insulin sensitivity and AIP, have not been thoroughly investigated.However, women, especially in certain populations, may have a higher prevalence of obesity and related metabolic disorders compared to men.

This study aims to address this gap by examining the effects of CT and HIIT on insulin sensitivity, AIP, and serum levels of CTRP1 and CTRP3 in overweight and obese women. We hypothesize that both CT and HIIT will lead to significant improvements in insulin sensitivity and reductions in AIP, with concurrent modulation of CTRP1 and CTRP3 levels. Furthermore, we hypothesize that HIIT will elicit greater improvements in these outcomes compared to CT, due to its potent effects on metabolic and inflammatory pathways.

Therefore, the primary objective of this study was to determine the effects of eight weeks of CT and HIIT on serum levels of CTRP1 and CTRP3, anthropometric indices, IR/IS, and AIP in overweight and obese women. Additionally, the impact of these interventions on glycemic control components, lipid profiles, and fitness components were assessed.

Materials and methods

Study design and participants

The participants were volunteer women classified as overweight or obese. The inclusion criteria were: age between 18 and 50 years; BMI ranging from 25 to 34.9 kg/m^2; no engagement in regular physical activity during the past three months; absence of physical or motor disabilities; non-use of alcohol, tobacco, or specific medications; and no diagnosis of particular medical conditions (including cardiovascular, respiratory, renal, metabolic diseases, or cancer). The exclusion criteria included the occurrence of acute complications related to the exercise intervention (such as acute cardiovascular, renal, or respiratory conditions); musculoskeletal injuries; non-adherence to the exercise regimen (missing three consecutive sessions or five sessions in total); and personal withdrawal from the study. Sample size calculations, based on a significance level of 0.05, 80% statistical power, the standard deviation of the main outcome variables (5.6 ng/mL), and the desired effect size for the primary outcome (9.5 ng/mL), determined that 11 participants were needed per group, resulting in a total sample size of 33 [29, 30]. CTRP3 was identified as the primary outcome variable [31].

$$N = \frac{4(\partial)^2(Z_{crit} + Z_{pwr})^2}{D^2} \rightarrow N = \frac{4(5.6)^2(1.96 + 0.842)^2}{9.5^2} = 10.94 \cong 11 \quad (1)$$

Each of the three groups required 11 individuals, totaling 33 participants. Based on the formula,They were randomly assigned to one of three groups: control (CON), HIIT, and CT (Table 1 and Fig. 1). Randomization was carried out using a simple method involving numbered opaque sealed envelopes, as described by Doig and Simpson [2]. In brief, thirty-three standard-sized sheets of paper were divided into three

Table 1 Demographic characteristics of participants

Parameter (unit)	CON ($n=10$)	HIIT ($n=9$)	CT ($n=10$)
Age (yrs)	33.3 ± 6.1	34.8 ± 3.3	33.1 ± 8.9
Height (m)	1.62 ± 0.08	1.58 ± 0.07	1.59 ± 0.05
Body weight (kg)	77.47 ± 9.33	73.51 ± 6.55	73.66 ± 3.30
BMI (kg/m2)	29.14 ± 2.35	29.17 ± 1.51	28.91 ± 2.0

CON Control, *HIIT* High-intensity interval training, *CT* Combined training, *BMI* Body mass index

categories, with each category containing eleven papers labeled with the intervention type. The papers were folded, placed in sealed envelopes, shuffled to ensure randomization, and then numbered from 1 to 33. Participants were assigned to their respective intervention groups based on the order of their recruitment. Participants were instructed to maintain their usual dietary habits throughout the study, and no significant differences were observed in baseline characteristics among the groups. This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. All participants provided written informed consent after receiving a detailed explanation of the study's objectives and procedures. The study protocol was approved by the Research Ethics Committee of Razi University of Kermanshah (IR.RAZI.REC.1403.033) on Jun 21, 2023 and registered with the Iranian Registry of Clinical Trials (IRCT20241207063967 N1). This study was also adhered to CONSORT guidelines.

Body composition evaluations

Body Composition Evaluations were conducted at the Bardia Gym in Tuyserkan, following the guidelines of the American College of Sports Medicine (ACSM) [31]. Height was measured using a Seca stadiometer (Germany,accuracy: ± 1 mm,2013), weight was measured with a HN300 T2 digital scale (Japan,accuracy: ± 0.1 kg,2021), and hip and waist circumferences were measured using a tape measure. Additionally, BFP was assessed using the seven-site skinfold method with a caliper based on ACSM guidelines [31].

Biochemical evaluations

Blood sampling was performed after 12 h of fasting. A specialist collected blood samples from the right arm's brachial vein using five cc syringes and placed them in non-vacuum gel clot tubes. The samples were then centrifuged at 3000 rpm for 15 min. After centrifugation, serum levels of TG, HDL, low-density lipoprotein

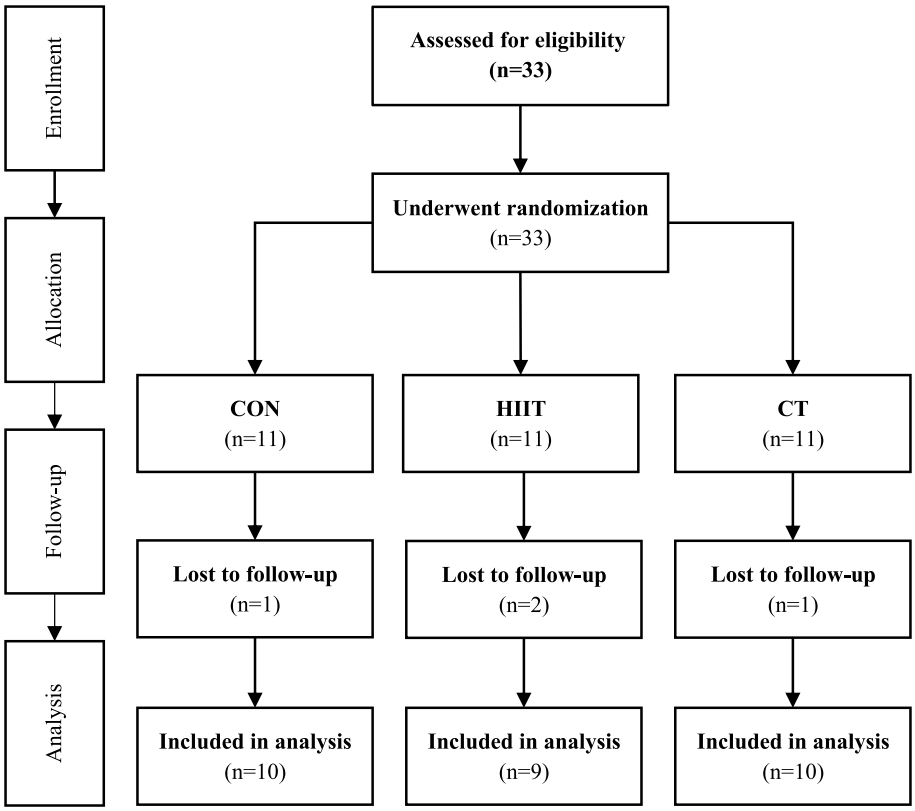

Fig. 1 Flowchart of trial participants. *CON:* control; *HIIT:* high-intensity interval training; *CT:* combined Training

(LDL), total cholesterol (TC), glucose, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were measured using appropriate kits (Pars Azmoon, Iran,2021), following the manufacturer's instructions, with an automated analyzer (Hitachi, Japan). To assess insulin and CTRPs, the ELISA method was employed. Serum insulin levels were measured using the Monobind ELISA kit (USA), with a sensitivity of 0.75 µIU/ml. Serum levels of CTRP1 were measured using the relevant ELISA kit with a sensitivity of 0.028 (BT LAB, China,2021). Serum levels of CTRP3 were assessed using the corresponding ELISA kit with a sensitivity of 0.026 ng/ml (BT LAB, China,2021), according to the manufacturer's instructions. The AIP, HOMA-IR, and quantitative IS check index (QUICKI) were evaluated using established methods [32–34].

Study groups

Control group

During the eight-week period, participants in this group continued with their regular daily activities. They were instructed to avoid engaging in any structured physical exercise and to refrain from taking specific medications (such as weight-loss drugs). To ensure compliance, these conditions were monitored every two weeks. Of the 11 participants in this group, one was excluded due to non-participation in the post-test evaluations.

Table 2 High-intensity interval training protocol

week	Work-rest intensity	Work-rest duration	Reps	sets	Rest between sets
1-2	50%– 100%	30s- 30s	4	4	5 min
3-4	50%– 100%	30s- 30s	6	4	5 min
5-6	50%– 100%	30s- 30s	6	5	5 min
7-8	50%– 100%	30s- 30s	6	6	5 min

Intensity is based on the maximum aerobic velocity (MAV)

High-intensity interval training group

Before beginning the main exercise routine, participants in this group performed a 10-min warm-up, consisting of light jogging and dynamic stretching. This was followed by the primary HIIT workout, which involved alternating intervals of exercise at 100% of maximal aerobic velocity (MAV) and recovery intervals at 50%, using a 30-s work-to-rest ratio. The number of sets ranged from four to six, and the number of repetitions varied from four to six per set, with five minutes of rest between sets. Training sessions were conducted three times per week. After each session, participants completed a five-minute cool-down with static stretching (Table 2). Two participants in this group were excluded due to inconsistent attendance.

Combined training group

Participants in this group engaged in both AT and RT for eight weeks, with three sessions per week. The training sessions followed a specific order, beginning with RT and then AT [35–40]. The RT included leg press, leg extension, leg curl, seated chest press, lat pulldown, cable bicep curls, and cable tricep extensions. The aerobic component involved running at 60–75% of heart rate reserve (HRR). Before and after each main session, participants performed a warm-up and cool-down routine same as the HIIT group (Table 3). One participant in this group was excluded due to irregular attendance. All repetitions, sets, and exercise intensities were supervised by the research team, and the exercises were frequently and carefully monitored throughout the research period.

Functional tests and familiarization

Participants'maximum oxygen consumption (VO2 max) was assessed using the beep test, one of the most valid field tests for measuring VO2 max, consisting of 21 levels. The test consisted of one-minute stages of continuous, incremental speed running. The running speed for participants started at 8.5 km/h, with an increase of 0.5

Table 3 Combined training protocol

Resistance training								
Week	1	2	3	4	5	6	7	7
Intensity	60%	65%	70%	65%	70%	75%	70%	70%
Reps	16	14	12	14	12	10	12	12
set	3	3	3	3	3	3	3	3
rest	60–90s	60–90s	60–90s	60–90s	60–90s	60–90s	60–90s	60–90s
Aerobic training								
Week	1	2	3	4	5	6	7	8
Intensity	60%	65%	70%	65%	70%	75%	70%	75%
Duration	15 min	20 min	25 min	20 min	25 min	30 min	25 min	30 min

Resistance and aerobic training intensities are based on the one repetition maximum and target heart rate, respectively

km/h at each level, continuing until they were unable to cover the 20-m distance between two cones [36]. Based on the level and the number of shuttles completed, $VO_{2\ max}$ was calculated. Additionally, the speed at the final stage was recorded and considered as the MAV. The MAV value was then used to determine the intensity for the HIIT sessions [38]. To determine the intensity of RT, an indirect one repetition maximum (1RM) test was conducted. The procedure involved the participant reaching a weight within 3–5 sets that allowed them to complete six correct repetitions (i.e., 6RM) [30]. The 1RM was then calculated using the Brzycki formula [41]. The intensity of AT was determined using a percentage of HRR (Buerer,FT90,Germany). First, the maximum heart rate (MHR) was calculated. Then, the resting heart rate (RHR) was subtracted from the MHR to find the HRR. After that, Based on the desired training intensity, the target heart rate (THR) was calculated. The intensity percentage was multiplied by the HRR, and the result was added to the RHR to determine the THR [42]. Additionally, a one-week familiarization period was implemented before starting the main training program for both the HIIT and CT groups. The goal of this phase was to familiarize participants with the types of exercises, proper execution of movements, and to prepare their bodies for the intensity of the upcoming sessions. During this week, the exercises were performed at low to moderate intensity, allowing participants to gradually get accustomed to their specific training protocols without excessive strain.

Statistical methods

Descriptive data are presented as mean ± standard deviation. Normality was checked with the Shapiro–Wilk test. A large p-value ($p = > 0.05$) indicates the data set is normally distributed, a low p-value ($p < 0.05$) indicates that it isn't normally distributed. A one-way ANOVA with post-hocTukey test was used to analyze the pre- to post-test changes in studied variables among the three groups. Repeated measures ANOVA assessed group × time interactions, with paired t-tests for within-group changes. Effect sizes were reported using partial eta-squared (ηp^2). values greater than 0.01 are considered small, those exceeding 0.06 are deemed moderate, and those surpassing 0.14 are classified as large. All analyses were performed at a 0.05 significance level using SPSS 26 (IBM, USA).

Results

Body composition

For BW, a significant main effect of time ($F_{1,26} = 62.177$, $p = 0.001$, $\eta_p^2 = 0.705$) and a significant time × group interaction ($F_{2,26} = 23.572$, $p = 0.001$, $\eta_p^2 = 0.645$) were observed. Significant decreases in BW occurred in the

Table 4 Study variables before and after training

Parameter (unit)		CON ($n = 10$)	HIIT ($n = 9$)	CT ($n = 10$)
Body weight (kg)	Pre	77.47 ± 9.33	73.51 ± 6.55	73.66 ± 3.30
	Post	77.62 ± 8.91	72.36 ± 6.54$^{**\&}$	72.06 ± 3.27$^{**\&}$
BMI (kg/m2)	Pre	29.14 ± 2.35	29.17 ± 1.51	28.91 ± 2.0
	Post	29.23 ± 2.21	28.71 ± 1.53$^{**\&}$	28.29 ± 2.01$^{**\&}$
WHR	Pre	0.959 ± 0.058	0.950 ± 0.042	0.941 ± 0.036
	Post	0.955 ± 0.055	0.942 ± 0.044**	0.928 ± 0.039**
BFP	Pre	25.6 ± 4.47	27.02 ± 1.84	26.9 ± 2.11
	Post	25.72 ± 4.58	26.01 ± 1.85$^{**\&}$	25.25 ± 2.13$^{**\&}$
TG (mg/dL)	Pre	173.8 ± 10.77	171.0 ± 12.34	165.9 ± 8.31
	Post	174.0 ± 10.17	168.33 ± 12.41$^{**\&}$	163.0 ± 7.54$^{**\&}$
LDL (mg/dL)	Pre	147.7 ± 12.31	149.0 ± 9.97	145.0 ± 13.58
	Post	148.0 ± 12.12	147.66 ± 9.32$^{**\#}$	163.8 ± 13.07$^{*\#}$
HDL (mg/dL)	Pre	39.0 ± 2.26	39.77 ± 2.99	39.1 ± 2.42
	Post	39.1 ± 2.21	40.44 ± 2.6*	39.8 ± 2.2*
TC (mg/dL)	Pre	221.2 ± 12.33	222.66 ± 12.54	217.4 ± 13.38
	Post	221.5 ± 12.34	221.11 ± 12.19$^{**\&}$	216.0 ± 12.92$^{*\&}$
AIP	Pre	0.648 ± 0.047	0.633 ± 0.040	0.628 ± 0.039
	Post	0.649 ± 0.044	0.619 ± 0.038$^{**\#}$	0.612 ± 0.033$^{**\#}$
Glucose (mg/dL)	Pre	100.0 ± 8.76	100.33 ± 9.40	92.40 ± 4.88
	Post	98.90 ± 7.72	97.11 ± 8.40$^{**\#}$	89.40 ± 4.27$^{**\#}$
Insulin (μIU/ml)	Pre	16.30 ± 2.16	16.88 ± 2.71	14.9 ± 1.19
	Post	16.10 ± 1.79	15.77 ± 2.22$^{**\#}$	13.9 ± 1.19$^{**\#}$
HOMA-IR	Pre	4.06 ± 0.89	4.23 ± 1.07	3.40 ± 0.43
	Post	3.95 ± 0.72	3.82 ± 0.86$^{**\#}$	3.07 ± 0.4**
QUICKI	Pre	0.312 ± 0.008	0.310 ± 0.010	0.318 ± 0.005
	Post	0.312 ± 0.007	0.314 ± 0.009$^{**\&}$	0.323 ± 0.005$^{**\&}$
CTRP- 1 (ng/ml)	Pre	63.46 ± 12.41	62.97 ± 12.94	58.69 ± 11.64
	Post	63.44 ± 12.43	62.63 ± 13.06$^{**\&}$	58.40 ± 11.52$^{**\#}$
CTRP- 3 (ng/L)	Pre	28.13 ± 4.76	26.48 ± 3.27	26.14 ± 3.39
	Post	28.1 ± 4.63	26.05 ± 3.21$^{*\#}$	25.75 ± 3.29$^{**\#}$
AST (IU/L)	Pre	22.30 ± 2.62	23.88 ± 1.90	22.90 ± 2.80
	Post	22.50 ± 2.32	20.77 ± 1.78$^{**\&}$	20.20 ± 2.25$^{**\&}$
ALT (IU/L)	Pre	25.50 ± 2.71	26.88 ± 1.90	26.50 ± 2.75
	Post	25.60 ± 2.22	24.33 ± 2.0$^{**\&}$	23.60 ± 2.63$^{**\&}$
1RM (kg)	Pre	27.8 ± 3.42	28.0 ± 4.24	28.2 ± 4.15
	Post	28.4 ± 2.98	28.7 ± 3.83	32.5 ± 4.79$^{**\&\$}$
VO2 max (ml.kg.min)	Pre	20.22 ± 1.09	20.08 ± 1.0	20.23 ± 1.21
	Post	20.13 ± 0.86	21.6 ± 0.98$^{**\&}$	21.74 ± 1.22$^{**\&}$

Significant differences compared to pre-test: $^{*}p < 0.05$, $^{**}p < 0.01$

Significant changes observed following the -week intervention compared to CON: $^{\#}p < 0.05$, $^{\&}p < 0.01$; and compared to HIIT: $^{\$}p < 0.01$

CON Control, *HIIT* High-intensity interval training, *CT* Combined training, *BMI* Body Mass Index, *WHR* Waist-to-hip Ratio, *BFP* Body Fat Percentage, *TG* Triglyceride, *LDL* Low-density lipoprotein, *HDL* High-density lipoprotein, *TC* Total cholesterol, *AIP* Atherogenic Index of Plasma, *HOMA-IR* Homeostatic Model Assessment for Insulin Resistance, *QUICKI* Quantitative Insulin Sensitivity Check Index, *CTRPs* C1Q/TNF-Related Proteins, *1RM* One-repetition Maximum, *VO2 max* Maximum oxygen consumption

HIIT ($p = 0.001$) and CT ($p = 0.001$) groups (Table 4). The changes in BW were significant in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups compared to the CON group. For BMI, a significant main effect of time ($F_{1,26} = 59.086$, $p = 0.001$, $\eta_p^2 = 0.694$) and a significant time × group interaction ($F_{2,26} = 25.657$, $p = 0.001$, $\eta_p^2 = 0.664$) were found. Significant decreases in BMI were noted in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups. The changes in BMI were significant in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups compared to the CON group (Table 4). For WHR, a significant main effect of time ($F_{1,26} = 25.583$, $p = 0.001$, $\eta_p^2 = 0.496$) were identified. Significant decreases in WHR were seen in the HIIT ($p = 0.008$) and CT ($p = 0.004$) groups. For BFP, a significant main effect of time ($F_{1,26} = 92.565$, $p = 0.001$, $\eta_p^2 = 0.781$) and a significant time × group interaction ($F_{2,26} = 35.768$, $p = 0.001$, $\eta_p^2 = 0.733$) were observed (Table 4). Significant decreases in BFP occurred in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups. The changes in BFP were significant in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups compared to the CON group. (Table 4). The heatmap correlation analysis between all variables are also shown in Fig. 2.

Lipid profile

For TG, a significant main effect of time ($F_{1,26} = 44.164$, $p = 0.001$, $\eta_p^2 = 0.629$) and a significant time × group interaction ($F_{2,26} = 14.028$, $p = 0.001$, $\eta_p^2 = 0.519$) were found. Significant decreases in TG occurred in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups. The changes in TG were significant in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups compared to the CON group (Table 4). For LDL, a significant main effect of time ($F_{1,26} = 10.5$, $p = 0.003$, $\eta_p^2 = 0.288$) and a significant time × group interaction ($F_{2,26} = 5.27$, $p = 0.012$, $\eta_p^2 = 0.288$) were observed. Significant decreases in LDL were noted in the HIIT ($p = 0.002$) and CT ($p = 0.024$) groups (Table 4). The changes in LDL were significant in the HIIT ($p = 0.021$) and CT ($p = 0.03$) groups compared to the CON group. For HDL, a significant main effect of time ($F_{1,26} = 7.766$, $p = 0.01$, $\eta_p^2 = 0.23$) were observed. Significant increases in HDL were noted in the HIIT ($p = 0.05$) and CT ($p = 0.025$) groups (Table 4). For TC, a significant main effect of time ($F_{1,26} = 16.973$, $p = 0.001$, $\eta_p^2 = 0.395$) and a significant time × group interaction ($F_{2,26} = 7.76$, $p = 0.002$, $\eta_p^2 = 0.374$) were found. Significant decreases in TC occurred in the HIIT ($p = 0.001$) and CT ($p = 0.025$) groups. The changes in TC were significant in the HIIT ($p = 0.005$) and CT ($p = 0.008$) groups compared to the CON group. For AIP, a significant main effect of time ($F_{1,26} = 21.062$, $p = 0.001$, $\eta_p^2 = 0.448$) and a significant time × group interaction ($F_{2,26} = 5.974$, $p = 0.007$, $\eta_p^2 = 0.315$) were observed (Table 4). Significant decreases in AIP occurred in the HIIT ($p = 0.002$) and CT ($p = 0.001$) groups. The changes in AIP were significant in the HIIT ($p = 0.023$) and CT ($p < 0.013$) groups compared to the CON group. (Table 4 and Fig. 3). There was also a negative correlation but significant between changes in HDL with changes of AIP($p < 0.01$) but not with HOMA-IR

	Weight_delta	BMI_delta	FAT_delta	WHR_delta	CTRP1_delta	TRP3_delta	TG_delta	TC_delta	HDL_delta	LDL_delta	AIP_delta	sulin_delta	ucose_delta	MA-IR_delta	uicki_delta	1RM_delta	2max_delta	AST_delta	ALT_delta
Weight_delta																			
BMI_delta	0.99191934																		
FAT_delta	0.96823728	0.9657258																	
WHR_delta	0.41951666	0.3685732	0.41462804																
CTRP1_delta	0.27625518	0.3437567	0.30673455	-0.20915546															
CTRP3_delta	0.54700154	0.57122	0.57487069	-0.02499607	0.484200832														
TG_delta	0.41499491	0.4341629	0.45980814	0.19921655	0.613727421	0.503321													
TC_delta	0.49833251	0.5207555	0.54749071	0.24755767	0.261840791	0.515476	0.366299												
HDL_delta	-0.2669412	-0.2725331	-0.2603979	-0.32103916	-0.30824964	-0.2385	-0.45155	-0.05002											
LDL_delta	0.46549485	0.4885387	0.51045618	0.33214793	0.227130337	0.492875	0.327868	0.89709	-0.32223										
AIP_delta	0.37726439	0.3876894	0.38339234	0.32176731	0.480363552	0.385132	0.727284	0.181912	-0.93779	0.370263									
Insulin_delta	0.24708045	0.2903079	0.31297671	-0.02165356	0.387683373	0.500837	0.503754	0.420724	-0.25654	0.420531	0.380484								
Glucose_delta	0.2683903	0.31572	0.2952964	0.17306563	0.479666285	0.442111	0.494926	0.472456	-0.20289	0.371171	0.347466	0.680667							
HOMA-IR_delta	0.22093054	0.2667719	0.27413297	0.05568328	0.416572637	0.491181	0.467767	0.430524	-0.25717	0.416997	0.36925	0.952213	0.839383						
Quicki_delta	-0.3418328	-0.3886307	-0.4090005	0.00810449	-0.44529289	-0.53601	-0.59563	-0.50375	0.239992	-0.44309	-0.40771	-0.95263	-0.74643	-0.89961					
1RM_delta	-0.4929205	-0.4809162	-0.4886399	-0.24034163	-0.18748637	-0.16854	-0.36413	-0.19293	0.285256	-0.15869	-0.36898	-0.08331	-0.1856	-0.04103	0.24136				
VO2max_delta	-0.6885851	-0.7107688	-0.7225644	-0.32336595	-0.51466094	-0.60069	-0.64369	-0.48041	0.47595	-0.43542	-0.61357	-0.53932	-0.52882	-0.5206	0.632786	0.341094			
AST_delta	-0.6209406	-0.6492592	-0.6297369	-0.20998943	-0.43864787	-0.33293	-0.43677	-0.40066	0.064162	-0.30132	-0.231	-0.3388	-0.32955	-0.33224	0.375564	0.13639	0.510146		
ALT_delta	-0.6561098	-0.6821966	-0.6842657	-0.25312959	-0.47871194	-0.34274	-0.47427	-0.32389	0.336407	-0.29648	-0.44801	-0.29691	-0.28418	-0.28073	0.352972	0.342754	0.652153	0.810765	

Fig. 2 The Heatmap Correlations analysis of studied variables

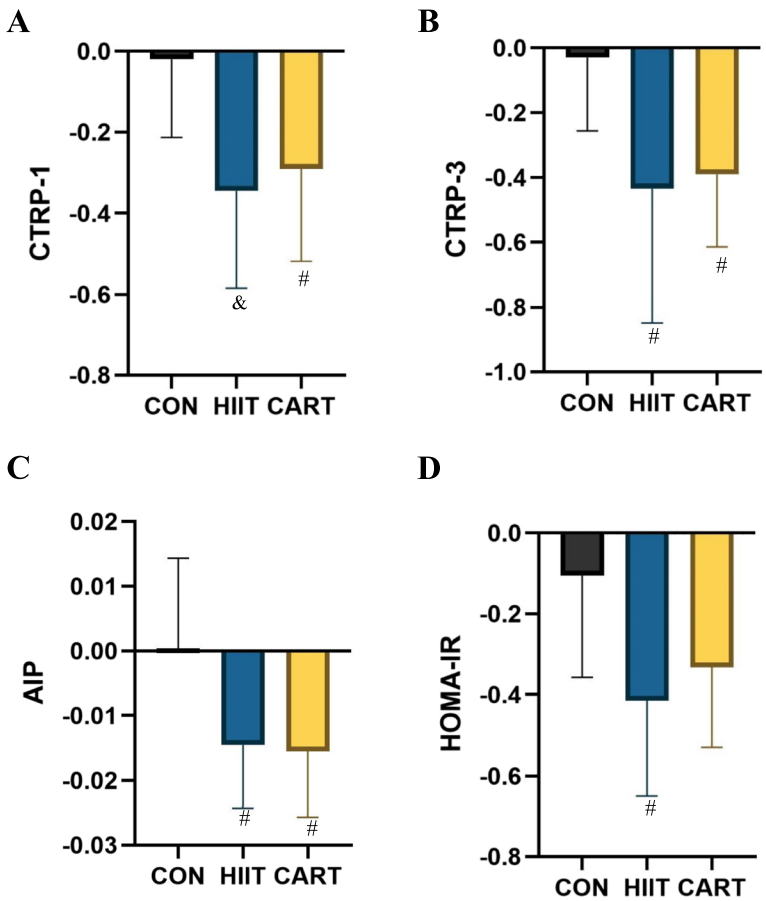

Fig. 3 Pre- to post-training changes. Changes in serum levels of (**A**) C1q/TNF-Related Protein- 1 and (**B**) C1q/TNF-Related Protein- 3. Changes in (**C**) atherogenic index of plasma, and (**D**) homeostatic model assessment for insulin resistance. Data are presented as means ± standard deviations. Significant differences compared to CON: $^{\#}p < 0.05$, $^{\&}p < 0.01$. *CON:* control; *HIIT:* high-intensity interval training; *CT:* combined Training

Table 5 Correlation between Changes in Anthropometric, performance and clinical variables

	BMI ($n = 30$)	AIP ($n = 30$)	HOMA-IR ($n = 30$)
HDL	− 0.273	− 0.938 **	− 0.257
LDL	0.489 **	0.37 *	0.417 *
AST	− 0.649 **	− 0.231	− 0.332
ALT	− 0.682 **	− 0.448*	− 0.281
VO2 max	− 0.711 **	− 0.614 **	− 0.521 **
1RM	− 0.481 **	− 0.369 *	− 0.04
CTRP- 1	0.344	0.48 **	0.417 *
CTRP- 3	0.571 **	0.385 *	0.491 **

Significant correlations are indicated as follows: *$p < 0.05$, **$p < 0.01$

BMI Body Mass Index, AIP Atherogenic Index of Plasma, HOMA-IR Homeostatic Model Assessment for Insulin Resistance, HDL High density lipoprotein, LDL Low density lipoprotein, AST Aspartate Aminotransferase, ALT Alanine Aminotransferase, VO2 max Maximum Oxygen Uptake, 1RM One Repetition Maximum, CTRPs C1Q/TNF-Related Proteins

($p = 0.175$; Table 5). The result also showed a significant correlation between changes of LDL with changes of BMI ($p = 0.007$), AIP($p = 0.048$) and HOMA-IR ($p = 0.023$; Table 5).

Glycemic control

For glucose, a significant main effect of time ($F_{1,26} = 63.596$, $p = 0.001$, $\eta_p^2 = 0.71$) and a significant time × group interaction ($F_{2,26} = 4.905$, $p = 0.016$, $\eta_p^2 = 0.274$) were found. Significant decreases in glucose occurred in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups. The changes in glucose were significant in the HIIT ($p = 0.025$) and CT ($p = 0.041$) groups compared to the CON group (Table 4). For insulin, a significant main effect of time ($F_{1,26} = 35.742$, $p = 0.001$, $\eta_p^2 = 0.579$) and a significant time group interaction ($F_{2,26} = 5.019$, $p = 0.014$, $\eta_p^2 = 0.279$) were observed. Significant decreases in insulin were noted in the HIIT ($p = 0.001$) and CT ($p = 0.001$) groups. The changes in insulin were significant in the HIIT ($p = 0.022$) and CT ($p = 0.041$) groups compared

to the CON group (Table 4). For HOMA-IR, a significant main effect of time ($F_{1,26}$= 44.481, p= 0.001, η_p^2= 0.631) and a significant time ×group interaction ($F_{2,26}$= 4.734, p= 0.018, η_p^2= 0.267) were identified. Significant decreases in HOMA-IR occurred in the HIIT (p= 0.001) and CT (p= 0.001) groups. The changes in HOMA-IR were significant in the HIIT (p= 0.018) group compared to the CON group (Table 4 and Fig. 3). For QUICKI, a significant main effect of time ($F_{1,26}$= 58.227, p= 0.001, η_p^2= 0.691) and a significant time ×group interaction ($F_{2,26}$= 8.347, p= 0.002, η_p^2= 0.391) were observed. Significant increases in QUICKI were noted in the HIIT (p= 0.001) and CT (p= 0.001) groups. The changes in QUICKI were significant in the HIIT (p= 0.01) and CT (p= 0.002) groups compared to the CON group (Table 4).

C1Q/TNF-related proteins

For CTRP- 1, a significant main effect of time ($F_{1,26}$= 28.246, p= 0.001, η_p^2= 0.521) and a significant time ×group interaction ($F_{2,26}$= 6.025, p= 0.007, η_p^2= 0.317) were found. Significant decreases in CTRP- 1 occurred in the HIIT (p= 0.003) and CT (p= 0.003) groups. The changes in CTRP- 1 were significant in the HIIT (p= 0.01) and CT (p= 0.029) groups compared to the CON group (Table 4 and Fig. 3). For CTRP- 3, a significant main effect of time ($F_{1,26}$= 26.574, p= 0.001, η_p^2= 0.505) and a significant time ×group interaction ($F_{2,26}$= 5.436, p= 0.011, η_p^2= 0.295) were observed. Significant decreases in CTRP- 3 occurred in the HIIT (p= 0.014) and CT (p= 0.001) groups. The changes in CTRP3 were significant in the HIIT (p= 0.017) and CT (p= 0.03) groups compared to the CON groups (Table 4 and Fig. 3). There was also a significant correlation between changes in CTRP- 1 with changes of AIP (p< 0.01) and HOMA-IR (p= 0.025;Table 5). The result also showed a significant correlation between changes of CTRP- 3 with changes of BMI (p= 0.001), AIP (p< 0.05) and HOMA-IR (p= 0.007;Table 5).

Aminotransferases

For AST, a significant main effect of time ($F_{1,26}$= 56.477, p= 0.001, η_p^2= 0.685) and a significant time ×group interaction ($F_{2,26}$= 17.733, p= 0.001, η_p^2= 0.577) were found. Significant decreases in AST occurred in the HIIT (p= 0.001) and CT (p= 0.001) groups. The changes in AST were significant in the HIIT (p= 0.001) and CT (p= 0.001) groups compared to the CON group (Table 4). For ALT, a significant main effect of time ($F_{1,26}$= 52.083, p= 0.001, η_p^2= 0.667) and a significant time ×group interaction ($F_{2,26}$= 15.052, p= 0.001, η_p^2= 0.537) were found. Significant decreases in ALT occurred in the HIIT (p= 0.001) and CT (p= 0.001) groups. The changes in ALT were significant in the HIIT (p= 0.001) and CT (p= 0.001) groups compared to the CON group (Table 4). There was also a significant.

Correlation between changes in AST with changes of BMI (p< 0.01;Table 5). The result also showed a negative correlation but significant between changes of ALT with changes of BMI and AIP (p< 0.01;Table 5).

Strength and maximal oxygen consumption

For strength, a significant main effect of time ($F_{1,26}$= 32.051, p= 0.001, η_p^2= 0.552) and a significant time ×group interaction ($F_{2,26}$= 13.271, p= 0.001, η_p^2= 0.505) were found. Significant increases in strength were observed in the CT (p= 0.001) group and the changes in strength were significant in this group compared to the HIIT (p= 0.001) and CON (p= 0.001) groups (Table 4). For $VO_{2\ max}$, a significant main effect of time ($F_{1,26}$= 111.718, p= 0.001, η_p^2= 0.811) and a significant time ×group interaction ($F_{2,26}$= 33.949, p= 0.001, η_p^2= 0.723) were observed. Significant increases in $VO_{2\ max}$ occurred in the HIIT (p= 0.001) and CT (p= 0.001) groups. The changes in $VO_{2\ max}$ were significant in the HIIT (p= 0.001) and CT (p= 0.001) groups compared to the CON group (Table 4). The result also showed a negative correlation but significant between changes of VO2 max with changes of BMI, AIP and HOMA-IR (p< 0.01;Table 5). The result also showed a negative correlation but significant between changes of 1RM with changes of BMI (p= 0.008) and AIP (p= 0.049; Table 5).

Discussion

This study demonstrated that both HIIT and CT are effective in improving key health parameters in overweight and obese women. Significant enhancements in body composition, lipid profile, glycemic control, liver enzyme levels, and functional capacity were accompanied by reductions in the adipokines CTRP1 and CTRP3. These findings suggest that structured exercise interventions can play a critical role in mitigating metabolic and cardiovascular risk factors while also influencing adipokine regulation, thereby contributing to overall metabolic health in this population.

Body composition

The findings of the present study indicate a significant improvement in body composition indices in the HIIT and CT groups. Consistent with these results, Tong et al. (2018) observed a significant reduction in BFP and android and gynoid fat mass after 12 weeks of HIIT [25]. Similarly, Talebi et al. (2021) reported improvements in body composition in overweight individuals after eight weeks of CT [43]. A meta-analysis by Wewege et al. (2017) demonstrated that HIIT is effective in reducing

body fat percentage and visceral fat, which are critical indicators of metabolic health in overweight and obese individuals [23].In general, exercise facilitates the conversion of white adipose tissue into brown adipose tissue, contributing to fat loss and weight reduction [44]. During HIIT, energy metabolism in high-intensity intervals primarily relies on carbohydrates as a substrate. However, the use of fatty acids from adipose tissue becomes the main fuel source for increased post-exercise metabolism [25]. On the other hand, AT stimulates fatty acid metabolism through mitochondrial oxidation in muscles to produce large amounts of ATP. AT with high ATP demand activates the AMPK pathway, which conserves ATP by inhibiting biosynthetic and anabolic pathways and stimulating catabolic pathways through increased glucose transport and fat metabolism [45]. The activation of AMP-activated protein kinase (AMPK) during exercise plays a crucial role in energy metabolism. AMPK activation inhibits anabolic pathways and stimulates catabolic pathways, leading to increased glucose uptake and fat oxidation. This mechanism is particularly relevant in CT, where the combination of AT and RT enhances mitochondrial biogenesis and fatty acid oxidation, contributing to improved body composition [46]. Meanwhile, RT which focuses on muscle hypertrophy, releases anti-inflammatory myokines and activates AMPK pathways, promoting body fat reduction. Additionally, AT is often associated with greater energy expenditure, while RT helps preserve muscle mass, altering fat mass composition [34]. Overall, it can be concluded that HIIT, by increasing post-exercise resting energy expenditure, and CT, by activating catabolic pathways and improving lean body mass, have led to a significant reduction in body composition components compared to the pre-test and the control group. The correlation analysis results showed that changes in Body Mass Index (BMI) were negatively correlated with VO2 max ($r = -0.711$, $p < 0.01$) and positively correlated with LDL ($r = 0.489$, $p < 0.01$) and CTRP- 3 ($r = 0.571$, $p < 0.01$). This suggests that reductions in BMI are associated with improvements in aerobic capacity, lipid profile, and adipokine levels, highlighting the interconnected nature of body composition and metabolic health.

Lipid profile

The findings of this study show a significant improvement in lipid profile indices in both HIIT and CT groups. Consistent with these findings, Khammassi et al. (2018) reported a significant reduction in TC and TG in obese individuals after 12 weeks of HIIT [46]. Similarly, Rossi et al. (2016) observed a significant increase in HDL after 16 weeks of CT. However, AIP showed no change in their study [47]. Different forms of exercise are crucial for improving the lipid profile, although the exact mechanisms are not fully understood. Exercise generally improves muscle capacity to use lipids instead of carbohydrates, thus lowering plasma lipid levels [48]. These effects are primarily on peripheral tissues like adipose tissue and the liver, resulting in increased activity of the enzyme lecithin–cholesterol acyltransferase [49], which transfers ester groups to HDL, known as "good" cholesterol. Increased activity of lipoprotein lipase is another potential mechanism that helps lower plasma fat after exercise [50]. Exercise training enhances the capacity of skeletal muscle to utilize lipids as a fuel source, reducing plasma lipid levels. Additionally, exercise-induced improvements in insulin sensitivity lead to better regulation of lipid metabolism, as insulin plays a key role in lipid storage and mobilization. The reduction in the atherogenic index of plasma (AIP) further supports the cardioprotective effects of both HIIT and CT [26]. Overall, it can be concluded that both HIIT and CT, in addition to improving body composition and insulin resistance, enhance the efficiency of enzymes involved in lipid homeostasis, thereby improving the lipid profile. The correlation analysis results showed that AIP, a marker of cardiovascular risk, showed a strong negative correlation with changes in HDL cholesterol ($r = -0.938$, $p < 0.01$). This indicates that improvements in HDL levels are associated with a reduction in AIP, suggesting that exercise-induced increases in HDL may contribute to a lower atherogenic risk. Conversely, LDL cholesterol was positively correlated with AIP ($r = 0.37$, $p < 0.05$), reinforcing the notion that reductions in LDL are beneficial for cardiovascular health, as they are associated with a decrease in AIP.

Glycemic markers

The findings of the study indicate significant improvements in glycemic control indices in the HIIT and CT groups. Consistent with the results of this study, Ahmad et al. (2023) reported improvements in glycemic markers after 12 weeks of low- and high-volume HIIT compared to pre-intervention and control groups [51]. Similarly, Sigal et al. (2007) observed significant improvements in glycemic markers in individuals with T2D following 22 weeks of CT [52]. Potential mechanisms for the increased glucose uptake by muscles during and after HIIT include higher utilization of muscle glycogen, improved IS, and increased insulin-independent glucose transporter- 4 expression [53]. Furthermore, studies indicate that exercise programs that incorporate a RT component, may be beneficial for enhancing insulin function and glycemic control in T2D. Increased muscle mass is associated with benefits for blood glucose control, as skeletal muscle is the largest insulin-sensitive tissue [54]. Additionally,

combined training is related to improvements in aerobic capacity independent of body weight changes, likely due to enhanced blood flow and increased tissue exposure to insulin and glucose [55]. The improvement in insulin sensitivity is mediated by several mechanisms, including increased muscle glycogen utilization during exercise, enhanced insulin signaling pathways, and increased muscle mass (particularly in CT). Skeletal muscle is the largest insulin-sensitive tissue in the body, and increased muscle mass from RT contributes to better glucose control. Additionally, exercise-induced improvements in blood flow and capillary density enhance tissue exposure to insulin and glucose, further improving glycemic control [52]. In summary, both HIIT and CT have positively influenced cardiorespiratory and musculoskeletal functions, leading to favorable metabolic adaptations and improvements in glucose and insulin levels. The correlation analysis results showed that HOMA-IR also correlated positively with LDL ($r = 0.417$, $p < 0.05$) and CTRP- 1 ($r = 0.417$, $p < 0.05$), suggesting that higher levels of LDL and CTRP- 1 are associated with greater insulin resistance. This further supports the idea that improvements in lipid profile and reductions in adipokine levels (such as CTRP- 1) may contribute to better glycemic control.

C1Q/TNF-related proteins

The findings of the present study indicate a significant decrease in CTRP1 and CTRP3 levels in the HIIT and CT groups. The results regarding the long-term effects of exercise on CTRP1 and CTRP3 levels are inconsistent. Following 12 weeks of HIIT Masoumzadeh et al. (2021) reported significant decreases in CTRP1 and significant increases in CTRP3 [56]. Choi et al. (2013) observed a reduction in CTRP3 levels in obese women after three months of CT [56]. Similarly, Hasegawa et al. (2018) reported that aerobic exercise training reduced CTRP3 levels, which were correlated with improved arterial stiffness in middle-aged and older adults [17]. These results align with those of the present study. However, Mirzendedel et al. (2019) reported increased serum levels of CTRP3 after 12 weeks of CT [18]. CTRP1 exerts insulin-sensitizing effects and enhancing fatty acid oxidation and energy expenditure [57]. It has been shown that levels of this substance are higher in individuals with MetS compared to healthy individuals [58]. The discrepancy in the findings regarding the effects of exercise on CTRP1 and CTRP3 levels across different studies can be attributed to several factors, including differences in study design, participant characteristics, exercise protocols, and measurement techniques. The reduction in CTRP1 and CTRP3 levels following CT may be attributed to the combined effects of aerobic and resistance training on lipid metabolism and glucose uptake. Aerobic exercise enhances mitochondrial biogenesis and fatty acid oxidation, while resistance training increases muscle mass, which is the largest insulin-sensitive tissue in the body [18, 57–62]. While, the reduction in CTRP1 level following HIIT may be linked to improved lipid metabolism and reduced inflammation of adipose tissue [18, 57–62]. CTRP1 activates the protein kinase B/Akt and p44/42-MAPK signaling pathways, leading to glucose uptake in muscle cells and enhanced insulin sensitivity. CTRP3, on the other hand, activates AMPK and enhances insulin signaling, contributing to improved glucose and lipid metabolism. The reduction in these adipokines post-exercise suggests that both HIIT and CT positively influence adipokine regulation, leading to improved metabolic health [18, 57–62]. CTRP1 concentrations are also elevated in diabetic individuals [60]. On the other hand, the administration of CTRP3 reduces glucose concentration in both insulin-sensitive wild-type mice and insulin-resistant ob/ob mice without affecting insulin or adiponectin levels [60]. CTRP3 concentrations are also elevated in diabetic individuals [61].

Given that HIIT and CT in the current study led to improvements in body composition, lipid profile, and glycemic control, it is plausible that the reductions in CTRP1 and CTRP3 are also associated with these positive changes. The correlation analysis results showed that CTRP- 1 and CTRP- 3, both adipokines involved in metabolic regulation, showed significant correlations with several metabolic markers. CTRP- 1 was positively correlated with HOMA-IR ($r = 0.417$, $p < 0.05$) and AIP ($r = 0.48$, $p < 0.01$), suggesting that higher levels of CTRP-1 are associated with greater insulin resistance and cardiovascular risk.

Aminotransferases

The findings of this study indicate a significant decrease in AST and ALT levels in the HIIT and CT groups. Consistent with these results, Hemmatinafar et al. (2020) reported reductions in AST and ALT levels after eight weeks of HIIT in overweight men [62]. Similarly, Piralaiy et al. (2024) observed significant improvements in AST and ALT following 12 weeks of CT in overweight and obese girls [63]. Studies indicate that both AT and RT can reduce AST and ALT levels by decreasing liver fat content, visceral fat content, and IR [64–66]. Some studies have shown that moderate-to-high intensity AT has the highest potential for reducing visceral fat in overweight and obese individuals [67, 68], while RT can improve insulin function, blood glucose control, and fat oxidation and storage in muscles [69]. The reduction in liver fat content and visceral fat is mediated by increased fatty acid oxidation and improved insulin sensitivity. Exercise

training enhances the capacity of the liver to metabolize fatty acids, reducing the accumulation of fat in hepatocytes. Additionally, the reduction in insulin resistance leads to better regulation of lipid metabolism in the liver, further contributing to improved liver enzyme levels [70]. Therefore, both HIIT and CT effectively reduced liver fat content, visceral fat, and IR, which, in turn, led to decreases in AST and ALT levels. Both AST and ALT, markers of liver function, showed negative correlations with BMI ($r = -0.649$, $p < 0.01$ for AST; $r = -0.682$, $p < 0.01$ for ALT) and VO2 max ($r = -0.711$, $p < 0.01$ for BMI; $r = -0.521$, $p < 0.01$ for HOMA-IR). This suggests that improvements in liver enzyme levels are associated with reductions in body fat and improvements in aerobic capacity, further supporting the role of exercise in improving liver health and metabolic function.

Maximum strength and oxygen consumption

The findings of the present study indicate a significant increase in 1RM in the CT group. Moreover, $VO_{2\ max}$ showed a significant increase in both the HIIT and CT groups. In line with the current study, Namboonlue et al. (2023) observed a significant increase in maximum strength following five weeks of CT in obese individuals [71]. Also, Khammassi et al. (2018) reported a significant increase in $VO_{2\ max}$ in obese individuals after 12 weeks of HIIT [46]. RT increases muscular strength due to neuromuscular adaptations, increased muscle cross-sectional area, and changes in connective tissue stiffness [72]. Additionally, the improvement in $VO_{2\ max}$ resulting from HIIT may be attributed to enhanced cardiac performance, enhanced mitochondrial density, and improved capillary density in skeletal muscle [73]. Furthermore, aerobic exercise enhances the activity of aerobic enzymes, intramuscular glycogen, mitochondrial density, and capillary density in muscles [74]. It can be concluded that engaging in CT leads to neuromuscular and cardiovascular adaptations that improve strength and $VO_{2\ max}$. On the other hand, while HIIT contributed to improved $VO_{2\ max}$, it did not have a significant effect on maximum strength, highlighting the importance of incorporating combined training to achieve comprehensive adaptations. HOMA-IR showed a significant negative correlation with VO2 max ($r = -0.521$, $p < 0.01$), indicating that improvements in aerobic capacity are associated with enhanced insulin sensitivity. This aligns with the known benefits of aerobic exercise on glucose metabolism and insulin signaling. Figure 4 illustrates how exercise training modulate changes in CTRPs concentration in overweight and obese women.

Strengths and limitations

This study's strengths include its randomized design, the inclusion of two distinct exercise modalities, focus on adipokines, adherence to consort giudlines and the comprehensive assessment of various health parameters. However, the study is limited by its relatively small sample size and short duration, which may not fully capture long-term effects. Additionally, the absence of dietary monitoring, no follow up period and male participants restricts the generalizability of the findings across genders and under different nutritional conditions.

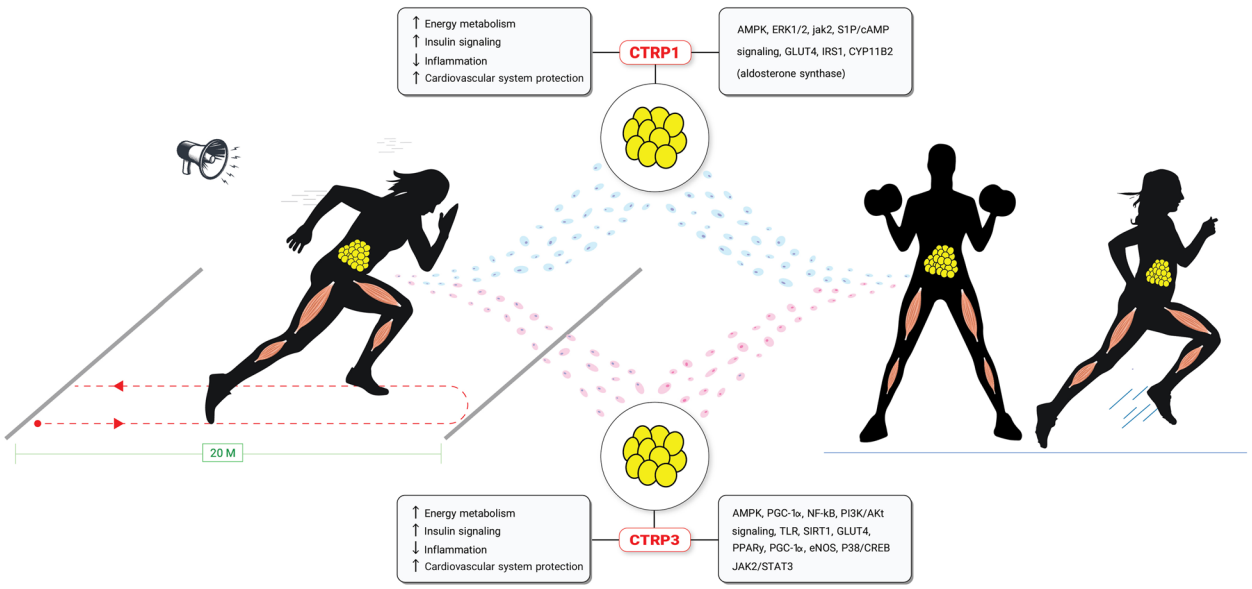

Fig. 4 The effects of exercise training on the regulation of CTRP1 and CTRP3. ↑ = increase; ↓ = decrease

Conclusions

Overall, the findings of this study indicated that both HIIT and CT protocols significantly improved body composition, lipid profile, glycemic components, and liver enzymes in overweight and obese women. Thus, both types of exercise can be effective methods for managing obesity and enhancing general health in women. While neither protocol showed clear superiority over the other, significant improvements in strength were observed only in the CT group. Furthermore, the results showed that HIIT and CT had a significant impact on serum adipokines, including CTRP1 and CTRP3 levels.Furthermore, HIIT can be recommended as a time-efficient exercise modality for individuals with limited time availability. The reduction in Atherogenic Index of Plasma (AIP) and improvements in lipid profile suggest that these exercise modalities can help mitigate cardiovascular risk factors. The reduction in CTRP1 and CTRP3 levels after eight weeks of training suggests a positive influence of these exercises on metabolic status and reduced risk factors associated with metabolic diseases. Due to the limitations of the small sample size, gender, and dietary control, as well as the limited training period, it is recommended that studies be conducted with longer periods, dietary control, and gender diversity.

Abbreviations

CT	Combined aerobic and resistance training
1RM	One repetition maximum
ACSM	American College of Sports Medicine
AIP	Atherogenic Index of Plasma
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
AT	Aerobic training
BFP	Body fat percentage
BMI	Body mass index
CTRP1	C1Q/TNF-related protein- 1
CTRP3	C1Q/TNF-related protein- 3
CTRPs	C1Q/TNF-related proteins
CVD	Cardiovascular diseases
FBS	Fasting blood glucose
HDL	High-density lipoprotein
HIIT	High-intensity interval training
HOMA-IR	Homeostasis model assessment of insulin resistance
HRR	Heart rate reserve
HTN	Hypertension
IR	Insulin resistance
IS	Insulin sensitivity
LDL	Low-density lipoprotein
MAV	Maximal aerobic velocity
MetS	Metabolic syndrome
MHR	Maximum heart rate
QUICKI	Quantitative insulin sensitivity check index
RHR	Resting heart rate
RT	Resistance training
T2D	Type 2 diabetes
TC	Total cholesterol
TG	Triglyceride
THR	Target heart rate
$VO_{2\,max}$	Maximum oxygen consumption
η_p^2	Partial eta-squared

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13102-025-01123-4.

Supplementary Material 1.

Acknowledgements
The authors would like to sincerely thank all participants for their invaluable contributions to this study.

Authors' contributions
F.S. and A.M. wrote the main manuscript text. All authors reviewed the manuscript.

Funding
This research did not receive any external funding.

Data availability
All data generated or analyzed during this study are included in this manuscript file.

Declarations

Ethics approval and consent to participate
This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. All participants provided written informed consent after being briefed on the research methods and objectives. The study was approved by the Research Ethics Committee of Razi University of Kermanshah (IR.RAZI.REC.1403.033) on Jun 23, 2023.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Sport Physiology, Faculty of Sport Sciences, Razi University, Kermanshah, Iran.

Received: 26 November 2024 Accepted: 19 March 2025
Published online: 08 April 2025

References

1. Pourfarzi F, Sadjadi A, Poustchi H, Amani F. Prevalence of overweight and obesity in Iranian population: A population-based study in northwestern of Iran. J Public Health Res. 2021;11(1):2475.
2. Garvey W. Clinical Definition of Overweight and Obesity: Evaluation and Management of Adiposity, Adiposopathy and Related Diseases. 2019. p. 121–43.
3. Ruban A, Stoenchev K, Ashrafian H, Teare J. Current treatments for obesity. Clin Med (Lond). 2019;19(3):205–12.
4. Wiechert M, Holzapfel C. Nutrition concepts for the treatment of obesity in adults. Nutrients. 2021;14(1):169.
5. Stephenson J, Smith CM, Kearns B, Haywood A, Bissell P. The association between obesity and quality of life: a retrospective analysis of a large-scale population-based cohort study. BMC Public Health. 2021;21(1):1990.
6. Zhang JS, Yeh WC, Tsai YW, Chen JY. The relationship between atherogenic index of plasma and obesity among adults in Taiwan. Int J Environ Res Public Health. 2022;19(22):14864.
7. Zhang X, Zhang X, Li X, Feng J, Chen X. Association of metabolic syndrome with atherogenic index of plasma in an urban Chinese population: A 15-year prospective study. Nutr Metab Cardiovasc Dis. 2019;29(11):1214–9.
8. Nakamura K, Fuster JJ, Walsh K. Adipokines: a link between obesity and cardiovascular disease. J Cardiol. 2014;63(4):250–9.

9. Clemente-Suárez VJ, Redondo-Flórez L, Beltrán-Velasco AI, Martín-Rodríguez A, Martínez-Guardado I, Navarro-Jiménez E, et al. The role of adipokines in health and disease. Biomedicines. 2023;11(5):1290.
10. Lee CY, Lee CH, Tsai S, Huang CT, Wu MT, Tai SY, et al. Association between serum leptin and adiponectin levels with risk of insulin resistance and impaired glucose tolerance in non-diabetic women. Kaohsiung J Med Sci. 2009;25(3):116–25.
11. Rodriguez S, Lei X, Petersen PS, Tan SY, Little HC, Wong GW. Loss of CTRP1 disrupts glucose and lipid homeostasis. Am J Physiol Endocrinol Metab. 2016;311(4):E678–97.
12. Xin Y, Lyu X, Wang C, Fu Y, Zhang S, Tian C, et al. Elevated circulating levels of CTRP1, a novel adipokine, in diabetic patients. Endocr J. 2014;61(9):841–7.
13. Shabani P, Emamgholipour S, Doosti M. CTRP1 in Liver Disease. Adv Clin Chem. 2017;79:1–23.
14. Moradi N, Fadaei R, Khamseh ME, Nobakht A, Rezaei MJ, Aliakbary F, et al. Serum levels of CTRP3 in diabetic nephropathy and its relationship with insulin resistance and kidney function. PLoS ONE. 2019;14(4):e0215617.
15. Seldin MM, Tan SY, Wong GW. Metabolic function of the CTRP family of hormones. Rev Endocr Metab Disord. 2014;15(2):111–23.
16. Schäffler A, Buechler C. CTRP family: linking immunity to metabolism. Trends Endocrinol Metab. 2012;23(4):194–204.
17. Hasegawa N, Fujie S, Horii N, Uchida M, Kurihara T, Sanada K, et al. Aerobic exercise training-induced changes in serum C1q/TNF-related protein levels are associated with reduced arterial stiffness in middle-aged and older adults. Am J Physiol Regul Integr Comp Physiol. 2018;314(1):R94-r101.
18. Mirzendedel Z, Attarzadeh Hosseini S, Bijeh N, Raouf Saeb AA. A comparison of the effects of twelve weeks combined training with different ordering on CTRP3, TNF-α, IL6 and Insulin resistance in women with type 2 diabetes. Iran J Endocrinol Metab. 2019;21(4):203–16.
19. Hashida R, Kawaguchi T, Bekki M, Omoto M, Matsuse H, Nago T, et al. Aerobic vs. resistance exercise in non-alcoholic fatty liver disease: A systematic review. J Hepatol. 2017;66(1):142–52.
20. Wilson JM, Marin PJ, Rhea MR, Wilson SM, Loenneke JP, Anderson JC. Concurrent training: a meta-analysis examining interference of aerobic and resistance exercises. J Strength Cond Res. 2012;26(8):2293–307.
21. Bouamra M, Zouhal H, Ratel S, Makhlouf I, Bezrati I, Chtara M, et al. Concurrent Training Promotes Greater Gains on Body Composition and Components of Physical Fitness Than Single-Mode Training (Endurance or Resistance) in Youth With Obesity. Front Physiol. 2022;13:869063.
22. Ho SS, Dhaliwal SS, Hills AP, Pal S. The effect of 12 weeks of aerobic, resistance or combination exercise training on cardiovascular risk factors in the overweight and obese in a randomized trial. BMC Public Health. 2012;12:704.
23. Wewege M, van den Berg R, Ward RE, Keech A. The effects of high-intensity interval training vs. moderate-intensity continuous training on body composition in overweight and obese adults: a systematic review and meta-analysis. Obes Rev. 2017;18(6):635–46.
24. Maillard F, Pereira B, Boisseau N. Effect of High-Intensity Interval Training on Total, Abdominal and Visceral Fat Mass: A Meta-Analysis. Sports Med. 2018;48(2):269–88.
25. Tong TK, Zhang H, Shi H, Liu Y, Ai J, Nie J, et al. Comparing Time efficiency of sprint vs. high-intensity interval training in reducing abdominal visceral fat in obese young women: a randomized. Controlled Trial Front Physiol. 2018;9:1048.
26. Jelleyman C, Yates T, O'Donovan G, Gray LJ, King JA, Khunti K, Davies MJ. The effects of high-intensity interval training on glucose regulation and insulin resistance: a meta-analysis. Obes Rev. 2015;16(11):942–61.
27. Batacan RB Jr, Duncan MJ, Dalbo VJ, Tucker PS, Fenning AS. Effects of high-intensity interval training on cardiometabolic health: a systematic review and meta-analysis of intervention studies. Br J Sports Med. 2017;51(6):494–503. https://doi.org/10.1136/bjsports-2015-095841. 2016. PMID: 27797726.
28. Dobiásová M, Frohlich J. The plasma parameter log (TG/HDL-C) as an atherogenic index: correlation with lipoprotein particle size and esterification rate in apoB-lipoprotein-depleted plasma (FER(HDL)). Clin Biochem. 2001;34(7):583–8.
29. Nasiri M, Monazzami A, Alavimilani S, et al. Modulation of hormonal, metabolic, inflammatory and oxidative stress biomarkers in women with polycystic ovary syndrome following combined (resistant and endurance) training: a randomized controlled trail. BMC Endocr Disord. 2025;25:1. https://doi.org/10.1186/s12902-024-01793-0.
30. Varmazyar I, Monazzami AA, Moradi M, McAinch AJ. Effects of 12-weeks resistance training and vitamin E supplementation on aminotransferases, CTRP-2, and CTRP-9 levels in males with nonalcoholic fatty liver disease: a double-blind, randomized trial. BMC Sports Sci Med Rehabil. 2024;16(1):185.
31. Medicine ACoS. ACSM's health-related physical fitness assessment manual: Lippincott Williams & Wilkins; 2013.
32. Placzkowska S, Pawlik-Sobecka L, Kokot I, Piwowar A. Indirect insulin resistance detection: Current clinical trends and laboratory limitations. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2019;163(3):187–99.
33. Ismaiel A, Ciobanu OS, Ismaiel M, Leucuta DC, Popa SL, David L, et al. Atherogenic index of plasma in non-alcoholic fatty liver disease: systematic review and meta-analysis. Biomedicines. 2022;10(9):2101.
34. Adachi H, Matsumura A, Enomoto M, Fukami A, Morikawa N, Nohara Y, et al. A J-shaped Relationship between Sleep Duration and the Risk of Insulin Resistance in a General Japanese Population. Intern Med. 2023;62(4):511–7.
35. Sheikholeslami-Vatani D, Siahkouhian M, Hakimi M, Ali-Mohammadi M. The effect of concurrent training order on hormonal responses and body composition in obese men. Sci Sports. 2015;30(6):335–41.
36. Mayorga-Vega D, Aguilar-Soto P, Viciana J. Criterion-Related Validity of the 20-M Shuttle Run Test for Estimating Cardiorespiratory Fitness: A Meta-Analysis. J Sports Sci Med. 2015;14(3):536–47.
37. Monazzami A, Momenpur R, Alipour E, Yari K, Payandeh M. Effects of Eight-Week Combined Resistance and Endurance Training on Salivary Interleukin-12, Tumor Necrosis Factor, Cortisol, and Testosterone Levels in Patients with Breast Cancer. Int J Cancer Manag. 2021;14(2):e109039. https://doi.org/10.5812/ijcm.109039.
38. Rohnejad B, Monazzami A. Effects of high-intensity intermittent training on some inflammatory and muscle damage indices in overweight middle-aged men. Apunts Sports Medicine. 2023;58:100404.
39. Monazzami A, Momenpour R, Alipoor E , Yari K, Payandeh M. The Effects of Concurrent Training on the Body Composition, Quality of Life, and Sleep Quality of Postmenopausal Women with Breast Cancer. J Kermanshah Univ Med Sci. 2020;24(3):e101186. https://doi.org/10.5812/jkums.101186.
40. Astinchap A, Monazzami A, Fereidoonfara K, Rahimi Z, Rahimi M. Modulation of Fibroblast Growth Factor-21 and βklotho Proteins Expression in Type 2 Diabetic Women with Non-alcoholic Fatty Liver Disease Following Endurance and Strength Training. Hepat Mon. 21(7):e116513. https://doi.org/10.5812/hepatmon.116513.
41. Wood T, Maddalozzo G, Harter R. Accuracy of Seven Equations for Predicting 1RM Performance of Apparently Healthy, Sedentary Older Adults. Meas Phys Educ Exerc Sci. 2002;6:67–94.
42. Kraemer WJ, Fleck SJ, Deschenes MR. Exercise physiology: integrating theory and application: Lippincott Williams & Wilkins; 2011.
43. Talebi N, Asadi M. Comparison of the Effect of Three Methods of Endurance, Resistance and Concurrent Training on Aerobic Fitness and Body Composition of Overweight Non-Athlete Male Students. New Approaches in Exercise Physiology. 2021;3(6):153–64.
44. Gonzalez-Gil AM, Elizondo-Montemayor L. The role of exercise in the interplay between myokines, hepatokines, osteokines, adipokines, and modulation of inflammation for energy substrate redistribution and fat mass loss: a review. Nutrients. 2020;112(6):1899.
45. Rejeki PS, Pranoto A, Rahmanto I, Izzatunnisa N, Yosika GF, Hernaningsih Y, et al. The positive effect of four-week combined aerobic-resistance training on body composition and adipokine levels in obese females. Sports (Basel). 2023;11(4):90.
46. Khammassi M, Ouerghi N, Hadj-Taieb S, Feki M, Thivel D, Bouassida A. Impact of a 12-week high-intensity interval training without caloric restriction on body composition and lipid profile in sedentary healthy overweight/obese youth. J Exerc Rehabil. 2018;14(1):118–25.
47. Rossi FE, Fortaleza AC, Neves LM, Buonani C, Picolo MR, Diniz TA, et al. Combined Training (Aerobic Plus Strength) Potentiates a Reduction in Body Fat but Demonstrates No Difference on the Lipid Profile in Postmenopausal Women When Compared With Aerobic Training With a Similar Training Load. J Strength Cond Res. 2016;30(1):226–34.

48. Whayne TF Jr. Atherosclerosis: current status of prevention and treatment. Int J Angiol. 2011;20(4):213–22.
49. Salehi Z, Salehi K, Moeini M, Kargarfard M, Sadeghi M. The Effect of Resistance Exercise on Lipid Profile of Coronary Artery Disease Patients: A Randomized Clinical Trial. Iran J Nurs Midwifery Res. 2017;22(2):112–6.
50. Buzdagli Y, Tekin A, Eyipinar CD, Öget F, Siktar E. The effect of different types of exercise on blood lipid profiles: A meta-analysis of randomized controlled studies. Sci Sports. 2022;37(8):675–87.
51. Ahmad AM, Mahmoud AM, Serry ZH, Mohamed MM, Abd Elghaffar HA. Effects of low-versus high-volume high-intensity interval training on glycemic control and quality of life in obese women with type 2 diabetes. A randomized controlled trial. J Exerc Sci Fit. 2023;21(4):395–404.
52. Sigal RJ, Kenny GP, Boulé NG, Wells GA, Prud'homme D, Fortier M, et al. Effects of aerobic training, resistance training, or both on glycemic control in type 2 diabetes: a randomized trial. Ann Intern Med. 2007;147(6):357–69.
53. de Oliveira Teles G, da Silva CS, Rezende VR, Rebelo ACS. Acute effects of high-intensity interval training on diabetes mellitus: a systematic review. Int J Environ Res Public Health. 2022;19(12):7049.
54. Maiorana A, O'Driscoll G, Goodman C, Taylor R, Green D. Combined aerobic and resistance exercise improves glycemic control and fitness in type 2 diabetes. Diabetes Res Clin Pract. 2002;56(2):115–23.
55. Masoumzadeh S, Jalali Dehkordi K, Kargarfard M. Effects of High Intensity Interval Training (HIIT) On CTRP1 and CTRP3 in Women with Type 2 Diabetes. Iranian Journal of Diabetes and Lipid Disorders. 2021;21(1):24–38.
56. Choi HY, Park JW, Lee N, Hwang SY, Cho GJ, Hong HC, et al. Effects of a combined aerobic and resistance exercise program on C1q/TNF-related protein-3 (CTRP-3) and CTRP-5 levels. Diabetes Care. 2013;36(10):3321–7.
57. Peterson JM, Aja S, Wei Z, Wong GW. CTRP1 protein enhances fatty acid oxidation via AMP-activated protein kinase (AMPK) activation and acetyl-CoA carboxylase (ACC) inhibition. J Biol Chem. 2012;287(2):1576–87.
58. Chalupova L, Zakovska A, Adamcova K. Development of a novel enzyme-linked immunosorbent assay (ELISA) for measurement of serum CTRP1: a pilot study: measurement of serum CTRP1 in healthy donors and patients with metabolic syndrome. Clin Biochem. 2013;46(1–2):73–8.
59. Shen L, Wang S, Ling Y, Liang W. Association of C1q/TNF-related protein-1 (CTRP1) serum levels with coronary artery disease. J Int Med Res. 2019;47(6):2571–9.
60. Peterson JM, Wei Z, Wong GW. C1q/TNF-related protein-3 (CTRP3), a novel adipokine that regulates hepatic glucose output. J Biol Chem. 2010;285(51):39691–701.
61. Choi KM, Hwang SY, Hong HC, Yang SJ, Choi HY, Yoo HJ, et al. C1q/TNF-related protein-3 (CTRP-3) and pigment epithelium-derived factor (PEDF) concentrations in patients with type 2 diabetes and metabolic syndrome. Diabetes. 2012;61(11):2932–6.
62. Hemmatinafar A, Fathi M, Ziaaldini MM. Effect of 8 weeks of HIIT on hepatic enzyme levels, lipid profile and body composition in overweight young men. Obesity Medicine. 2020;18:100233.
63. Piralaiy ED, Nikoukheslat S, Arbabi F, Rahbar S. The effect of 12 weeks of high intensity functional training and combined aerobic-resistance training on the levels of AST, ALT, AST/ALT and body composition indices in overweight and obese girls. J Appl Health Stud Sport Physiol. 2024;11(1):261–77.
64. Hejazi K, Hackett D. Effect of exercise on liver function and insulin resistance markers in patients with non-alcoholic fatty liver disease: a systematic review and meta-analysis of randomized controlled trials. J Clin Med. 2023;12(8):3011.
65. Glass OK, Radia A, Kraus WE, Abdelmalek MF. Exercise Training as Treatment of Nonalcoholic Fatty Liver Disease. Journal of Functional Morphology and Kinesiology. 2017;2(4):35.
66. Hong F, Liu Y, Lebaka VR, Mohammed A, Ye W, Chen B, et al. Effect of Exercise Training on Serum Transaminases in Patients With Nonalcoholic Fatty Liver Disease: A Systematic Review and Meta-Analysis. Front Physiol. 2022;13:894044.
67. Hong HR, Jeong JO, Kong JY, Lee SH, Yang SH, Ha CD, et al. Effect of walking exercise on abdominal fat, insulin resistance and serum cytokines in obese women. J Exerc Nutrition Biochem. 2014;18(3):277–85.
68. Recchia F, Leung CK, Yu AP, Leung W, Yu DJ, Fong DY, et al. Dose-response effects of exercise and caloric restriction on visceral adiposity in overweight and obese adults: a systematic review and meta-analysis of randomised controlled trials. Br J Sports Med. 2023;57(16):1035–41.
69. Winters-Stone KM, Kenfield SA, Van Blarigan EL, Moe EL, Ramsdill JW, Daniel K, et al. Effect of Increasing Levels of Web-Based Behavioral Support on Changes in Physical Activity, Diet, and Symptoms in Men With Prostate Cancer: Protocol for a Randomized Controlled Trial. JMIR Res Protoc. 2018;7(11):e11257.
70. Thyfault JP, Bergouignan A. Exercise and metabolic health: beyond skeletal muscle. Diabetologia. 2020;63(8):1464–74. https://doi.org/10.1007/s00125-020-05177-6.
71. Namboonlue C, Namboonlue S, Sriwiset P, Jaisuk J, Buttichak A, Muangritdech N, Saengjan W. Effects of Concurrent Resistance and Aerobic Training on Body Composition, Muscular Strength and Maximum Oxygen Uptake in Men with Excess Weight. Teor metod fiz vihov. 2023;23(3):389–96.
72. Knuttgen H, Kraemer W. Terminology and Measurement in Exercise Performance. The Journal of Strength & Conditioning Research. 1987;1:1–10.
73. Astorino TA, Allen RP, Roberson DW, Jurancich M. Effect of high-intensity interval training on cardiovascular function, VO2max, and muscular force. J Strength Cond Res. 2012;26(1):138–45.
74. Chtara M, Chamari K, Chaouachi M, Chaouachi A, Koubaa D, Feki Y, et al. Effects of intra-session concurrent endurance and strength training sequence on aerobic performance and capacity. Br J Sports Med. 2005;39(8):555–60.

Publisher's Note