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# **Original Article**

# The Effects of Endurance and Resistance Training on Systemic Inflammatory Markers and Metabolic Syndrome Parameters in Overweight and Obese Men

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#### Abstract

**Introduction:** The purpose of this study was to investigate the effects of endurance and resistance training on systemic inflammatory markers and metabolic syndrome parameters in overweight and obese men.

**Methods:** 33 volunteer participants (BMI=27.39 kg/m<sup>2</sup>) were randomly divided into three groups (n=11), namely, an endurance training (ET) group, a resistance training (RT) group, and a control group. The ET and RT groups trained for eight weeks at three sessions a week and 150 min per week. Before the training and 72 hours after the last exercise session, blood samples were collected from the subjects for assays on interleukin-1 $\beta$  (IL-1 $\beta$ ), IL-6, tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), adiponectin, intracellular adhesion molecule-1 (ICAM-1), high-sensitivity C-reactive protein (hs-CRP), glucose, insulin, and blood lipids and lipoproteins. The percentage body fat, waist circumference, WHR, and blood pressure of the subjects were also measured before and after the training protocols. Analysis of variance (ANOVA) with repeated measures test and significant level of 0.05 was used for all statistical analysis.

**Results:** After training, the ET and RT groups showed a significant decrease in hs-CRP, percentage body fat, insulin and insulin resistance index, triglycerides, and total cholesterol (P<0.05). Both groups likewise showed a significant increase in adiponectin (P<0.05). Only the changes in LDL-C and TNF- $\alpha$  were significant between the ET and RT groups (P<0.05).

**Conclusion:** In general, the effects of resistance training on some of the systemic inflammatory markers and metabolic syndrome parameters of the overweight and obese men were incomparable to those of endurance training.

Keywords: Training, Systemic Inflammation, Metabolic Syndrome, Obesity

#### Introduction

Overweight and obesity lead to more than 2.8 million deaths every year worldwide, thereby increasing public health costs and reducing quality of life (1). These conditions have adverse metabolic effects on blood pressure, cholesterol and triglyceride levels, and insulin resistance (1) and is one of the most important risk factors for cardiovascular disease, metabolic syndrome (MetS), arthritis, and type II diabetes (2, 3). Obese individuals have also been shown to exhibit high blood markers of systemic inflammation (4, 5). Increased levels

of inflammatory markers, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), are associated with chronic diseases, including coronary artery disease, diabetes, and cancer (6). MetS, which occurs with visceral obesity, dyslipidemia, hyperglycemia, and arterial hypertension, has become one of the most severe public health challenges worldwide (7). Its incidence increases with the prevalence of obesity (8), elevates the risk of cardiovascular diseases, and reduces longevity (9). MetS is affected by adiponectin, which is an adipokine that is specifically expressed and secreted from adipose tissue and is directly responsible for the body's sensitivity to insulin (9, 10). The relationship between adiponectin and MetS has been investigated in a growing number of studies (7, 10, 11). Another symptom associated with MetS is chronic low-grade inflammation, which can be reduced through physical activities, such as endurance training (ET) and resistance training (RT), as nonpharmacological interventions (6). Research on endurance training (ET), particularly the dynamic form, found a positive effect on the risk factors associated with MetS (12). Although investigations usually focus on ET intervention, RT seems to be an effective and cost-effective somewhat measure for preventing and treating cardiovascular disease (13). Some studies indicated that resistance training (RT) improves MetS (14), as evidenced by the 34 % lower incidence of the disorder among individuals undergoing the intervention (15). Other studies provided no confirmation of such effect (16), thus highlighting the need for further research into the influence of RT on MetS parameters (12). Exercise training partly affects inflammation and MetS by reducing fat mass, but it also improves inflammation independent of body composition (17). Other studies suggested that the reduction in weight-related inflammation markers is independent of exercise training (18, 19). Although significant evidence supports the role of physical activity in inflammatory biomarkers reducing and improving MetS parameters, further study is still needed on what type of exercise activity offers the best benefits. The physiological effects of ET and the inflammatory adaptation associated with it differ from those of RT. Most studies used the effects of ET on cytokine levels as the most important inflammatory marker (20); less evidence is available regarding the effects of RT on such markers (21) and MetS parameters (14). Motivated by these issues, the current research investigated the effects of ET and RT on the

systemic inflammatory markers and MetS parameters of overweight and obese men.

### Methods

A total of 33 overweight and obese men (body mass index  $[BMI] > 25 \text{ kg/m}^2$ ), who were nonsmokers and non-alcoholics and had no history of chronic disease, initially volunteered for participation in the study. It should be noted that the subjects of the present research did not have a history of participation in a regular exercise program one year before to study began. The subjects were randomly divided into three groups (n = 11 each), namely, an ET group, an RT group, and a control group (CON). Twelve subjects of the present study had a BMI>30 kg/m<sup>2</sup> and 19 had a BMI > 25. Then, it was necessary to homogenize the three group's subjects according to BMI. The Committee on Ethics for Research of the Kurdistan University of Medical Sciences approved the study protocols. During the whole training protocol, the control group was told to do their daily routine activities and avoid spontaneous physical activity. All of the subjects were college students and used similar dormitory meals and lived in similar condition. Percentage body fat was determined measurements skinfold (Harpenden via Skinfold Fat Caliper, Baty, UK) of pectoral, abdominal, and femoral points and was estimated using the Jackson and Pollock (1978) equations (22). Waist circumference was measured using an ergonomic circumference measuring tape (Seca 201, Humborg, Germany). Before the training protocol and two days after the last training session, the resting blood pressure of the subjects were measured using а sphygmomanometer (UM-102A/ STA, A & D Medical, UK) positioned on the left hand after 20 minutes of sitting. The maximum oxygen consumption (VO<sub>2max</sub>) of the subjects was measured using the Balke treadmill protocol (RUN700, TechnoGym, Italy) (23) and a breath-by-breath gas analyzer (Metalyzer 3B, Cortex Biophysic, Germany). The point at

which the minute ventilation (VE)/VO2 ratio did not increase in proportion to the VE/carbon dioxide production (VCO2) or the point at which the VE increased non-linearly was regarded as the first ventilatory threshold (VT1); the point at which both the VE/VO2 and VE/VCO2 increased non-linearly was considered the second ventilatory threshold (VT2) (25). Before the training was commenced, two sessions were devoted to having the subjects acclimatize themselves to the equipment and the correct way of performing the exercises. One-repetition maximum (1 RM) tests were conducted for all the subjects. The training groups performed ET and RT protocols for eight weeks at three sessions per week and 150 min per week. The RT group did leg presses, leg extensions, leg curls, bench presses, lateral pulldowns, lateral raises, triceps pushdowns, arm curls, and basic abdominal crunches. The training program started with one set of the aforementioned exercise movements as a warm-up (40 %- 50 % 1 RM for eight repetitions), which lasted for 10 minutes. The first four weeks (weeks 1-4) of training involved three sets with 10 maximal repetitions (10 RM) and 60 seconds of rest between sets; the next four weeks (weeks 5-8) consisted of three sets with eight maximal repetitions and 90 seconds of rest between sets (25). The 1RM of the subjects were re-measured each week, and the training program was adjusted on the basis of probable new records. The subjects were asked to perform stretching exercises during the training sessions to maintain flexibility. The ET group warmed up for 10 minutes with moderate running (45 %- 50 % VO<sub>2max</sub>), after which they proceeded with the training for about 150 minutes per week in accordance with the intensity and duration levels listed in Table 1. The subjects' ventilatory thresholds were re-measured every two weeks, and the training program was adjusted on the basis of probable new records. Each ET session was completed with 10 minutes of moderate voluntary running and stretching for cooling

down. Before the training, blood samples were collected in a laboratory at 8-10 am after the subjects went on 12-hour overnight fasting. Forty-eight hours after the last training session (26), fasting blood samples were again taken under the same pre-training conditions. At each blood sampling phase, 10 mL of blood was drawn from the antecubital vein of the left hand and transferred to a tube containing ethylenediaminetetraacetic acid. To isolate the plasma and serum from the cells, the samples were placed at room temperature for 10 to 15 minutes. After isolating plasma, blood samples centrifuged for 10 minutes at 2500 g and 4°C. The serum was then poured into 0.5 mL micro tubes, and the samples were frozen at -80°C until analysis. Note that the laboratory technicians were unaware of the groupings of subjects. Measurements of serum the intracellular adhesion molecule-1 (ICAM-1), IL-6, IL-1 $\beta$ , TNF- $\alpha$ , and adiponectin were performed using Quantikine enzyme-linked immunosorbent assay (ELISA) kits (Biotechne, Minneapolis, USA) with sensitivities of 0.254 ng.mL<sup>-1</sup>, 1 pg.mL<sup>-1</sup>, 0.7 pg.mL<sup>-1</sup>, 5/5  $pg.mL^{-1}$ , and 0.891  $ng.mL^{-1}$ , respectively. Serum high-sensitivity CRP (hs-CRP) was measured using a colorimetric sandwich ELISA assay kit (Monobind Inc, USA) with a sensitivity of 0.014 µg.mL<sup>-1</sup>. Plasma blood glucose was measured using an EnzyChrom<sup>™</sup> assay kit (BioAssay Systems, USA), and plasma insulin was determined using an immunoradiometric assay kit (Cisbio Bioassays, France). All the measurements were performed duplicate and according to the manufacturers' instructions. In all the cases, the intraassay and interassay coefficients were below 7 %. Insulin resistance was measured via the homeostasis model assessment of insulin resistance (HOMA-IR) (27). This index was measured on the basis of fasting insulin and glucose levels using the following formula: Fasting insulin ( $\mu$ U.mL<sup>-1</sup>) × Fasting glucose  $(mmol.L^{-1})/22.5$ . Plasma glucose concentration was obtained in mg.dL<sup>-1</sup>, which was converted into  $mmol.L^{-1}$  by multiplying

the former value by 18.02. Kolomogorov– Smirnov and Leven's tests were carried out to determine the normality of distribution and homogeneity of variances, respectively. Because the distribution of all the variables was normal and homogeneity of variance was found, a two-way repeated-measures analysis of variance (ANOVA) (time  $\times$  condition) and Bonferroni correction were implemented to examine between-group differences. When such differences and/or interaction-significant differences were found, Bonferroni's post-hoc test was applied for pairwise comparisons. One-way ANOVA was performed to compare the variables' baseline values. A Student's ttest was conducted to compare the pre- and post-training values of each group. All the data analyses were conducted using SPSS 21.0 (Statistics IBM SPSS, Inc., Chicago, IL, USA). The significance level was set at 0.05, and the data were expressed as mean  $\pm$ SD in all the cases.

Table 1. ET training protocol.	Table	1.	ET	training	protocol.
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<b>Endurance Training Protocol</b>	1-4wk	5-8wk
Running 10-15 bpm below VT1 (min)	20	-
Running at VT 1(min)	20	20
Running 10-20 bpm above VT1(min)	10	20
Running at VT2(min)	-	10
Total training time (min)	~50	~50

VT: Ventilatory Threshold, bpm: breathe per minute

#### Results

The baseline means and standard deviations of the variables (pre-test) are shown in Tables 2-4. The baseline values of the variables were similar across the randomized groups, with no significant differences among them. The comparison of pre- and post-training values showed a significant decrease in body mass and BMI and a significant increase in VO2max in the ET and RT groups (Table 2). The main results on the variables are presented in the MetS parameter and inflammatory biomarker columns in Tables 3 and 4, respectively. MetS parameters: percentage body fat, waist circumference, waist-to-hip ratio (WHR), blood lipids and lipoproteins, blood pressure and insulin resistance were measured in this study as MetS parameters (32, 33). The two-way repeated-measures ANOVA showed a significant difference in percentage body fat among the groups (P =0.009). After training protocol, a significant decrease in percentage body fat was observed in the ET (from 30.41 % to 23.66 %, P = 0.001) and RT (from 29.48 % to 24.98 %, P = 0.001) groups. Only the ET group showed a

significant decrease in waist circumference (from 98.9 to 92.6 cm, P = 0.037) and WHR (from 0.97 to 0.91, P = 0.041). Blood glucose levels significantly decreased in the ET (from 7.00 to 6.6 mmol.L<sup>-1</sup>, P = 0.013) and CON  $(6.92 \text{ to } 6.22 \text{ mmol.L}^{-1}, P = 0.035)$  groups. Despite a decrease of about 10% in the blood glucose concentrations of the RT group, the reduction was not significant. Although no significant difference in insulin levels was found among the groups, a significant decrease was found in the ET (from 8.39 to 6.4 µU.mL<sup>-</sup> <sup>1</sup>, P = 0.001) and RT (from 7.74 to 6.89  $\mu$ U.mL<sup>-1</sup>, P = 0.013) groups. The ET, RT, and CON groups exhibited significant decreases of 38 %, 21 %, and 16% in HOMA-IR, respectively. However, systolic blood pressure (SBP) decreased significantly in the ET group (from 132 to 123 mmHg, P = 0.011); no significant differences in SBP were found in the RT group, and no significant differences in diastolic blood pressure (DBP) were found whiten and between the ET and RT groups (P > 0.05). The ET group showed a significant decrease of 24.6% in low-density lipoprotein cholesterol (LDL-C). The results of the

Bonferroni post-hoc test indicated a significant difference in LDL-C between the ET and RT groups (P = 0.012).

	Data are presented as Mean ±SD.						
Variable Grou		After 8wk training	Change (%)	P (pre- to post training)	P (between groups)		
Percentage Endura	ance $30.41 \pm 2.35$		-22.19	0.001*			
body fat Resista			-15.33	0.001*	0.009*		
(%) contr	col $29.78 \pm 3.22$	$30.02 \pm 2.14$	0.80	0.747			
Waist Endura	ance $98.5 \pm 23.1$	$92.6\pm27.3$	-5.98	0.037*			
Circumsta Resista	ance $102.4 \pm 31.8$	$97.9\pm20.7$	-4.39	0.089	0.134		
nce(cm) contr	rol $97.6 \pm 25.6$	$98.1 \pm 24.3$	0.51	0.835			
Endura	ance $0.97 \pm 0.13$	$0.91 \pm 0.10$	-6.18	0.041*			
WHR Resista	ance $0.99 \pm 0.12$	$0.95 \pm 0.13$	-4.04	0.064	0.054		
contr	col $0.97 \pm 0.09$	$0.98 \pm 0.11$	1.03	0.963			
Endur	rance $7.00 \pm 1.22$	$6.06 \pm 0.73$	-13.43	0.013*			
$\begin{array}{c} \text{Glucose} \\ \text{(mmol. } \text{L}^{-1} \text{)} \end{array} \xrightarrow{\text{Elitude}} \\ \end{array}$	tance $6.40 \pm 1.34$	$5.75 \pm 0.61$	-10.16	0.185	0.290		
(mmol. L) cont	trol $6.92 \pm 1.01$	$6.22 \pm 0.85$	-10.12	0.035*			
Endur	rance $8.39 \pm 1.29$	$6.04 \pm 0.83$	-28.01	0.001*			
Insulin (	tance $7.74 \pm 1.12$	$6.89 \pm 0.66$	-10.98	0.013*	0.063		
$(\text{qU.mL}^{-1})$ Resist	trol $8.50 \pm 1.64$	$7.87 \pm 1.24$	-7.41	0.136			
Endu	rance $2.63 \pm 0.71$	$1.62 \pm 0.26$	-38.40	0.001*			
HOMA-IR Resist	tance $2.24 \pm 0.70$	$1.76 \pm 0.23$	-21.43	0.041*	0.115		
cont	trol $2.60 \pm 0.55$	$2.19 \pm 0.47$	-15.77	0.007*			
Endur		$123 \pm 12$	-6.81	0.011*			
SBP Resist	tance $134 \pm 12$	$133 \pm 10$	-0.74	0.262	0.065		
(mmHg) cont	trol $128\pm 9$	$127 \pm 13$	0.76	0.313			
Endur	rance $85 \pm 9$	$84 \pm 10$	-1.17	0.089			
DBP Resist		87± 7	0.00	0.546	0.431		
(mmHg) (mmHg)		$85\pm9$	1.19	0.264			
LDL-c Endur		3.34± 2.52	-24.60	0.002*†			
$(\text{mmol.L}^{-1})$ Resist	tance $4.39 \pm 1.83$	$4.28 \pm 1.68$	-2.50	0.068	0.043*		
(mmol.L) cont	trol $4.21 \pm 2.35$	$4.23\pm\ 2.14$	0.47	0.478			
Endu	rance $0.97 \pm 0.75$	$1.28 \pm 0.83$	-31.95	0.010*			
HDL-c Resist	tance $1.03 \pm 0.64$	$1.22\pm 0.79$	-18.44	0.063	0.001*		
(mmol.L <sup>-1</sup> ) Kesist	trol $1.10 \pm 0.78$	$1.12\pm 0.88$	1.81	0.940			
Endu		$1.86 \pm 0.65$	-34.50	0.001*			
IG (mmol.L Design		$2.13 \pm 0.44$	-23.92	0.037*	0.014*		
$\frac{1}{cont}$		$2.92 \pm 0.69$	0.68	0.538			
Endu		3.46± 1.43	-38.21	0.009*			
I otal Colst. Pagint		$3.44 \pm 1.61$	-37.34	0.004*	0.001*		
(mmol.L <sup>-1</sup> ) Kesist		$5.27 \pm 2.02$	2.92	0.180			

**Table 3.** Metabolic syndrome parameters before and after the training in ET, RT and CON groups.Data are presented as Mean ±SD.

\*=significant difference between pre- and post- exercise values.  $\dagger$ =significant difference between the short and long RI resistance exercise sessions. Significance set at p < 0.05 for all comparisons.

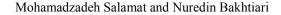
characteri	istic	Baseline	After 8wk training	Change (%)	P (pre- to posttest)	P (between groups)
Age	Endurance	$21.90 \pm 1.75$				0.768 <sup>a</sup>
(year)	Resistance	$23.16\pm2.09$				
	control	$21.85\pm2.05$				
Height	Endurance	$171.87\pm5.90$				0.801 <sup>a</sup>
(cm)	Resistance	$173.65\pm4.16$				
	control	$174.61\pm3.89$				
Body	Endurance	$81.90\pm5.50$	$75.25\pm4.39$	-8.11	0.001*	0.031* <sup>b</sup>
mass	Resistance	$81.20\pm4.20$	$78.33 \pm 5.29$	-3.53	0.008*	
(kg)	control	$83.80\pm4.45$	$83.97 \pm 4.81$	-0.01	0.725	
BMI	Endurance	$27.73 \pm 1.42$	$25.49 \pm 1.32$	-8.07	0.001*	0.170 <sup>b</sup>
(kg/m2)	Resistance	$26.93 \pm 1.12$	$25.98 \pm 1.67$	-3.52	0.008*	
	control	$27.50 \pm 1.55$	$27.54 \pm 1.46$	0.14	0.778	
Vo <sub>2max</sub>	Endurance	$31.64\pm2.34$	$39.83\pm2.28$	25.88	0.001*	
(ml.kg <sup>-</sup>	Resistance	$31.60 \pm 1.30$	$38.59\pm2.03$	22.12	0.001*	0.031* <sup>b</sup>
$^{1}.min^{-1}$ )	control	$33.56 \pm 2.43$	$33.24\pm2.46$	-0.95	0.551	

**Table 2.** Subjects characteristics before and after the training in ET, RT and CON groups. Data are presented as Mean ±SD.

a: a one-way ANOVA was run to determine baseline values differences between the ET, RT and CON groups. b: a tow-way repeated measures ANOVA was run to determine baseline and after training differences between the ET, RT and CON groups.

The ET and RT groups showed a significant increase of about 32% and a non-significant increase of about 18% in high-density lipoprotein cholesterol (HDL-C), respectively. Both ET and RT groups exhibited a significant decrease in triglyceride levels (35% and 23% respectively) and total cholesterol (38% and 37%, respectively) (P < 0.05). However, there were no significant differences in this regard between the groups (P > 0.05). The mean $\pm$ SD, percentage of changes, and between- and within-group P values of the MetS parameters are presented in Table 3. Inflammatory biomarkers: No significant difference in ICAM-1 concentrations was found before and after training and among the groups (P > 0.05), but the highest reduction was found in the ET group (about 7%, from 223.64 to 208.13 pg.mL<sup>-1</sup>). A significant difference in IL-1 $\beta$  and TNF- $\alpha$  was found among the groups (P = 0.001 and P = 0.024, respectively); that is, the ET and CON groups showed a significant

difference in IL-1 $\beta$  (P = 0.001), and the ET and RT groups exhibited a significant difference in TNF- $\alpha$  (P = 0.001). The results indicated a significant difference in hs-CRP among the groups (P = 0.021). Although no significant difference was found between the ET and RT groups, they showed a remarkably significant decrease of about 63% and 50% in hs-CRP, respectively. A significant difference in adiponectin concentration was found among the groups (P = 0.026), specifically between the ET and CON groups and between the RT and CON groups. No significant difference in this regard was found between the ET and RT groups. The comparison of the pre- and posttraining values reflected a significant increase in the ET (4.42 to 7.67 pg.mL<sup>-1</sup>, P = 0.003) and RT (4.37 to 7.17 pg.mL<sup>-1</sup>, P = 0.001) groups. The mean±SD, percentage of changes, and between- and within-group P values of the inflammatory biomarkers are presented in Table 4.



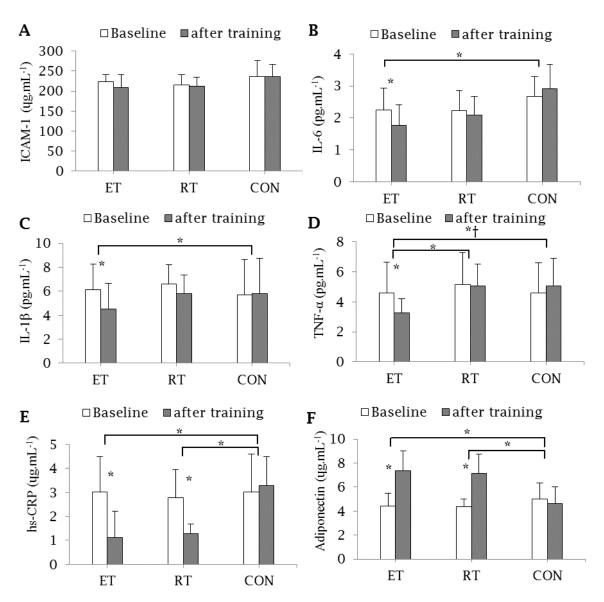


Fig 1. Effects of Endurance and Resistance Training on Systemic Inflammatory Markers in Overweight and Obese Men. Serum concentration of ICAM-1 (A) showed no significant differences between the baseline and after training values and neither no significant differences between the ET, RT and CON (P>0.05). After training, significant decrease was found in IL-6 (B) and IL-1 $\beta$  (C) only in ET group (P=0.001 and P=0.003, respectively) TNF- $\alpha$  serum concentration (D) was significantly decreased after ET training (P=0.037) and significant difference was seen between the ET and RT groups (P=0.029). Furthermore, baseline and after training comparison of hs-CRP values (E) showed a significant decreases in ET (P=0.001) and RT (P=0.006) groups. Also, after the training protocols, significant decrement was showed in adiponectin serum concentrations (F) in ET (P=0.003) and RT (P=0.001) groups. significant differences was seen between the ET and CON groups in case of IL-6, IL-1 $\beta$ , TNF- $\alpha$ , hs-CRP and adiponectin and also between the RT and CON in hs-CRP and adiponectin. Other comparisons didn't showed any significant changes.

Data are presented as means  $\pm$  SD. \*=significant difference at p < 0.05.  $\dagger$ =significant difference at p < 0.01.

#### Discussion

Eight weeks of training significantly decreased body mass and percentage body fat and significantly increased VO2max in the endurance and resistance training groups. These results indicated that both the training interventions were of sufficient intensity and duration to produce physiological changes. Obesity is considered one of the major challenges for human health. In relation to this disorder, studies showed that adipose tissue

can produce several proinflammatory cytokines, thus indicating that the amount of fat mass is associated with inflammatory marker values. Inflammatory markers are higher than normal levels in obese individuals (5). The baseline inflammatory marker values of the subjects in the present study were higher than normal values-an indication of some degree of chronic low-grade inflammation, as reported in the literature (28). According to the literature, our study's subjects display some symptoms of MetS (8), high percentages body fat and waist circumferences, high-normal blood pressure (29), and high levels of glucose, lipids and blood lipoproteins (30). Generally, the results of the current work showed that waist circumference, WHR, blood glucose, SBP, LDL-C, IL-1β, IL-6, and TNF-α significantly changed only in the ET group. Percentage body fat, insulin concentration, HOMA-IR, HDL-C, triglyceride levels, total hs-CRP. cholesterol, and adiponectin significantly changed in both the ET and RT groups. However, neither of these groups showed significant changes in ICAM-1 and DBP. Taken together, the findings showed that the effects of RT on some of the systemic inflammatory markers and MetS parameters of the overweight and obese men were comparable to those of ET. The results also showed that hs-CRP was significantly reduced by the ET and RT interventions. The secretion of this acute phase protein from the liver is regulated by IL-6 and TNF- $\alpha$  and other cytokines (31). Researchers showed that exercise training can reduce baseline CRP conservations (34). In one study, four weeks of regular intense exercise training improved CRP and adiponectin in patients with impaired glucose tolerance and type 2 diabetes but not in healthy subjects (32). Similar results were obtained in the present research. Note, however, that our study subjects were healthy with high percentages of body fat. As mentioned earlier, obese people often have high levels of systemic inflammatory markers (4, 5). Exercise training was found to be

effective only for people with high CRP (33), although a significant decrease was observed in healthy elderly people (34). The subjects in the present study also had CRP values that were relatively higher than normal values. The effects of physical training mechanisms on inflammatory symptoms are generally unclear, but such training may contribute to the reduction of fat mass (4). Nicklas et al. (2004) showed that changes in IL-6 and CRP were independent of BMI changes (18), and Fisher et al. (2011) indicated that ET and RT did not have an independent effect on systemic inflammation (35). Regular physical activities are also associated with the reduction of adhesion molecules (36). Changes in adhesion molecules likely affect part of the positive effects of exercise training on inflammatory markers. The secretion of adhesion molecules from vascular endothelia can be regulated by proinflammatory cytokines in the blood (37). In the present study, despite the significant reduction in IL-6, IL-1 $\beta$ , and TNF- $\alpha$  in the ET group, no significant changes in ICAM-1 were observed. Insulin resistance is one of the most important parameters of MetS. Although the mechanisms of physical activities on insulin sensitivity are insufficiently understood, such activities may increase the active uptake of skeletal muscle glucose through a process that is regulated by the transfer of glucose transporter type 4 (GLUT4) to the plasma membrane and T tubules. The mechanism that underlies insulin sensitivity improvement associated with exercise can be related to some factors such as IL-6, adiponectin, glycogen synthesis/content, fiber type, and oxidative capacity (38). In the present study, reduced HOMA-IR was observed in all the groups. Insulin resistance is believed to be associated with obesity and BMI (39), whereas RTinduced improvement in insulin sensitivity is attributed to an increase in fat-free mass (40). Lindegaard et al. (2008) demonstrated that RT increases insulin-dependent glucose uptake (41). An interesting finding showed that circuit ET can improve insulin sensitivity,

independent of changing body weight, WHR, and BMI (42). Another factor associated with insulin resistance is TNF- $\alpha$ . In line with the results of Smart (2011), our findings showed a significant effect of ET on TNF- $\alpha$  values (43). In contrast with the results of the present study, Prestes et al. (2009) discovered that despite a significant decrease in the plasma concentration of IL-6, leptin, and restein, the concentration of IL-15 and TNF- $\alpha$  did not significantly change (44). The current research found a significant decrease in IL-1 $\beta$  in the ET group; IL-1 $\beta$  has been shown to reduce insulin sensitivity (45). Therefore, the reduction in IL-1ß may contribute to the decrease in insulin resistance after exercise. The CON group showed a significant decrease in fasting glucose, which also led to a decrease in HOMA-IR in this group. This finding can be ascribed to the subjects' possible stresses and factors other psychological that they experience. Serum levels of IL-6 in the fasting state are directly correlated to other insulin resistance parameters, including fasting insulin levels, HOMA-IR, and WHR indexes, whereas adiponectin is inversely associated with these parameters. Evidence suggested that adiponectin plays a role in energy hemostasis and the metabolism of fatty acids. High levels of IL-6 production consistent with low levels of adiponectin produced from adipose tissue may contribute to obesity-induced insulin resistance by increasing plasma fatty acids (41). Adiponectin has also been shown to affect HDL-C concentrations partly through its anti-atherosclerotic properties (46). After the ET and RT interventions in the current work, adiponectin levels significantly increased and HDL-C values decreased by 32% and 18% in the ET and RT groups, respectively. A significant decrease in HOMA-IR was also found in the ET, RT, and CON groups after training. At least part of the decrease in insulin resistance can be attributed to the significant increase in adiponectin. Because adiponectin directly sensitizes the body to insulin (7, 10), it plays a role in insulin resistance and, thereby, MetS. The percentages body fat of the subjects significantly decreased after ET and RT. In line with the association between obesity, especially visceral obesity, and systemic inflammation, body fat decrements may reduce levels of pre-inflammatory cytokines, particularly IL-6. IL-6 can also affect the liver cells for the increased production of CRP. A decrease in body fat mass, which was observed in the present study, can therefore be responsible for the reduction in some inflammatory markers. SBP, which is a MetS parameter, significantly decreased in the ET group.

#### Conclusion

In general, the results indicated that ET and RT can improve some of the MetS parameters and inflammatory markers in overweight and obese individuals. Although ET has had some advantages over RT in some factors, significant differences were only found in terms of LDL-C and TNF-α. Suggesting that, as with the implementation of ET, the use of RT can effectively improve the parameters of MetS and inflammatory markers in overweight and obese individuals. However, future research is needed to determine the optimal intensity and duration of endurance training and resistance training regimens in overweight and obese individuals with metabolic syndrome and low grade systemic inflammation.

# Ethical issues

No applicable.

#### **Authors' contributions**

Khalid Mohamadzadeh Salamat: Conception or design of the work, data collection, Drafted manuscript, performed Data analysis and interpretation and revised manuscript.

Nuredin Bakhtiari: Performed experiments and Interpreted results of experiments, Critical revision of the article and Final approval of the version to be published,

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