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Single and Concurrent Effects of Endurance and Resistance Training on Plasma Visfatin, Insulin, Glucose and Insulin Resistance of Non-Athlete Men with Obesity

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ABSTRACT

The purpose of the present study was to investigate the effect of endurance (ET), resistance (RT) and concurrent training (CT) on plasma levels of visfatin, insulin, glucose and insulin resistance of non-athlete men with Obesity. It was a semi-experimental study. Thirty six men [age: 21.48 (0.25), and BF%: 27.39 (0.52)], voluntarily participated in this study after public announcement in university. Main inclusion and exclusion criteria was healthy (no physical illness and inability), obesity [based on WHO's definition body fat percentage (BF%) of over 25] and non-athlete (without regular training during week). They were randomly divided into three groups (n=12) for ET, RT and CT. For 8 weeks (3sessions/week), the candidates participated in ET (25-40 min at 65-85% of maximum heart rate), RT (5exercises, 6sets, intensity: 50-80% of one repetition maximum, volumes: 5, 8 and 12repetitions) and CT (one or a half-term ET and then RT with 3 sets). Blood samples were taken 48 h before the first training session and 48 h after the last training session. The BF% in ET was significantly less than that in RT ($p<0.01$), and in CT, it was less than that for both ET and RT ($p<0.01$). Plasma visfatin only, in CT was significantly less than that in RT ($p<0.01$). Plasma insulin levels in CT were significantly higher than that in ET and RT ($p<0.01$). Plasma glucose levels in CT were less than that in ET and RT, significantly ($p<0.01$). Insulin resistance only in CT was less than that in ET significantly ($p<0.01$). In general, the present study showed that maybe, CT have more effect on the body composition, glucose metabolism and insulin resistance adjustment, which can be effective in preventing obesity and adjusting adipocytokines such as visfatin.

KEY WORDS: ***Endurance Training, Resistance Training, Visfatin, Insulin Resistance, Obesity.***

INTRODUCTION

Visfatin is a adipocytokine protein with multiple functions and may act as an intermediary of autocrine, paracrine and endocrine that has different functions such as cell proliferation, nicotinamide synthesis and glucose homeostasis (1). It is mainly secreted from subcutaneous adipose tissue (2) and is involved in the pathogenesis of insulin resistance (3). Such as insulin, Visfatin has semi-insulin function and high affinity for the insulin receptor

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(4). Studies have shown an increase in visfatin with diabetes mellitus (5, 6). Although, visfatin is linked to insulin receptors and has semi-insulin activity, researchers reported that its role in systematic biosynthesis of NAD+ (essential for the function of B cells of the pancreas), helps to regulate glucose homeostasis (7). Although, visfatin is linked to insulin receptors, the link is in a separate location from insulin and by binding to these receptors, it causes reduction in glucose release from the liver and stimulates glucose uptake in adipose cells and muscles (8). Plasma concentration of visfatin is increased in obese subjects (9, 10) and those with type 2 diabetes (11). According to reports, an increase in insulin levels may be a compensatory mechanism for defects in insulin action (12). Inhibitory effect of insulin on the secretion of visfatin is well declared in the literature (5, 13).

In other side, Hyperglycemia causes increasing plasma concentrations of visfatin and there is a negative feedback mechanism of insulin on stimulated visfatin release by glucose (5). Intravenous and also oral glucose intake has different effects on visfatin serum (5, 13). Oral glucose intake during glucose tolerance test causes a rapid reduction in visfatin (13). There is a theory that hyper-insulin during glucose tolerance test may likely suppress visfatin (3). Researchers reported that hyper-insulin results in a significant reduction in visfatin but lipid injection into a vein results in a significant increase in visfatin (3). In addition, increased serum levels of visfatin under insulin resistance conditions are positively correlated with the body weight (3).

But in general, the pathogenesis of insulin resistance in humans is complex, so confirming a simple relationship between visfatin and insulin resistance is difficult (14). It is likely that visfatin ratio to circulating insulin is more important than levels of insulin circulation alone to prevent the development of insulin resistance and metabolic syndrome in obese patients (14). A reduction in ratio of circulating visfatin to insulin in metabolic syndrome, a negative relationship between visfatin ratio to insulin and anthropometric parameters of visfatin are responsible for insulin resistance in obese individuals with metabolic syndrome (14). But, the ratio is higher in healthy obese individuals as compared to obese individuals with metabolic syndrome (14).

Weight loss after exercise program causes reduced serum levels of visfatin (15) and this reinforces the theory that reduced visfatin is associated with improved insulin sensitivity (3). Much evidence show that physical activity is associated with reduced cardiovascular diseases which cause mortality in the general population, and in patients with type 2 diabetes mellitus. In fact, meta-analysis studies showed that exercise under supervision is effective in improving cardio respiratory fitness and also in controlling blood sugar levels and other cardiovascular risk factors (16). Further evidence support the importance of aerobic exercise in treating patients with type 2 diabetes mellitus, improved blood sugar control, body composition and cardiovascular risk factors (17). Poehlman *et al.* (2000) investigated the effect of resistance and endurance training on insulin sensitivity in young women. They showed that both endurance and resistance training improved glucose clearance in young women with different mechanisms (18). Patients with type 2 diabetes mellitus may also have additional benefits of the control of blood sugar, body composition and cardiovascular risk factors with combined aerobic and resistance training. So many organizations recommend both endurance and resistance training for all adults, including those with type 2 diabetes mellitus (19). It has been reported that a combination of aerobic and resistance training is more effective on controlling blood sugar than either type of training alone (20).

The combination of resistance and aerobic training has been established as the more effective training for the control of blood glucose, insulin action and reform cardiovascular risk factors (21). Considering the intensity, a study showed that low-moderate intensity training is more effective than moderate-high intensity training (22), while another study reported that high-intensity training is more effective in improving blood sugar control (23).

Since visfatin is secreted from visceral adipose tissue, exercise, due to its effects on the reduction of visceral adipose tissue of the body and as a result of improvement of some adipokines, can also be effective in reducing

serum visfatin (3). Some studies have examined the effect of aerobic training on visfatin. In this regard, Haus *et al.* (2009) observed a reduction in plasma visfatin due to 12 weeks of aerobic training in obese men and women (24). Mohammadi *et al.* (2010) also stated that 8 weeks of endurance training caused reduction in plasma visfatin (25). In contrast, McKenzie *et al.* (2008) observed increased plasma visfatin after 6 months of aerobic training at 70% of maximum heart rate in healthy individuals and patients with impaired glucose (26). In addition, the relationship between visfatin and insulin resistance index is unknown. Some studies did not find any relationship between insulin resistance and visfatin (27, 28), while in a study, Lee *et al.* (2010) observed that doing 12 weeks aerobic training significantly reduced visfatin and improved insulin resistance index in obese women (29). On the other hand, no change in the level of plasma visfatin have been reported after aerobic training (30).

There are also conflicting results on the effect of resistance and combined training. Seo *et al.* (2011) in a study reported the effect of 12 weeks of combined training [3 sets of 10 repetition maximum (10RM) resistance exercise as well as aerobic exercise at an intensity of 60-70% of their heart rate reserve (HRR)] on visfatin and factors of metabolic syndrome in middle-aged obese women. They observed a significant reduction in visfatin which was associated with reduced fasting glucose (31). Ghanbari-Niaki *et al.* (2010) showed that doing a rapid session of anaerobic running training (7 sets of 10-s running of 35 x 6 m with one minute rest between sets) in 60 young men with high physical fitness was associated with a significant increase in plasma visfatin levels and insulin, blood glucose concentration and insulin resistance index immediately after training (32). A study showed that eight weeks of circuit resistance training with 60 to 70% of one repetition maximum significantly reduced the levels of visfatin, and this change was associated with weight loss and body fat percentage (33). In contrast, Jorge *et al.* (34) showed that visfatin levels in response to 12 weeks of resistance training was significantly increased, whereas insulin resistance remained unchanged.

This inconsistency in research results can be due to various factors such as fat content and its distribution, inflammatory conditions, hormones and other factors which include the type and intensity of exercise done. Given that all the three methods of training: resistance, endurance and combined training affect improvement in cardiovascular risk factors, up till now, no study has investigated the comparison of these three methods on traditional and new risk factors for heart disease and determined which training method is more effective in improving these factors, therefore, there is a need to conduct more researches to better understand the factors influencing the synthesis and release of visfatin and clarify its role. o Simultaneous effect of aerobic, resistance and combined training on levels of glucose, visfatin and insulin resistance index has not been investigated. So, the purpose of the present study was to compare 8 weeks of endurance, resistance and combined training on levels of plasma visfatin, insulin, glucose, and insulin resistance in young non-athlete men with obesity.

MATERIALS AND METHODS

The study was conducted in accordance with the policy statement of the Declaration of the Iranian Ministry of Health and approved by the research ethics committee of the Iranian Sport Sciences Research Institute.

Participants. After the announcement and invitation to participate in this study, subjects with full knowledge of the time, place, manner of conducting the test and its objectives, voluntarily participated, then an informed consent was collected and a medical physical health certificate was received for the exercise, and then based on height, weight, body fat percentage and BMI, they were divided into three equal groups of 12 members of endurance, resistance and combined training as simple random. Main inclusion and exclusion criteria was healthy (no physical illness and inability), obesity [based on WHO's definition body fat percentage (BF%) of over 25] and non-athlete (without regular training during week). During the study, 1 member of the endurance training group and 3 members of the resistance training group were removed. In order to control subjects' diet nutritional status, questionnaire was also

used and in order to meet the same dietary model, subjects were provided with some recommendations by the researcher.

Data Collection. In order to measure the weight and height, scales and stadiometer SECA Model made in Germany were used with precision of 0.1 kg and 0.1 mm. In order to determine and calculate the percentage of body fat, caliper device Yagami Japan was used and the three-point method of chest, abdomen and thigh, and Pollock-Jackson formula were used.

Variables. Independent variable was type of training (endurance, resistance and concurrent training). Dependent variables were weight, BMI, BF%, levels of plasma visfatin, insulin, and glucose, and insulin resistance.

Training Protocol. Subjects participated in training of 3 sessions per week for 8 weeks. Endurance training group participated in a running program and the duration and intensity of each participant in the training were gradually increased at the end of each step. In the first and second weeks, subjects had an activity for 25 min at 65% maximum heart rate (HRmax). During the third to fifth weeks, they had an activity for 35 min with 65 to 75% (HRmax) and during the sixth to 8th weeks, they had an activity for 40 min with 75 to 85% HRmax. Beurer rate monitor was used to control training intensity. In the resistance training group, the first one repetition maximum of subjects was determined and they did their training in accordance with Table 1. Also, in order to apply the main overload, one repetition maximum of subjects was re-calculated every two weeks. In the concurrent training group, both endurance and resistance training was done in a session so that first resistance training and then endurance training program was implemented, with the condition that for the sake of training volume, in endurance training, the time was half and in resistance training, time sets were halved (60 and 50% of the final set, and one set of 80% was removed).

Table 1. Resistance Training Characteristics

Exercise	Characteristics						
Leg Press	Intensity (1RM)	50%	60%	70%	80%	80%	50%
	Volume (r)	8	8	8	5	5	8
Knee Extension	Intensity (1RM)	50%	60%	70%	80%	80%	50%
	Volume (r)	8	8	8	8	8	8
Lat Pulldown	Intensity (1RM)	50%	60%	70%	80%	80%	50%
	Volume (r)	12	12	12	12	12	12
Biceps Curls	Intensity (1RM)	50%	60%	70%	80%	80%	50%
	Volume (r)	12	12	12	12	8	12
Dead Lift	Intensity (1RM)	50%	60%	70%	80%	80%	50%
	Volume (r)	5	5	5	3	3	5

1RM: One Repeat Maximum. r: Repetition

Blood Sampling and Measurements. Before starting the first week of training and 48 h after the last session of training, blood samples were taken. Five mL of venous blood was taken from forearm vein of each subject in the sitting position by a laboratory specialist and put in sterile tubes containing anticoagulant EDTA. Then, it was centrifuged for 10 min at a speed of 3000-3500 rpm and the plasma obtained was put in 1 ml micro tubes, in order to implement the next steps, and it was stored at -80°C. Plasma visfatin was measured by EIA kit (SIGMA-ALDRICH, USA; CN: RAB0377) with measurement sensitivity of 0.778 ng/mL, plasma insulin was measured by human kit of ELISA (Multispecies Specificity, Japan: CN: RSCYK060R) and blood fasting glucose was measured using Iranian kits purchased from MAN, Iran Co. by glucose oxidase method. Insulin resistance was calculated by the following equation:

HOMA-IR = [Insulin (mU/L) × Glucose (mmol/L)] ÷ 22.5

Statistical Analysis. For descriptive statistics, mean and standard error of the mean were used to determine normal data distribution, and Kolmogorov-Smirnov test and analysis of

covariance (ANCOVA) were used to compare the three groups of data. All data were analyzed using software SPSS, and a significance level of 0.05 was considered.

RESULTS

The participants in this study were 36 healthy non-athlete men, aged 21.48 ± 1.46 years and with height of 173.27 ± 5.47 (cm).

The difference in weight changes in obese non-athlete men in the groups was not significant after 8 weeks of training (F= 1.14, p= 0.335). But the difference in changes in BMI and BF% were significant [(F= 21.1, p= 0.001, respectively) and (F= 15.13, p= 0.001), respectively] (Table 2), and after 8 weeks, BMI in obese non-athlete men involved in endurance training was significantly less than that in resistance training (p= 0.001) and that in concurrent training was lower than that in both endurance and resistance training [(p= 0.005) and (p= 0.001), respectively]. Also, BF% in obese non-athlete men after 8 weeks of endurance training was significantly less than that in resistance training (p= 0.01), and in concurrent training, it was lower than that in both endurance and resistance training [(p= 0.01) and (p= 0.001), respectively] (Table 3).

Table 2. Analysis of Covariance statistic of training groups

Variable	Group	Time	Mean ± SE	Adjusted Mean ± SE	ANCOVA statistic	
					F	p
Weight[a] (kg)	ET	preTest	90.55 ± 3.6	86.49 ± 0.13	1.14	0.335
		postTest	87.5 ± 3.7			
	RT	preTest	92.22 ± 5.3	86.53 ± 0.14		
		postTest	89.17 ± 5.1			
	CT	preTest	86.61 ± 3.7	86.27 ± 0.12		
		postTest	83.46 ± 3.5			
BMI[b] (kg/m^2)	ET	preTest	29.86 ± 1.3	28.69 ± 0.038	21.1	0.001**
		postTest	28.7 ± 1.2			
	RT	preTest	31.48 ±1.7	28.89 ± 0.041		
		postTest	30.44 ± 1.6			
	CT	preTest	28.64 ± 0.9	28.53 ± 0.035		
		postTest	27.38 ± 0.9			
BF[c] (%)	ET	preTest	26.9 ± 1.01	24.68 ± 0.33	15.13	0.001**
		postTest	24.12 ± 1.1			
	RT	preTest	28.21 ± 0.5	26.04 ± 0.35		
		postTest	27.21 ± 1.2			
	CT	preTest	27.05 ± 0.7	23.47 ± 0.3		
		postTest	23.07 ± 0.9			

BMI: Body Mass Index. **BF:** Body Fat. **ET:** Endurance Training. **RT:** Resistance Training. **CT:** Concurrent Training. **a:** Covariates appearing in the model are evaluated at the following values: weight.pre = 89.5097. **b:** Covariates appearing in the model are evaluated at the following values: BMI.pre = 29.8581. **c:** Covariates appearing in the model are evaluated at the following values: BF.pre = 27.3481. ****:** significant level at p<0.01.

Table 3. Pairwise Comparison based on ANCOVA statistic

Variable	Groups Comparison	Mean Difference	p
BMI (kg/m^2)	ET-RT	- 0.198 ± 0.056	0.001**
	ET-CT	0.159 ± 0.052	0.005**
	RT-CT	0.357 ± 0.055	0.001**
BF (%)	ET-RT	- 1.359 ± 0.49	0.01**
	ET-CT	1.212 ± 0.45	0.01**
	RT-CT	2.571 ± 0.47	0.001**

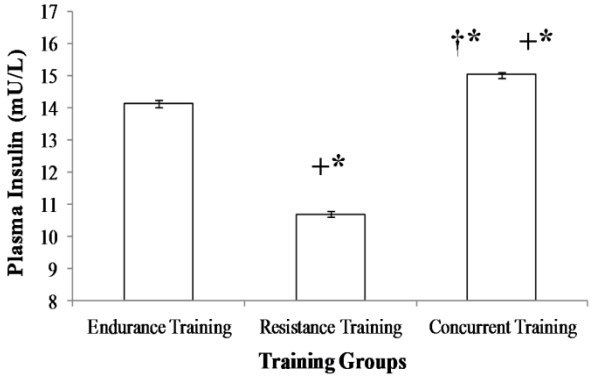
BMI: Body Mass Index. **BF:** Body Fat. **ET:** Endurance Training. **RT:** Resistance Training. **CT:** Concurrent Training. ****:** significant level at p<0.01.

The difference in plasma visfatin changes in obese non-athlete men in the groups after 8 weeks of training was statistically significant (F= 6.54, p= 0.005); and the difference in the changes only after 8 weeks of training was significant in concurrent and resistance training (mean difference= 2.066 ± 0.6, p= 0.004), but other differences between endurance and resistance training, and endurance with concurrent training were not significant [(p= 0.405) and (p= 0.136), respectively] (Figures 1).

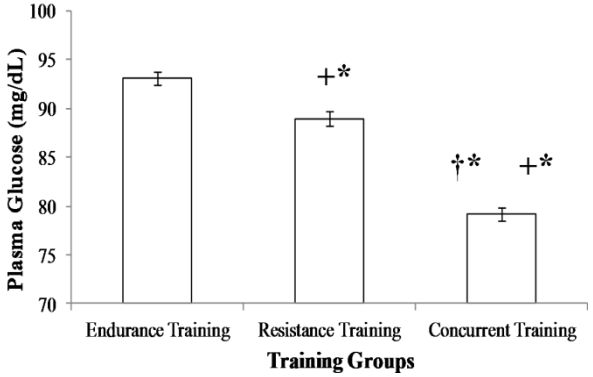
Figures 1. *Plasma Visfatin of non-athlete men with obesity*. †*: different from Resistance Training group at p=0.004. Adjusted Mean ± SE: Covariates appearing in the model are evaluated at the following values: Visfatin.Pre = 10.9125.

The difference in changes in plasma insulin was significant (F= 461.91, p= 0.001); and after 8 weeks, plasma insulin in obese non-athlete men involved in endurance training was significantly higher than that in resistance training (mean difference= 3.43 ± 0.15, p= 0.001) and that in concurrent training was higher than that for both endurance and resistance training [(mean difference= 0.9 ± 0.16, p= 0.001), respectively and (mean difference= 4.33 ± 0.15, p= 0.001)] (Figures 2).

The difference in changes in plasma glucose was also significant (F= 107.79, p= 0.001); and after 8 weeks, plasma glucose in obese non-athlete men involved in resistance training was significantly less than that in endurance training (mean difference= 4.11 ± 1.03, p= 0.001) and that in concurrent training was higher than that for both endurance and resistance training [(mean difference= 13.9 ± 0.9, p= 0.001), respectively and (Mean Difference= 9.8 ± 1.02, p= 0.001)] (Figures 3).



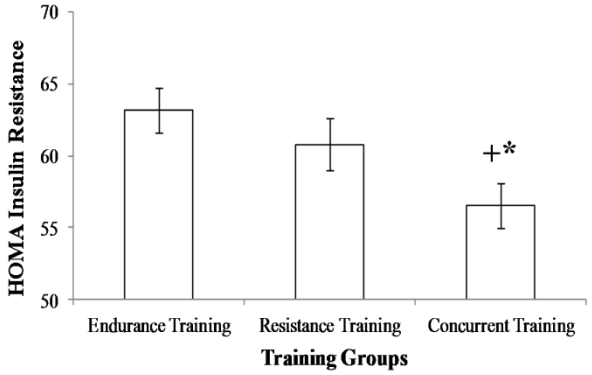
Figures 2. *Plasma Insulin of non-athlete men with obesity*. +*: different from Endurance Training group at p=0.001. †*: different from Resistance Training group at p=0.001. Adjusted Mean ± SE: Covariates appearing in the model are evaluated at the following values: insulin.pre = 15.3988.



Figures 3. *Plasma Glucose of non-athlete men with obesity*. +*: different from Endurance Training group at p=0.001. †*: different from Resistance Training group at p=0.001. Adjusted Mean ± SE: Covariates appearing in the model are evaluated at the following values: Glucose.Pre = 106.7288.

The difference in changes in insulin resistance was also significant (F= 4.58, p= 0.019); and after 8 weeks, only insulin resistance in obese non-athlete men involved in concurrent training was significantly less than that of endurance training (mean difference= 6.68 ± 2.22, p= 0.016), but other differences between endurance and resistance training, and resistance

and concurrent training were not significant [(p= 0.93) and (p= 0.27), respectively] (Figures 4).



Figures 4. Insulin Resistance of non-athlete *men with obesity*. +*: different from Endurance Training group at p=0.016. Adjusted Mean ± SE: Covariates appearing in the model are evaluated at the following values: Insulin Resistance.Pre = 65.3281.

DISCUSSION

Visfatin is known as an adipokine in visceral adipose tissue of human and mice and also its plasma levels are increased by obesity and insulin resistance, indicating its important role in insulin resistance. It has been shown that it has insulin-like function that can be linked to insulin receptors and as a result, causes reduction of blood glucose levels (32).

The results of the present study showed that the plasma visfatin levels were significantly different in obese inactive individuals after 8 weeks of training with different protocols of resistance, endurance and concurrent training. Visfatin levels in concurrent training group were significantly lower than that in resistance training group. But no significant difference was observed between concurrent and endurance training groups as well as between endurance and resistance training groups. Previous studies have shown that plasma visfatin level is significantly correlated with visceral fat mass, weight and body mass index, in this regard, Fukuhara *et al*. (8) showed that visfatin serum level is associated with visceral adipose tissue; in another study, a positive relationship was reported between plasma visfatin concentration and indices of body composition and body fat percentage (35). Brandt *et al*. (2005)(35) also pointed out a positive relationship between plasma visfatin and body fat percentage. So that, serum visfatin concentration was higher in obese subjects as compared to thin ones and a significant reduction in plasma visfatin level was observed by weight loss after stomach surgery (36). The findings of the present study also showed that after 8 weeks, BMI in obese non-athletic men involved in endurance training was significantly lower than that in resistance training, and in concurrent training, it was lower than that in both endurance and resistance training. Also, body fat percentage in obese non-athletic men after 8 weeks of endurance training was significantly lower than that in resistance training, and in concurrent training, it was lower than that in both endurance and resistance training. So one of the reasons for lower levels of plasma visfatin in concurrent training group as compared to resistance and endurance training groups is likely, further reduction in body mass index and body fat percentage in concurrent training group. In this regard, Haider *et al.* (2006) after four months of cycling, observed reduced visfatin levels at the end of two and four months of training and at eight months of non-training, it was stable (37). Lee *et al.* (2010) reported that after twelve weeks of aerobic training on overweight subjects, there was desirable changes in body composition by reduced visfatin (29). Choi *et al.* (2007) by investigating the effect of 12 weeks of combined endurance and resistance training, observed reduced visfatin (30). Seo *et al.* (2011) also after twelve weeks of combined training on middle-aged women, reported reduced visfatin (31) which is consistent with the findings of the present study. In the present study, visfatin levels also after 8 weeks of concurrent training in obese men were less as compared to the resistance training. It seems that endurance training together with resistance training (combined training) may provide optimized conditions in the reduction of fat, especially central fat, and cause improvement in lipid and glucose metabolism process. It is likely that increased growth hormone secretion together with concurrent training has caused visfatin secretion. As

stated in animal studies, growth hormone causes suppression of visfatin gene expression in adult cells secreting visfatin (38).

According to the findings of the present study, it was found that concurrent training group had higher levels of plasma insulin, and lower plasma glucose levels than the both resistance and endurance training groups, and lower insulin resistance index than only endurance training groups. In Stefanov *et al.* (2012) study, a significant inverse relationship was observed between physical activity and insulin resistance level which is in line with previous reports that emphasizes on beneficial effects of physical activity on insulin sensitivity in different populations (39). It has been shown that physical activity through changes in body fat mass and also mechanisms independent on fat mass, such as increased GLUT4 transport and consequently glucose uptake in skeletal muscle, improves the capacity of skeletal muscles for fat oxidation, increase muscle cells' fat transfer and reduces the amount of fat metabolites (39). In this regard, Jorge *et al.* (2011) also observed no change in HOMA-IR index after 12 weeks of combined aerobic and resistance training (34). This difference in the results may reflect different methods used to evaluate insulin sensitivity. For example, researchers have stated that Hyperinsulinemic euglycemic clamp is more sensitive in evaluating insulin action (34). In another study, it was reported that when compared with endurance training, combination of endurance and resistance training similarly caused reduction of weight but have less positive effects on insulin sensitivity (40). Recent studies support the role of resistance training in the modulation of muscle signaling pathways during fasting through the inhibition of AKT/PKB path (41). Signaling path of AKT/PKB indicates a primary molecular mechanism by which insulin regulates glucose transfer in the skeletal muscle. Therefore, a reduction in signaling path of AKT/PKB in skeletal muscle by resistance training may help explain slight improvement in insulin sensitivity in resistance training, while by concurrent training, which is in line with other researches, insulin sensitivity was more increased (40).

Studies indicated that visfatin is an adipokine with insulin-like function and this effect varies depending on the amount of insulin., It has been reported that hyperglycemia causes increasing visfatin levels (42). In this regard, Haider *et al.* (5) demonstrated that insulin injection in diabetic patients prevents increasing plasma visfatin, therefore, reduced insulin occurred after pancreatic beta cells dysfunction occurred which may be compensated for changes in visfatin concentration (43). The researchers have also reported that visfatin is positively correlated with blood glucose concentration, and negatively correlated with insulin concentration (5). Given that in this study, in concurrent training group when compared with other groups, blood glucose concentration was less and insulin concentration was high, perhaps these changes in glucose and insulin levels have caused reduced visfatin in concurrent training group within 8 weeks.

Also, in a study, it was shown that increasing visfatin concentration is associated with increased insulin resistance. It was suggested that increased levels of visfatin in obese individuals may be a compensatory mechanism in the early steps of the development of insulin resistance (14). A reported positive correlation between visfatin levels and insulin resistance index supports this hypothesis (44).

In the present study, insulin resistance index in concurrent training group was lower than that in other groups after 8 weeks of training. Given that reduced levels of visfatin were associated with a reduction in insulin resistance, probably reduced plasma levels of visfatin are due to reduced insulin resistance.

CONCLUSION

The findings of the present study showed that levels of visfatin, glucose, body mass index and body fat percentage in the group of concurrent training were lower than that in groups of resistance and endurance training, and also insulin resistance of concurrent training were lower in than only endurance training group, showing that the effect of semi-insulin is more in concurrent training group. According to the results, probably, concurrent training had more effect on body composition, glucose metabolism and insulin resistance adjustment which can be effective in preventing obesity and adipokines' adjustment.

APPLICABLE REMARKS

- Concurrent training (endurance and resistance training) can be more effective on glucose regulation.
- Concurrent training can be more effective on obesity.

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