Published online 2017 October 30.

Research Article

Effects of Resistance and Aerobic Exercise Training or Education Associated with a Dietetic Program on Visfatin Concentrations and Body Composition in Overweight and Obese Women

Mehdi Kargarfard,^{1,*} Ardalan Shariat,² Ina Shaw,³ Parastoo Haddadi,¹ and Brandon S Shaw³

¹Department of Exercise Physiology, Faculty of Sport Sciences, University of Isfahan, Isfahan, Iran ²Sports Medicine Research Centre, Neuroscience Institute, Tehran University of Medical Sciences, Tehran, Iran ³Department of Human Movement Science, University of Zululand, Private Bag X1001, KwaDlangezwa, 3886, Kwazulu-Natal, Republic of South Africa

Corresponding author: Mehdi Kargarfard, Department of Exercise Physiology, Faculty of Sport Sciences, University of Isfahan, Hezar Jerib St, P. O. Box 81746-7344, Isfahan, Iran. Tel: +98-3137934245, Fax: +98-3136687572, E-mail: m.kargarfard@spr.ui.ac.ir

Received 2017 April 20; Revised 2017 July 06; Accepted 2017 August 28.

Abstract

Background: Obesity, as a chronic disease, is becoming increasingly prevalent especially among women.

Objectives: The purpose of this study was to evaluate and compare the effects of resistance training (RT), concurrent resistance training and aerobic training (RT + AT) on visfatin concentrations and body composition in overweight and obese women.

Methods: An eight-week pretest-posttest design with two experimental exercising groups and one control group was used. 45 overweight and obese women were randomized into resistance training (RT), concurrent resistance and aerobic training (RT + AT) or an education-only groups (EDU). All participants received a dietary and education/counselling intervention. However, only the RT and RT + AT group participated in the eight-week exercise training.

Results: Repeated measures analysis showed that visfatin was significantly decreased by 8.5% in the RT (P = 0.02) and 29.2% in the RT + AT (P < 0.001), but not in EDU (3.7%; P = 0.22). BMI was decreased in the RT (6.8%; P < 0.001), RT + AT (8.1%; P < 0.001) and EDU (4.4%; P < 0.001), while BF% decreased in the RT (6.9%; P = 0.001), RT + AT (13.1%; P = 0.001), and EDU (4.9%; P = 0.020). WHR was decreased in the RT (2.4%; P = 0.001), in the RT + AT (4.2%; P = 0.002) and EDU (2.5%; P = 0.02). VO_{2max} (mL.kg⁻¹.min⁻¹) increased in the RT (16.3%; P = 0.004), RT + AT (37.7%; P = 0.001), but not in EDU (7.2%; P = 0.72). The absolute value of VO_{2max} (L.min⁻¹) also significantly increased in RT + AT (0.25%; P < 0.05) compared to baseline, but did not differ between the groups.

Conclusions: While RT and education-alone improved body composition and aerobic capacity, structured regular exercise incorporating both RT and AT may be required to improve visfatin and VO_{2max} in overweight and obese females.

Keywords: Adipokine, Physical Exercise, Endurance Training, Strength Training

1. Background

During the past few decades, the incidence of overweightness and obesity has reached epidemic levels, and at same time, there was are increases in numerous overweight/obesity-related diseases such as certain types of cancer, heart disease, and also diabetes (1). Weight loss via lifestyle modification is one of the suggested ways to treat or prevent these diseases (2). Typical weight loss related to lifestyle modification is around 5% -10% of baseline weight, but it can bring health benefits (3).

Although overweightness and obesity result from an imbalance between energy intake and expenditure, the pathophysiology of this condition (an imbalance between energy intake and expenditure) is best explained by an enlargement and/or increase in fat cells (4). This increase in adipose tissue, which as an active endocrine organ, is responsible for the secretion of various peptides called adipocytokines, which themselves play an important role in regulating energy reception and consumption (5). One such peptide is visfatin, which is primarily produced by visceral adipose tissue (and skeletal muscles, liver, bone marrow, and lymphocytes) and whose gene expression and plasma concentrations are found to be increased in obese individuals (6).

Visfatin is recognized as the formerly described Nicotinamide phosphoribosyltransferase (Nampt), and is a new adipokine which is a colony-stimulating factor activating the synthesis of pre-beta cells isolated from lymphocytes (7, 8). At present, it is speculated that visfatin alters fat metabolism either directly or indirectly (9). Support for

Copyright © 2017, Asian Journal of Sports Medicine. This is an open-access article distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License (http://creativecommons.org/licenses/by-nc/4.0/) which permits copy and redistribute the material just in noncommercial usages, provided the original work is properly cited.

the role of visfatin in the development of overweightness and obesity arises from those studies that demonstrate increased visfatin concentrations in overweight and obese individuals (10, 11). In addition, these elevated visfatin concentrations and its gene expression in fat cells in obese individuals provides convincing evidence of the relationship of visfatin with visceral fat mass and body mass index (12).

However, the research pertaining to the effects of exercise on plasma visfatin concentrations are not yet conclusive, and some contradictory results have been found. While it has been demonstrated that visfatin increases after the first phase of exhaustive aerobic exercise, and remains high for 24 hours following the exercise bout (13), a reduction of plasma visfatin levels or even the absence of change in visfatin levels following aerobic exercise have also been reported (14). In spite of the majority of published results having utilized only aerobic training in their protocols, data from recent studies suggests that resistance training may be an effective alternative to aerobic training for improving body composition in obese individuals (10, 15), since resistance training has been shown to preferentially reduce visceral and subcutaneous tissue in the abdominal region (10). Further, previous studies may indicate a possibility that a combination of both aerobic and resistance training may reduce visfatin even more than either aerobic or resistance training alone due to superior improvements in body composition, especially abdominal fat (10), and even superior improvements in self-reported dietary restriction, above those of these sole modes of exercise training (16). Recently, some studies have suggested that plasma visfatin is reduced after aerobic exercise training in sedentary, older, obese men and women (17). In addition, Mehdizadeh et al. (2016) demonstrated that their aerobic exercise group displayed more improvements in their weight, body fat percentage (BF%) and the waist-to-hip ratio (WHR) indices than resistance training participants. Moreover, the combined exercise training (aerobic-resistance) was more effective than the aerobic exercise in the improvement of BF%, but visfatin levels decreased equally in both aerobic and combined (aerobic-resistance) groups (18).

However, the changes observed in body composition, and thus possibly visfatin, following resistance training may be due to the type of resistance training that an individual engages in (19). As such, this study was constructed to evaluate and compare the effects of resistance training (RT), concurrent resistance and aerobic training (RT + AT) and education-only (EDU) on plasma visfatin concentrations and body composition in overweight and obese women. We hypothesized that an eight-week RT or RT + AT can improve visfatin levels and body composition in overweight and obese women, with superior improvements arising from the concurrent training (i.e. RT + AT).

2. Methods

2.1. Subjects

The study was approved (24 March 2016) by the University of Isfahan, Isfahan, Iran and the director of the Sepahan health clinic, Isfahan, Iran (ID:07041395/012). The study complied with the principles in the declaration of Helsinki. The present study employed a pretest-posttest design with two experimental groups and one control group. Participants were recruited (June 2016) from patients registered at the Sepahan health clinic, Isfahan, Iran, and were screened and received approval by their attending specialists for participation in the study. Of the 97 patients, 45 eligible overweight and obese women were randomized, using a random numbers table, into one of three groups, namely; a resistance training group (RT) (n = 15), concurrent resistance and aerobic training group (RT + AT) (n =15) or an education-only group (EDU) (n = 15). Participants were not aware of the randomized nature of the study nor were they privy to the existence of the other groups. Prior to participation, all participants were required to give written informed consent to participate in the study and all participants were informed of their right to discontinue the study at any point (20).

Following written informed consent, eligibility was determined using defined study inclusion and exclusion criteria. All participants were required to be free of any absolute or relative contraindications to exercise (21) and all participants were medically and clinically stable and ambulant without any aids. Eligibility criteria included being overweight and obese (BMI > 25 kg.m⁻²), adult females with a previously sedentary lifestyle (did not participate in regular exercise more than twice a week), stable body mass (\pm 2 kilograms) over the past year, were not making use of medication and/or supplementation affecting metabolism, particularly fat metabolism, and had no change in their regular medication usage for at least six months prior to enrolling in the study. There were no statistically significant differences between the participants in three groups regarding the mean of clinical and treatment characteristics.

Forty-one participants completed the study. Of the initial 15 participants in the RT group, 3 (6.7%) participants were excluded with 1 being due to personal circumstances, 1 being unable to regularly participate in the exercise training, and 1 sustaining an injury not related to the study that limited their ability to participate in testing. Of the initial 15 participants in the RT + AT group, 1 (2.2%) participant dropped out after study due to failing to participate in the final measurements. The final sample size was confined to 41 (RT: n = 12, RT + AT: n = 14 and EDU: n = 15). For these 41 participants, the analysis of the baseline data determined that the data had a normal distribution. Demographic and baseline characteristics of participants in each group are shown in Table 1.

2.2. Measurements

One week prior to the respective interventions, and at least 48 hours following the last training session, all participants underwent an identical battery of tests.

2.2.1. Anthropometric Variables

Anthropometric measurements were carried out according to the methods proposed by the International Society for the Advancement of Kinanthropometry (ISAK) (22). Body mass (BM) was measured in kilograms on a calibrated medical scale (Trojan, BSA16056v, Duteck Industrial co. Ltd, Taiwan), whilst stature was measured, to the nearest millimeter, using a standardised wall mounted stadiometer (Seca Stadiometer, 216, Seca, USA). Participants were required to wear minimal clothing and no shoes whilst the same technician completed these tests. Body Mass Index (BMI) was calculated by dividing the participant's body mass (kg) by stature squared (m^2) and expressed as kilograms per square meter (kg.m⁻²). Waist circumference (WC) and hip circumference (HC) measurements were measured to the nearest centimetre using a non-distendable measuring tape (MyoTape Body Tape Measure, Accufitness, USA). WC measurements were taken at the narrowest part of the torso, above the umbilicus and below the xiphoid process while HC measurements were taken at the maximal circumference of buttocks. The waistto-hip ratio (WHR) was determined by dividing the WC by the HC (waist circumference (cm) divided by hip circumference (cm)). In order to calculate percentage body fat, skinfold (triceps, suprailium, and abdominal) measurements were taken on the right side of the body using a Harpenden skinfold calliper (Harpenden, HSB-BI, ATICO Medical Pvt. Ltd, United Kingdom). Percentage body fat (BF%) was calculated from the equation: 1.089733-0.0009245 (x) + 0.0000025 (x2) - 0.0000979 (y); where x = the sum of triceps, suprailium, and abdominal skinfolds (in mm) for women, and y = age in years (18).

2.2.2. Blood Variables

Blood samples were obtained following a 12-hour fast, collected into vacutainer tubes with Ethylene Diamine Tetracetic Acid (EDTA) and stored at -80°C. Visfatin was analyzed using commercially available enzyme-linked immunosorbent assay (ELISA) kits (Phoenix Peptides, Karl-

sruhe, Germany) with an interassay and intraassay coefficient of variation of less than 6%. The sensitivity of measurement method was 1.5 ng.mL⁻¹.

2.2.3. Maximal Oxygen Consumption (VO2max)

Maximal oxygen consumption (VO_{2max}) was estimated using the modified Bruce protocol graded treadmill test (23). The initial workload required that participants walk for 4 minutes at a 2.5% grade and 4.8 km.h⁻¹. While the speed remained at a constant 4.8 km.h⁻¹, each ensuing work level lasted 1 minute with the grade increasing by 2.5%, until a 15% grade was reached. At this point, speed was increased by 0.8 km.h⁻¹ until volitional fatigue, symptoms of dyspnea, or a level of perceived exertion of 19 to 20 according to the Borg scale (24) were reported, or until relative indications for termination of an exercise test became apparent (25). The following Bruce protocol formula was utilized for estimating VO_{2max} in women: VO_{2max} = 4.38 × T - 3.9; where T = total time on the treadmill measured as a fraction of a minute (23).

2.3. Dietary Intervention

A well-nourished meal, in which six types of food had been incorporated, constituted the dietary intervention framework. Moreover, all participants were prescribed a balanced hypocaloric diet (500 - 1000 kcal.wk⁻¹) consisting of 25 - 30 kcal.kg⁻¹, ~ 30% fat, ~ 50% carbohydrate, and ~ 20% protein (26).

2.4. Education Intervention Protocol

In addition to the dietary intervention, all participants received an education/counselling intervention at the beginning of the study and at weekly intervals throughout the study. At these sessions, recordings of body mass loss, as well as the provision of suggestions for healthier eating habits and an analysis of the food participants ate formed part of the participants' weekly visits to the nutritionist. Information utilized during these sessions was gleaned via a 1-day dietary recall, which the participants were required to be complete every two weeks. American diabetes association exchange lists were the primary sources of information used for dietary advice, while behavior modification was used as the secondary source (27). The dietary advice proposed that 50% of energy intake should arise from carbohydrates, 30% from fats, and 20% from proteins (27).

2.5. Training Programs

While all participants received the dietary and education interventions for the duration of the study, participants were randomly assigned to either the EDU, RT or RT

Table 1. Demographic and Baseline Characteristics of Participants in Each Group $(N = 41)^a$	

Variables	RT (N = 12)	RT + AT(N = 14)	EDU (N = 15)	P Value
Age, y	42.00 ± 5.41	41.43 ± 6.41	39.33 ± 6.34	0.48
Stature, cm	161.42 ± 5.03	158.75 ± 7.12	160.83 ± 4.77	0.46
Body mass, kg	89.68 ± 9.25	90.27 ± 9.76	88.66 ± 11.86	0.92
BMI, kg.m ⁻²	34.52 ± 4.18	35.85 ± 3.50	34.28 ± 4.60	0.56
WHR	0.94 ± 0.03	0.96 ± 0.04	0.95 ± 0.08	0.61
Body Fat, %	38.80 ± 4.02	41.57 ± 3.54	38.75 ± 4.69	0.14
VO _{2max} , mL.kg ⁻¹ .min ⁻¹	19.91 ± 3.90	20.70 ± 4.59	20.16 ± 3.09	0.87
VO _{2max} , L.min ⁻¹	1.78 ± 0.41	1.89 ± 0.54	1.79 ± 0.43	0.78

Abbreviations: BMI, Body Mass Index; cm, Centimeters; EDU, Education Group; kg, kilograms; kg.m⁻², Kilograms Per Square Meter; mL.kg⁻¹.min⁻¹, Milliliters Per Minute Per Kilogram; RT, resistance training groups; RT + AT, Resistance Training and Aerobic Training Group; VO_{2max}, Maximal Oxygen Consumption; WHR, Waist-to-Hip Ratio. ^aValues are means ± standard deviation.

+ AT. The EDU received only dietary and education interventions for eight weeks while the RT and RT + AT completed progressive physical conditioning that included resistance training or concurrent resistance and aerobic training, respectively.

Participants in the RT and RT + AT exercised for eight weeks using RT thrice weekly for 40 - 60 minutes. The resistance training protocol began with a warm-up utilizing 10 minutes of low-intensity running (heart rate < 100 beats.min⁻¹), whole-body stretching and kinetic/dynamic movements. Both the RT and RT + AT participants performed the following resistance training protocol using 3 sets of 6 - 12 repetitions (at 67% - 85% one-repetition maximum (1-RM)). Since quantity of muscle output and the stimulation of neural adaptation (neural drive) was emphasized, eight whole-body, multi- and single-joint exercises targeting isolated muscle groups were utilized in the resistance training protocols. The order of the exercises was implemented according to the "(Alternated) Push and "Pull" and "Core", Then Assistance Exercises" order proposed by the national strength and conditioning association (NSCA) (28): bench press, latissimus dorsi pull-downs, shoulder press, arm curls, abdominal crunch, leg press, leg curl and standing calf raise, with 30 - 90 seconds rest allowed between each set.

The training program was concluded by completing a cool-down, which included a three-minute walk (heart rate < 100 beats.min⁻¹) followed by 5 minutes of whole-body stretching. To determine training intensity, and to measure 1-RM, an indirect method was utilized (29). In addition to the resistance training protocol, the RT + AT performed additional aerobic training using a treadmill thrice weekly for the eight weeks beginning at an intensity of 55% maximum heart rate (HR_{max}) for 20 minutes per session. The

intensity of the aerobic training protocol was increased weekly via a simultaneous 2% increase in intensity and 2minute increase in duration, resulting in a final training intensity of 75% HR_{max} for 34 minutes. The participants completed a mean of 23 sessions of the possible 24, thus demonstrating a 96% compliance.

2.6. Statistical Analysis

Descriptive data are displayed as means \pm SD. The normality of distribution was checked for all variables with the Kolmogorov-Smirnov test. All variables were normally distributed. A one-way analysis of variance (ANOVA) test was performed to examine differences in the baseline characteristics of the participants between the RT, RT + AT and EDU groups. Differences across time (baseline and postintervention) between the RT, RT + AT and EDU groups, and for the interaction between time and group, were tested for significance using a mixed factor 3 \times 2 ANOVA with repeated-measures for each outcome measure. Mauchly's Test of Sphericity was used, with any violations adjusted by use of the Greenhouse-Geisser correction (GG). In the presence of a statistically significant F ratio, post hoc analyses were carried out using paired-samples t-tests for time, or independent t-tests for groups, adjusted using the Bonferroni correction. The Pearson correlation was calculated to examine the relationship between visfatin and other variables such as body mass, BMI, WHR, BF%, and VO_{2max}. Statistical significance was set at P < 0.05 and data were analyzed using commercial software (Statistical Package for Social Sciences (SPSS) Version 20, Chicago, IL).

3. Results

Analysis of the data revealed that visfatin was significantly ($P \le 0.05$) decreased by 8.5% in the RT group (P =

0.001) and by 29.2% in the RT + AT group (P = 0.001), but not in the EDU group (3.7%; P = 0.062) (Table 2). In addition, a significant between-group interaction was found for the three groups for visfatin (P = 0.001). In terms of body composition, the RT group were found to have significantly decreased body mass by 6.89% (P = 0.001), the RT + AT group by 8.34% (P=0.001) and the EDU group by 6.34% (P=0.001). In addition, BMI was found to be significantly decreased in the RT group (6.8%; P = 0.001), the RT + AT group (8.1%; (P = 0.001), and the EDU group (4.4%; P = 0.001), while BF% significantly decreased by 6.9% in the RT group (P = 0.001), 13.1% in the RT + AT group (P = 0.001), and by 4.9% in the EDU group (P = 0.020). WHR was found to be decreased by 4.2% in the RT + AT group (P = 0.001), by 2.4% in the RT group (P = 0.001) and by 2.5% in the EDU group (P = 0.002), With regards to aerobic fitness, the RT group were found to have significantly increased VO_{2max} by 16.3% (P = 0.001), and the RT + AT group by 37.7% (P = 0.020), but not in the EDU group (7.2% increase P = 0.72). In addition, a significant between-group interaction was found for the three groups (P = 0.030). The correlation coefficients between the changes in VO_{2max} , body composition and visfatin are shown in Table 3.

4. Discussion

The purpose of this study was to evaluate and compare the effects of resistance training, concurrent resistance and aerobic training and education-only on plasma visfatin concentrations and body composition in overweight and obese women. Findings of this study confirmed our main hypothesis that an eight-week RT or RT + AT can improve the level of Visfatin and body composition among overweight and obese women significantly.

Admittedly, there is substantial evidence on the positive outcome of exercise training among older adults with regards to insulin levels and glucose tolerance (30). However, the amount of data on circulating visfatin as an effect of exercise training remains insufficient (31). However, the exercise-induced reduction in visfatin seen in the present study is consistent with the relatively few, but exclusively aerobic, exercise training studies examining plasma visfatin (17, 32, 33). Specifically, Seo et al. (2011) demonstrated a similar trend of decreased visfatin levels following aerobic training with concomitant decreases in body mass in young overweight Korean women (34). Similarly, Brema et al. (2008) found decreased visfatin levels, in addition to waist circumference, in 30 15-year-old obese participants with type 2 diabetes before and after 12 weeks of aerobic training performed at 75% of maximum aerobic power for one hour utilizing four sessions weekly (35). Lee et al. (2010) too observed that 12 weeks of aerobic training resulted in reduced visfatin concentrations in obese females (10).

A lack of determination in the relationships connecting visfatin with body composition responses and changes in cardiorespiratory fitness resulting from exercise activities exists (31) and this finding may uniquely indicate that visfatin may not be influenced by body composition changes in the short-term. This is because the present study importantly demonstrates that even though the EDU group, which participated in no exercise intervention, significantly reduced their body mass by 6.34%, BMI by 6.30%; BF% by 5.03%, and WHR by 3.16%, they failed to reduce their visfatin levels. This supposition and finding is further supported by a previous finding that visfatin is upregulated by hypoxia, inflammation and hyperglycaemia and downregulated by insulin, somatostatin and statins, all of which are influenced by exercise and exercise training (36). Further, while a positive correlation has been found between visceral adipose tissue visfatin gene expression and body mass index (BMI), the relationship between subcutaneous fat visfatin and BMI is seen to be negative suggesting that visfatin regulation may differ depending on different fat patterns (37).

This study compared eight weeks of RT, concurrent training and education-only interventions, in conjunction with a hypocaloric diet and demonstrated that RT, concurrent training and counselling/education on general lifestyle and increased physical activities can improve body composition. However, only the exercise interventions improved aerobic capacity and visfatin concentrations in the sample of overweight and obese women.

The findings of previous studies have found that RT and concurrent training (10), as well as education-only with hypocaloric diet interventions improve body composition. However, while RT has previously been found to improve (38) or result in an unchanged WHR (10), in this study, WHR was found to be decreased following eight weeks of RT. This is important in that WHR is an indicator of central obesity, which itself is a risk factor for insulin resistance and metabolic syndrome. However, it is important to note that while WHR decreased following EDU, RT and RT + AT, visfatin concentrations concomitantly decreased only in the RT and RT + AT groups. As such, it appears that WHR may not have as a positive relationship with visfatin concentrations as previously proposed. A loss of excess total body fat accumulation is also important following exercise intervention. This is because excess total body fat, and not only abdominal visceral fat, results in an increased secretion of adipokines (39). This increase in overall adiposity then results in a change in insulin signals effectively disrupting insulin-dependent glucose uptake,

Table 2. Comparison of Effects of Resistance Training, Concurrent Resistance and Aerobic Training or Education on Visfatin Concent	trations And Body Composition in Over-
weight and Obese Women $(n = 41)^a$	

Variables	RT (N	RT (N=12)		Δ %	RT + AT (N = 14)		P Value*	Δ %	EDU (N=15)		PValue*	Δ %	(T x G)	(Between G)
	Pre-test	Post-test	-		Pre-test	Post-test	-		Pre-test	Post-test	-		Р	Р
Body mass, kg	89.7 ± 9.3	83.5 ± 8.2	< 0.001	-6.8	90.3 ± 9.8	82.7 ± 8.1	< 0.001	-8.1	88.7 ± 11.9	84.8 ± 11.7	< 0.001	-4.4	0.03	0.99
Body mass Index, kg.m ⁻²	34.5 ± 4.2	32.2 ± 3.9	< 0.001	-6.8	35.9 ± 3.5	32.9 ± 2.9	< 0.001	-8.1	34.3 ± 4.6	32.8 ± 4.6	< 0.001	-4.4	0.02	0.78
Waist-to-hip ratio	0.94 ± 0.04	0.92 ± 0.04	0.001	-2.4	0.97 ± 0.04	0.92 ± 0.04	0.002	-4.2	0.95 ± 0.08	0.93 ± 0.07	0.02	-2.5	0.31	0.81
Body Fat, %	38.8 ± 4.0	36.1 ± 3.7	0.001	-6.9	41.6 ± 3.5	36.1 ± 6.3	0.003	-13.1	38.8 ± 4.7	36.8 ± 5.0	0.02	-4.9	0.05	0.68
Visfatin, ng.mL ⁻¹	19.1 ± 7.7	16.9 ± 5.6	0.02	-8.5	19.6 ± 5.9	13.5 ± 3.5	< 0.001	-29.2	18.6 ± 6.6	17.6 ± 5.8	0.22	-3.7	0.001	0.71
VO _{2max} , mL.kg-1.min ⁻¹	19.9 ± 3.9	23.0 ± 4.8	0.004	16.3	20.7 ± 4.6	26.9 ± 4.7	< 0.001	37.7	20.2 ± 3.1	21.2 ± 3.2	0.72	7.2	0.002	0.14
VO _{2max} ,	1.8 ± 0.4	1.9 ± 0.5	0.13	8.54	1.9 ± 0.5	2.6 ± 0.6	0.046	25.07	1.8 ± 0.4	1.8 ± 0.4	0.78	3.35	0.16	0.18

Abbreviations: BMI, Body Mass Index; cm, Centimeters; EDU, Education Group; kg, kilograms; kg.m², Kilograms Per Square Meter; mL.kg⁻¹.min⁴, Milliliters Per Minute Per Kilogram; ng.mL⁻¹, Nanograms Per Millilitre; RT, resistance training groups; RT + AT, Resistance Training and Aerobic Training Group; VO_{2max}, Maximal Oxygen Consumption; WHR, Waits-to-Hip Ratio.

Table 3. Pearson Correlations Between the Changes in Body Composition, Visfatin and VO_{2max}

Variables		Body Mass	BMI	WHR	BF%	VO _{2max}	Visfatin
Body mass							
	Pearson correlation		0.990 ^a	0.330 ^b	0.319 ^b	-0.106	0.225
	Sig. (2-tailed)		0.000	0.04	0.04	0.51	0.16
BMI							
	Pearson correlation			0.325 ^b	0.301	-0.106	0.229
	Sig. (2-tailed)			0.04	0.06	0.51	0.15
WHF	t.						
	Pearson correlation				0.251	-0.212	0.030
	Sig. (2-tailed)				0.11	0.18	0.85
BF%							
	Pearson correlation					0.060	0.111
	Sig. (2-tailed)					0.71	0.49
VO _{2n}	nax						
	Pearson correlation						-0.158
	Sig. (2-tailed)						0.32
Visfa	ntin						
	Pearson correlation						
	Sig. (2-tailed)						

^aCorrelation is significant at the 0.01 level (2-tailed).

^bCorrelation is significant at the 0.05 level (2-tailed).

and brings about insulin resistance via compensatory increases in blood insulin levels (39).

This study found a significant increase in both the relative and absolute VO_{2max} in the RT + AT group. However, there was no significant difference between the groups. While the increase in aerobic capacity following RT and concurrent training are also not novel (40), the significant increase in relative, but not absolute, aerobic capacity following education-only intervention must be noted with caution. This is because this increase in relative aerobic capacity arises from a decreased body mass, which is utilized to calculate relative VO_{2max} , and not necessarily an increase

in consumption, transportation and/or utilization of oxygen in aerobic metabolism (40), as demonstrated by the non-significant increase in absolute aerobic capacity.

While growing evidence suggests that AT can reduce visceral fat and visfatin concentrations (14), only a limited amount of research has been conducted thus far on the effects of RT and concurrent RT and AT. In particular, the effects of exercise on visfatin and other adipocytokines are a matter of controversy and it seems that visfatin concentrations are reduced as a result of weight loss even in the absence of exercise, such as following gastrectomy (14). However, this study is unique in that it demonstrated that while the EDU significantly decreased their body mass (and improved their other body composition measures), this positive change in body composition was not accompanied by a concomitant positive decrease in visfatin levels.

Seo et al. (34) reported decreased levels of visfatin following 12 weeks of concurrent training (RT using 3 sets with 10 repetition maximum, and AT at an intensity of 60% - 70% heart rate reserve) in 20 middle-aged obese women. In addition, their study similarly found improvements in body mass, BF%, and WHR following concurrent training. Mehdizadeh et al. (18) too found that 12 weeks of 3 days weekly concurrent training (RT for 20 minutes and AT for 25 minutes) resulted in improvements in body mass, and BF% in non-diabetic overweight women. While it previously may have appeared that the aerobic component of concurrent training may have improved visfatin levels, this study uniquely demonstrates that RT only may improve visfatin levels, albeit not to the extent of concurrent training. In this regard, the results of some studies suggest that RT could result in body mass (and specifically fat mass) loss and increase insulin sensitivity in active tissues, such as skeletal muscles. The addition of RT to AT may prove crucial in that RT has an improved ability to stimulate greater insulin sensitivity in active tissues and to increase the amount of such active tissue via muscle hypertrophy (40), especially following hypertrophy RT (41).

While the findings of this study are novel, this study does have some limitations such as the small sample size and the short-term intervention of only eight weeks. In addition, it should be noted that when considering ecological validity, this study made use of specifically overweight and obese participants and only middle-age women. As such, it is still not clear whether the positive findings, specifically related to visfatin are due to age and/or sex, and if visfatin is mechanistically linked to insulin secretion, more sensitive measures of insulin secretion should be included in future studies.

4.1. Conclusion

The present study found improved body composition, visfatin and VO_{2max} in overweight and obese females following both aerobic and concurrent resistance and aerobic training with a hypocaloric dietary programs, but not following a hypocaloric dietary and eduction only program. While these findings demonstrate the beneficial effect of exercise on visfatin, further studies are required to explore the accurate mechanisms responsible for the effects of exercise, and specifically resistance training either alone or in combination with aerobic training, on visfatin. The findings of this study provide feasible alternatives for health professionals to implement either sole aerobic or concurrent resistance and aerobic training exercise interventions in the enhancement of body composition, VO_{2max} and visfatin in overweight and obese patients. Importantly, this study may for the first time indicate that visfatin may not be influenced by body composition changes in the short-term, but rather by exercise in the short-term.

Acknowledgments

The authors would like to thank the students who participated in this study and deputy vice chancellor for research, University of Isfahan, Isfahan, Iran.

Footnotes

Authors' Contribution: Study concept and design, Ardalan Shariat; data acquisition, data analysis and interpretation, Mehdi Kargarfard and Parastoo Haddadi; drafting of the manuscript and critical revision of the manuscript for important intellectual content, Ina Shaw and Brandon S Shaw; statistical analysis, administrative, technical, and material support, and study supervision, Mehdi Kargarfard and Brandon S Shaw. All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Ethical Approval: The study was approved (24 March 2016) by the University of Isfahan, Isfahan, Iran and the director of the Sepahan health clinic, Isfahan, Iran (ID:07041395/012). The study complied with the principles in the declaration of Helsinki.

Declaration of Conflicting Interests: The author(s) declared no potential conflicts of interests with respect to the research, authorship, and/or publication of this article.

Funding/Support: There was no grant or funding for this research and it was performed using the personal budgets of researchers.

References

- Foster-Schubert KE, Alfano CM, Duggan CR, Xiao L, Campbell KL, Kong A, et al. Effect of diet and exercise, alone or combined, on weight and body composition in overweight-to-obese postmenopausal women. *Obesity (Silver Spring).* 2012;**20**(8):1628-38. doi: 10.1038/oby.2011.76. [PubMed: 21494229].
- Rock CL, Flatt SW, Pakiz B, Taylor KS, Leone AF, Brelje K, et al. Weight loss, glycemic control, and cardiovascular disease risk factors in response to differential diet composition in a weight loss program in type 2 diabetes: a randomized controlled trial. *Diabetes Care*. 2014;37(6):1573–80. doi: 10.2337/dc13-2900. [PubMed: 24760261].
- Franz MJ, Boucher JL, Rutten-Ramos S, VanWormer JJ. Lifestyle weightloss intervention outcomes in overweight and obese adults with type 2 diabetes: a systematic review and meta-analysis of randomized clinical trials. *J Acad Nutr Diet.* 2015;**115**(9):1447-63. doi: 10.1016/j.jand.2015.02.031. [PubMed: 25935570].
- Bray GA. Obesity: the disease. J Med Chem. 2006;49(14):4001-7. doi: 10.1021/jim0680124. [PubMed: 16821759].
- Caprio S, Perry R, Kursawe R. Adolescent Obesity and Insulin Resistance: Roles of Ectopic Fat Accumulation and Adipose Inflammation. *Gastroenterology*. 2017;**152**(7):1638–46. doi: 10.1053/j.gastro.2016.12.051. [PubMed: 28192105].
- Berndt J, Kloting N, Kralisch S, Kovacs P, Fasshauer M, Schon MR, et al. Plasma visfatin concentrations and fat depot-specific mRNA expression in humans. *Diabetes*. 2005;54(10):2911–6. [PubMed: 16186392].
- Hosseinian M, Banitalebi E, Amirhosseini SE. Effect of 12 weeks of intensive interval and combined training on apolipoprotein A and B, Visfatin and Insulin resistance in overweight middle-aged women with type 2 diabetes. *Horizon Med Sci.* 2016;22(3):237-45.
- Cho SY, Roh HT. Effects of aerobic exercise training on peripheral brain-derived neurotrophic factor and eotaxin-1 levels in obese young men. *J Phys Ther Sci.* 2016;28(4):1355–8. doi: 10.1589/jpts.28.1355. [PubMed: 27190482].
- Mellick PF, Feger BJ, Oberlin DJ, Davis PG, Wideman L. High-Intensity Exercise and Carbohydrate Supplementation do not Alter Plasma Visfatin. J Sports Sci Med. 2017;16(1):69–76. [PubMed: 28344453].
- Lee KJ, Shin YA, Lee KY, Jun TW, Song W. Aerobic exercise traininginduced decrease in plasma visfatin and insulin resistance in obese female adolescents. *Int J Sport Nutr Exerc Metab.* 2010;**20**(4):275–81.
- Kargarfard M, Lam ET, Shariat A, Asle Mohammadi M, Afrasiabi S, Shaw I, et al. Effects of endurance and high intensity training on ICAM-1 and VCAM-1 levels and arterial pressure in obese and normal weight adolescents. *Phys Sportsmed.* 2016;44(3):208–16. doi: 10.1080/00913847.2016.1200442. [PubMed: 27291761].
- Martinez Larrad MT, Corbaton Anchuelo A, Fernandez Perez C, Perez Barba M, Lazcano Redondo Y, Serrano Rios M, et al. Obesity and Cardiovascular Risk: Variations in Visfatin Gene Can Modify the Obesity Associated Cardiovascular Risk. Results from the Segovia Population Based-Study. Spain. *PLoS One*. 2016;**11**(5):0153976. doi: 10.1371/journal.pone.0153976. [PubMed: 27166797].
- Jackson AS, Pollock ML, Ward A. Generalized equations for predicting body density of women. *Med Sci Sports Exerc.* 1980;12(3):175–81. [PubMed: 7402053].
- Lawrence KE, Shaw I, Shaw BS, Toriola AL. Influence of callisthenic training on anthropometry in overweight and obese individuals. *Afri J Phys Health Educ Recr Dance*. 2013;3(1):211–9.
- 15. Norton K, Olds T. Anthropometrica: A textbook of body measurement for sports and health courses. UNSW Press; 1996.
- Bales CW, Starr KNP, Orenduff MC, McDonald SR, Molnar K, Jarman AK, et al. Influence of protein intake, race, and age on responses to a weight reduction intervention in obese women. *Current Dev Nut.* 2017.
- Moez RAA, Said AA. Aerobic exercise in obese type 2 diabetic patients: Effect on plasminogen activator inhibitor 1 and visfatin levels. *Inter J Therap Rehab Res.* 2016;5(4):63–9.

- Mehdizadeh A, Hamzezadeh S, Tofighi A. Investigation of plasma visfatin changes in women with type 2 diabetes followed by endurance, resistance and combined exercise: The role of lipid profile, glycemic indices and insulin resistance. J Diabetes Metab. 2016;7(703):2.;7(703):2.
- Soori R, Rezaeian N, Khosravi N, Ahmadizad S, Taleghani HM, Jourkesh M, et al. Effects of water-based endurance training, resistance training, and combined water and resistance training programs on visfatin and ICAM-1 levels in sedentary obese women. *Sci Sport.* 2017;**32**(3):144–51.
- 20. Sadeghi H, Hakim MN, Hamid TA, Amri SB, Razeghi M, Farazdaghi M, et al. The effect of exergaming on knee proprioception in older men: A randomized controlled trial. *Arch Gerontol Geriatr.* 2017;**69**:144–50. doi: 10.1016/j.archger.2016.11.009. [PubMed: 27923177].
- Fearnbach SN, Silvert L, Keller KL, Genin PM, Morio B, Pereira B, et al. Reduced neural response to food cues following exercise is accompanied by decreased energy intake in obese adolescents. *Int J Obes (Lond).* 2016;40(1):77-83. doi: 10.1038/ijo.2015.215. [PubMed: 26449418].
- Shaw BS, Shaw I, Mamen A. Contrasting effects in anthropometric measures of total fatness and abdominal fat mass following endurance and concurrent endurance and resistance training. J Sports Med Phys Fitness. 2010;50(2):207–13. [PubMed: 20585300].
- 23. Chavda V V RMPCGPMHSC, Chavda VV, Rajput MH, Parmar C, Gokhale PA, Mehta HB, et al. Predicted maximal oxygen consumption (VO2MAX) values obtained during the maximal treadmill test using different protocols. *Natl J Integr Res Med.* 2013;**4**(2):149–55.
- 24. Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc*. 1982;**14**(5):377-81. [PubMed: 7154893].
- Medicine AC of S. ACSM's guidelines for exercise testing and prescription. China: Lippincott Williams and Wilkins; 2013.
- Shariat A, Kargarfard M, Danaee M, Bahri Mohd Tamrin S. Intensive resistance exercise and circadian salivary testosterone concentrations among young male recreational lifters. *J Strength Cond Res.* 2015;29(1):151–8. doi: 10.1519/JSC.000000000000632. [PubMed: 25051005].
- Inzucchi SE, Bergenstal RM, Buse JB, Diamant M, Ferrannini E, Nauck M, et al. Management of hyperglycemia in type 2 diabetes, 2015: a patient-centered approach: update to a position statement of the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes Care*. 2015;**38**(1):140–9. doi: 10.2337/dc14-2441. [PubMed: 25538310].
- 28. HaffGG, Triplett NT. Essentials of strength training and conditioning. 4th edition ed. Human Kinetics; .
- Shaw BS, Shaw I, Brown GA. Resistance exercise is medicine: Strength training in health promotion and rehabilitation. *Inter J Ther Rehab.* 2015;**22**(8):233.
- 30. AbouAssi H, Slentz CA, Mikus CR, Tanner CJ, Bateman LA, Willis LH, et al. The effects of aerobic, resistance, and combination training on insulin sensitivity and secretion in overweight adults from STRRIDE AT/RT: a randomized trial. *J Appl Physiol* (1985). 2015;**118**(12):1474–82. doi:10.1152/japplphysiol.00509.2014. [PubMed: 25882384].
- Chang YH, Chang DM, Lin KC, Shin SJ, Lee YJ. Visfatin in overweight/obesity, type 2 diabetes mellitus, insulin resistance, metabolic syndrome and cardiovascular diseases: a meta-analysis and systemic review. *Diabetes Metab Res Rev.* 2011;27(6):515–27. doi: 10.1002/dmrr.1201. [PubMed: 21484978].
- 32. Kadoglou NP, Fotiadis G, Kapelouzou A, Kostakis A, Liapis CD, Vrabas IS. The differential anti-inflammatory effects of exercise modalities and their association with early carotid atherosclerosis progression in patients with type 2 diabetes. *Diabet Med.* 2013;30(2):41-50. doi: 10.1111/dme.12055. [PubMed: 23078531].
- 33. Djelic M, Trajkovic V, Dumanovic MS, Mazic S, Durmic T, Jeremic R, et al. Pro-inflammatory cytokines responses to acute exercise in athletes and sedentary controls: Association with body composition and insulin sensitivity. *Endo Abs.* 2016;**41**(2):P824.

- Seo DI, So WY, Ha S, Yoo EJ, Kim D, Singh H, et al. Effects of 12 weeks of combined exercise training on visfatin and metabolic syndrome factors in obese middle-aged women. *J Sports Sci Med.* 2011;**10**(1):222– 6. [PubMed: 24149317].
- Brema I, Hatunic M, Finucane F, Burns N, Nolan JJ, Haider D, et al. Plasma visfatin is reduced after aerobic exercise in early onset type 2 diabetes mellitus. *Diabetes Obes Metab.* 2008;**10**(7):600–2. doi: 10.1111/j.1463-1326.2008.00872.x. [PubMed: 18476987].
- 36. Wandrag I, Siervo M, Riley HL, Khosravi M, Fernandez BO, Leckstrom CA, et al. Does hypoxia play a role in the development of sarcopenia in humans? Mechanistic insights from the Caudwell Xtreme Everest Expedition. *Redox Biol.* 2017;13:60–8. doi: 10.1016/j.redox.2017.05.004. [PubMed: 28570949].
- Rabkin SW. The relationship between epicardial fat and indices of obesity and the metabolic syndrome: a systematic review and meta-analysis. *Metab Syndr Relat Disord*. 2014;12(1):31–42. doi:

10.1089/met.2013.0107. [PubMed: 24219127].

- Nesic N., Seper V., Davidovic Cvetko E.. The impact of strength training on the changes in one's physique and resting energy expenditure. *Periodic Biol.* 2014;116(1):71–5.
- Trayhurn P. Hypoxia and adipocyte physiology: implications for adipose tissue dysfunction in obesity. *Annu Rev Nutr.* 2014;34:207–36. doi: 10.1146/annurev-nutr-071812-161156. [PubMed: 24819450].
- Mann S, Beedie C, Jimenez A. Differential effects of aerobic exercise, resistance training and combined exercise modalities on cholesterol and the lipid profile: review, synthesis and recommendations. *Sports Med.* 2014;44(2):211-21. doi: 10.1007/s40279-013-0110-5. [PubMed: 24174305].
- Bellamy LM, Joanisse S, Grubb A, Mitchell CJ, McKay BR, Phillips SM, et al. The acute satellite cell response and skeletal muscle hypertrophy following resistance training. *PLoS One.* 2014;9(10):109739. doi: 10.1371/journal.pone.0109739. [PubMed: 25313863].