



## Effect of exercise training type on plasma levels of vaspin, nesfatin-1, and high-sensitivity C-reactive protein in overweight and obese women

Mehdi Mogharnasi<sup>a</sup>, Hossein TaheriChadorneshin<sup>b,\*</sup>, Nadiieh Abbasi-Deloei<sup>c</sup>

<sup>a</sup> Department of Sport Sciences, University of Birjand, Birjand, Iran

<sup>b</sup> Department of Sport Sciences, University of Bojnord, Bojnord, Iran

<sup>c</sup> Department of Sport Sciences, University of Sistan and Baluchestan, Zahedan, Iran

### ARTICLE INFO

#### Keywords:

Endurance training  
Resistance training  
Vaspin  
Nesfatin-1  
High-sensitivity C-Reactive protein  
Overweight  
Obese  
Women

### ABSTRACT

**Background:** Vaspin, nesfatin-1 and high-sensitivity C-reactive protein (hs-CRP) act as main risk factors of inflammation and cardiovascular health. There are contradictory results about the effects of exercise training on these obesity-related factors. Therefore, the purpose of present study was to elucidate the effect of exercise training type on plasma levels of vaspin, hs-CRP, and nesfatin-1 in overweight and obese women.

**Methods:** Thirty-four overweight and obese women (age  $22.29 \pm 2.49$  years) were randomly assigned to the endurance, resistance and sedentary control groups. The 8-week endurance and resistance exercise trainings were conducted at 65–80% of maximal heart rate and one repetition maximum, respectively. Fasting blood samples were taken before and 48-hr after the last exercise training session. The serum concentrations of vaspin, nesfatin-1 and hs-CRP were measured using commercially available ELISA kit. The intra- and inter-group comparisons were performed by *t*-test and one-way ANOVA at a significant level of  $P < 0.05$ , respectively.

**Results:** Both of exercise trainings caused a significant reduction in plasma levels of vaspin ( $P = 0.001$  and  $P = 0.005$  for endurance and resistance exercise trainings, respectively) and hs-CRP ( $P = 0.008$  and  $P = 0.007$  for endurance and resistance exercise trainings, respectively). In contrast, a significant increase in plasma levels of nesfatin-1 ( $P = 0.001$  for both of endurance and resistance exercise trainings) and maximal oxygen consumption ( $P = 0.001$  and  $P = 0.003$  for endurance and resistance exercise trainings, respectively) were obtained.

**Conclusions:** Both of endurance and resistance exercise training protocols promote the cardiovascular health of overweight and obese women by improving obesity-related factors.

### 1. Introduction

Obesity is recognized as one of the major health problems in the world that poses a number of health-threatening factors including increased levels of lipids, diabetes and the risk of chronic diseases like cardiovascular disease and hypertension. Therefore, the importance of developing preventive and control programs for obesity cannot be overstated (Bastien et al., 2014; Jakicic et al., 2001). In recent decades, many studies have examined the relationship between adipose tissue, pathologic function and the development of CVDs risk, which is mainly activated through the release of chemical intermediates in form of autocrine/paracrine/endocrine, thereby regulating cardiovascular functions and a wide range of other biological processes (Ouchi et al., 2011; Feijóo-Bandín et al., 2016). In reality, the function of adipose tissue as an endocrine organ is characterized by the secretion of several bioactive

substances as adipose tissue or adipocyte-derived agents, which have pre-inflammatory or anti-inflammatory function (Bastien et al., 2014; Ouchi et al., 2011). The irregular production and secretion of these adipokines is associated with adipose tissue dysfunction, which can contribute to the pathogenesis of obesity-related complications (Ouchi et al., 2011).

Vaspin (visceral adipose tissue-derived serine) as a member of serine protease inhibitor family was first isolated and identified from visceral adipose tissue of Otsuka Long-Evans Tokushima Fatty (OLETF) rats (Hida et al., 2005). Vaspin is a protein with a molecular mass of 47 kD that is secreted from adipose tissue and is involved in insulin resistance (Ouchi et al., 2011; Hida et al., 2005). It has been shown that high expression of vaspin is associated with insulin resistance in young obese Sprague-Dawley rats (Gao et al., 2013). In contrast, nesfatin-1 as a polypeptide with 82-amino acid is derived from the post-translational

\* Corresponding author. Department of Sport Sciences, University of Bojnord, Bojnord, North Khorasan 9453155111, Islamic Republic of Iran.  
E-mail address: [kh.taheri\\_62@yahoo.com](mailto:kh.taheri_62@yahoo.com) (H. TaheriChadorneshin).

process of Nucleobindin2 (NUCB2) gene in hypothalamus of rat (Azamar-Llamas et al., 2017). Nesfatin-1 acts as an anti-inflammatory agent and improves insulin sensitivity by increasing insulin release from beta cells (Azamar-Llamas et al., 2017). It has been reported that any change in nesfatin-1 level is linked to weight change (Azamar-Llamas et al., 2017; Tsuchiya et al., 2010). In this context, a negative relationship has been reported between fasting nesfatin-1 level and BMI in non-obese males (Tsuchiya et al., 2010). Moreover, it has been shown that fasting nesfatin-1 in patients with type-2 diabetes is significantly lower than healthy subjects and patient with type-1 diabetes. The cause of this disparity is not known, but the decline in fasting nesfatin-1 may be due to one of the hormonal regulators of appetite associated with diabetic overeating (Li et al., 2010). Lifestyle changes such as increased physical activity are considered as the first intervention for improving glucose homeostasis, reducing excessive body fat and preventing metabolic disorders (Hill et al., 2007; Kang et al., 2009). In this relation, Chaolu et al. (2011) studied the effect of exercise training and high-fat diet on plasma levels of nesfatin-1 in rats, observing that a high-fat diet reduced plasma levels of nesfatin-1, but this decrease was controlled and suppressed by exercise training (Chaolu et al., 2011). As for the effect of exercise on vaspin, it has been demonstrated that acute and chronic exercise training (including cycling and swimming) leads to a significant reduction in plasma concentrations of vaspin (Oberbach et al., 2010).

Lipid profile disorders and inflammatory markers are among the major risk factors of cardiovascular disease. Among cardiovascular markers, high-sensitivity C-reactive protein (hs-CRP) is the most prominent inflammatory and predictor of cardiovascular disease, which has been shown to be strongly associated with coronary artery disease caused by trauma, stress and disease (Parrinello et al., 2015; Koenig, 2013; Kubota et al., 2010). In this context, it has been indicated that hs-CRP level in athletes with metabolic syndrome is lower than individuals with low physical fitness (Fayh et al., 2013). In addition, it has been reported that long-term aerobic and strength exercise trainings can significantly reduce hs-CRP and BMI in obese children (Kamal and Ragy, 2012) and adults (Martins et al., 2010). However, hs-CRP levels revert to the baseline level after a period of detraining (Martins et al., 2010).

Therefore, as indicated by above reports, there is a paucity of data on the effect of exercise training types on plasma levels of vaspin, nesfatin-1 and hs-CRP, which are directly related to obesity and act as a predictor of heart disease. The importance of this study lies in its exploration of exercises that are most effective in improving the health of overweight and obese women. Endurance exercise training increases the maximum oxygen and energy consumption by lipid oxidation while resistance exercise training, by building up the mass and lean muscle strength, improves body composition (Hill et al., 2007). Hence, this study was undertaken to answer the following question: “What is the effect of endurance and resistance exercise trainings on plasma levels of vaspin, nesfatin-1 and hs-CRP, and factors associated with obesity in overweight and obese females?”

## 2. Materials and methods

### 2.1. Subjects

The current study was approved by the Human Subjects Protection Committee of the University of Sistan and Baluchestan (Iran). Also, all procedures were implemented in accordance with the 1975 Declaration of Helsinki, and its 1996 revision. Thirty-four overweight and obese female students (studying at the University of Sistan and Baluchestan, Iran), with a mean age of  $22.29 \pm 2.49$  years and regular menstrual periods participated in this semi-experimental study on a voluntary basis. They completed Physical Activity Readiness Questionnaire (PAR-Q). Also, the physical fitness level was evaluated by Baecke physical activity questionnaire. Students resided in a dormitory and were on the

same diet. All students were examined by a physician. A written consent form was obtained from the participants after explaining the possible benefits and risks of the research. They did not have any chronic or acute diseases, and were not using any dietary supplements or drugs. Subjects were randomly assigned to the endurance ( $n = 12$ ), resistance ( $n = 12$ ) and sedentary control ( $n = 10$ ) groups.

### 2.2. Anthropometric and physiological measurements

Anthropometric measurements were carried out during the first visit of laboratory. The subjects' height was measured by a wall-mounted stadiometer (Sahand CO. Tabriz, Iran) and their weight was measured using a digital scale (Sahand CO. Tabriz, Iran) while they were bare-footed and wearing light clothing. Body fat percentage was predicted from the skinfold measurement using specialized calipers (Yagami model, Japan). A 3-site skin fold equation of Jackson and Pollock (triceps, thigh, and suprailiac) was used to estimate body density, and body fat percentage was subsequently calculated using the Siri equation. Thereafter, body fat mass (BFM) was calculated by multiplying body fat percentage by body weight (Jackson et al., 1980). All anthropometric measurements were determined before and after endurance and resistance exercise trainings. Moreover, maximal oxygen consumption ( $VO_{2max}$ ), as a physiological parameter, was measured before and after training using One Mile Walking test (the Rockport test).

### 2.3. Exercise training protocols

Endurance exercise training was performed based on the overload principle over a period of 8 weeks, 4 days per week, 1 session per day. Students ran at 65% maximum heart rate (HRmax) for 20 min on the first session, and the duration of exercise was increased by 2 min per week until 34 min was achieved by the 8th week. Also, running intensity was increased by 5% every two weeks until 80% HRmax was achieved by the 8th week (Donges et al., 2010). Intensity of endurance exercise training was controlled by a pedometer (POLAR F92ti, Finland).

Resistance exercise training was performed based on the overload principle over a period of 8 weeks, 4 days per week, 1 session per day. Initially, participants received instruction on how to work with resistance training devices. Then, maximal muscle strength was determined according to Brzycki equation. Resistance exercise trainings were conducted at eight stations. The exercises, intended to stress major muscle groups, consisted of lat pull-down, bench press, leg press, calf exercise, biceps curls, leg curls, lateral raise by dumbbell, and overhead press. Participants were asked to perform each exercise for 2–4 sets, 8–12 repetitions, with a 60–90 s break between each station and 2–3 min rest between each circuit. The length of resistance exercise training in the first week was 20 min, which was progressively increased to 35 min by the 8th week (Mogharnasi et al., 2017; Jorge et al., 2011a).

At the beginning and end of endurance and resistance exercise trainings, warm-up and cool-down were performed for 10 min at moderate intensity in form of jogging and stretching exercises (Mogharnasi et al., 2017). During the study period, students were prohibited from engaging in any kind of physical activity other than the study intervention (Jorge et al., 2011a). Students in the sedentary control group were asked to perform routine daily activities of without any exercise training intervention (Mogharnasi et al., 2017; Jorge et al., 2011a).

### 2.4. Blood sampling

12-h fasting blood samples were taken from an antecubital vein before and 48 h after last exercise training secession. Blood samples (5 ml) were taken by a laboratory specialist between 8:00 to 9:00 a.m. Samples were injected directly into anticoagulant tubes. Plasma was prepared by centrifugation (Eppendorf Centrifuge, Mini Spin R,

Germany) at 3000 rpm for 5 min and stored at  $-70^{\circ}\text{C}$  for further analysis. The plasma levels of vaspin (Cat.No:CK-E10968) and nesfatin-1 (Cat.No:CK-E90098), as well as hs-CRP (Cat.No:CK-E11183) were measured using a human commercial ELISA kit (Eastbiopharm, Hangzhou Eastbiopharm CO.,LTD., China). The sensitivities of kits were 0.13, 0.15, and 10 ng/ml for vaspin, nesfatin-1 and hs-CRP, respectively. In accordance with the manufacturer's instructions, we add prepared samples to monoclonal antibody enzyme well which is pre-coated for 60 min at  $37^{\circ}\text{C}$ . Plate washed five times. Then, chromogen solutions add to each well. Gently mixed, incubate for 10 min at  $37^{\circ}\text{C}$  away from light. In the following, stop solution add into each well to stop the reaction. Finally, the absorbance of vaspin, nesfatin-1, and hs-CRP were measured at 450 nm by Anthos 2020 microplate reader (Biochrom CO, England) within 10 min.

### 2.5. Statistical analysis

Statistics analyses were performed by Statistical Package for Social Sciences (SPSS Inc., Chicago, USA) software, version 16.0. All the data were tested for homogeneity of variance and normality by Levene's and Shapiro–Wilk's test, respectively. The intra-group comparison was performed by dependent *t*-test and inter-group comparison was conducted by one-way ANOVA followed by LSD post hoc test. A significant level of  $P < 0.05$  was assumed. Data are presented as mean  $\pm$  standard deviation.

### 3. Results

The results of study did not reveal any significant difference between pre-test values of age ( $P = 0.54$ ), height ( $P = 0.47$ ), weight ( $P = 0.91$ ), BMI ( $P = 0.81$ ), BFM ( $P = 0.89$ ), WHR ( $P = 0.20$ ) and  $\text{VO}_2\text{max}$  ( $P = 0.84$ ) of the three groups (Table 1).

Intra-group comparisons showed that endurance exercise training induced a significant reduction in weight ( $P = 0.003$ ), BMI ( $P = 0.002$ ), BFM ( $P = 0.001$ ), and WHR ( $P = 0.011$ ) (Table 2). Besides, significant reductions in weight ( $P = 0.005$ ), BMI ( $P = 0.004$ ), BFM ( $P = 0.001$ ), and WHR ( $P = 0.013$ ) were observed following resistance exercise training (Table 2). However, inter-group comparisons did not show any significant difference between weight ( $P = 0.627$ ), BMI ( $P = 0.347$ ), and BFM ( $P = 0.397$ ) (Table 2). In contrast to resistance exercise training ( $P = 0.066$ ), endurance exercise training led to a significant reduction in WHR as compared to the sedentary control group ( $P = 0.007$ ) (Table 2). However, no significant difference between the subjects' WHR in endurance and resistance exercise training groups was observed at the end of protocol ( $P = 0.351$ ).

$\text{VO}_2\text{max}$ , as a marker of cardiorespiratory fitness, was significantly increased following 8-week endurance ( $P = 0.001$ ) and resistance ( $P = 0.003$ ) exercise trainings (Table 3). Furthermore, inter-group comparison suggested a greater increase in  $\text{VO}_2\text{max}$  induced by endurance ( $P = 0.001$ ) and resistance ( $P = 0.001$ ) exercise trainings in

**Table 1**

Anthropometric and physiological measurements in overweight and obese women.

Variable	Group		
	Endurance	Resistance	Sedentary control
Age (year)	22.81 $\pm$ 2.44	22.50 $\pm$ 2.67	21.50 $\pm$ 2.41
Height (cm)	158.55 $\pm$ 5.33	161.60 $\pm$ 7.74	159.20 $\pm$ 6.40
Weight (kg)	76.17 $\pm$ 6.81	77.96 $\pm$ 9.71	77.69 $\pm$ 13.72
BMI (kg/m <sup>2</sup> )	30.31 $\pm$ 2.48	29.73 $\pm$ 1.49	30.51 $\pm$ 3.99
BFM (kg)	26.82 $\pm$ 5.88	26.38 $\pm$ 5.84	25.33 $\pm$ 9.60
WHR	0.82 $\pm$ 0.04	0.84 $\pm$ 0.04	0.85 $\pm$ 0.02
$\text{VO}_2\text{max}$ (ml/kg/min)	36.32 $\pm$ 4.69	37.31 $\pm$ 3.63	36.93 $\pm$ 3.22

Abbreviations: BFM, Body Fat Mass; BMI, Body Mass Index;  $\text{VO}_2\text{max}$ , Maximal Oxygen Consumption; WHR, Waist-to-Hip Ratio.

**Table 2**

Effect of endurance and resistance exercise training on anthropometric measurements in overweight and obese women.

Variables	Groups	Pre-test	Post-test
Weight (kg)	Endurance	76.17 $\pm$ 6.81	74.36 $\pm$ 7.26*
	Resistance	77.96 $\pm$ 9.71	76.30 $\pm$ 9.43*
	Sedentary control	77.69 $\pm$ 13.72	78.72 $\pm$ 13.36
BFM (kg)	Endurance	26.82 $\pm$ 5.88	24.38 $\pm$ 5.56*
	Resistance	26.38 $\pm$ 5.84	24.58 $\pm$ 5.38*
	Sedentary control	25.33 $\pm$ 9.60	26.30 $\pm$ 9.68
BMI (kg/m <sup>2</sup> )	Endurance	30.31 $\pm$ 2.48	29.63 $\pm$ 2.51*
	Resistance	29.73 $\pm$ 1.49	29.12 $\pm$ 1.49*
	Sedentary control	30.51 $\pm$ 3.99	30.92 $\pm$ 3.91
WHR	Endurance	0.82 $\pm$ 0.04	0.81 $\pm$ 0.03*#
	Resistance	0.84 $\pm$ 0.04	0.83 $\pm$ 0.04*
	Sedentary control	0.85 $\pm$ 0.04	0.86 $\pm$ 0.02

\* Significantly different within group by the paired *t*-test, \*:  $P < 0.05$ .

# Significantly different than sedentary control group, #:  $P < 0.05$ .

Abbreviations: BMI, Body Mass Index; BFM, Body Fat Mass, WHR, Waist-to-Hip Ratio.

**Table 3**

Effect of endurance and resistance exercise training on physiological and obesity-related factors in overweight and obese women.

Variables	Groups	Pre-test	Post-test
$\text{VO}_2\text{max}$ (ml/kg/min)	Endurance	36.32 $\pm$ 4.69	43.39 $\pm$ 3.98*#
	Resistance	37.31 $\pm$ 3.63	41.76 $\pm$ 3.04*#
	Sedentary control	36.93 $\pm$ 3.22	35.97 $\pm$ 1.42
Vaspin (pg/ml)	Endurance	1.73 $\pm$ 0.42	1.01 $\pm$ 0.30*
	Resistance	1.68 $\pm$ 0.25	1.12 $\pm$ 0.28*
	Sedentary control	1.88 $\pm$ 0.29	1.74 $\pm$ 0.76
hs-CRP (pg/ml)	Endurance	4620 $\pm$ 2885	595 $\pm$ 155*
	Resistance	4411 $\pm$ 2658	666 $\pm$ 151*
	Sedentary control	6105 $\pm$ 3878	6235 $\pm$ 172
Nesfatin-1 (ng/ml)	Endurance	13.07 $\pm$ 1.62	14.38 $\pm$ 1.70*#
	Resistance	12.02 $\pm$ 0.79	14.17 $\pm$ 0.64*#
	Sedentary control	13.24 $\pm$ 0.76	12.08 $\pm$ 0.98

\* Significantly different within group by the paired *t*-test, \*:  $P < 0.05$ .

# Significantly different than sedentary control group, #:  $P < 0.05$ .

Abbreviations: High-sensitivity C-reactive protein (hs-CRP);  $\text{VO}_2\text{max}$ , Maximal Oxygen Consumption.

comparison with the sedentary control group (Table 3). However, there was no significant difference between the subjects'  $\text{VO}_2\text{max}$  in endurance and resistance exercise training groups at the end of protocol ( $P = 0.230$ ) (Table 3).

With regard to obesity-related factors, our findings indicated that endurance and resistance exercise trainings significantly reduced plasma levels of vaspin ( $P = 0.001$  and  $P = 0.005$ , respectively) and hs-CRP ( $P = 0.008$  and  $P = 0.007$ , respectively), while inter-group comparison did not reveal any significant difference between subjects' vaspin ( $P = 0.107$ ) and hs-CRP ( $P = 0.412$ ) (Table 3). In contrast, plasma levels of nesfatin-1 were significantly increased following endurance ( $P = 0.001$ ) and resistance ( $P = 0.001$ ) exercise trainings (Table 3). Besides, inter-group comparison exhibited a remarkable increase in nesfatin-1 induced by endurance ( $P = 0.002$ ) and resistance ( $P = 0.006$ ) exercise trainings compared to the sedentary control group (Table 3). However, no significant difference between the subjects' nesfatin-1 in endurance and resistance exercise training groups was observed at the end of protocol ( $P = 0.735$ ) (Table 3).

### 4. Discussion

The results of the present study revealed a significant decline in plasma levels of vaspin and hs-CRP, as well as anthropometric measurements of BMI, body weight, BFM and WHR following endurance and resistance exercise trainings. However, the reduction in the

endurance group was higher than that of the resistance group, but the difference was not significant. In contrast, both endurance and resistance exercise training led to a significant rise in plasma levels of nesfatin-1 and  $\text{VO}_{2\text{max}}$ . There was a significant difference between experimental and sedentary control groups in terms of  $\text{VO}_{2\text{max}}$  and WHR values.

Vaspin, as a protein with 47 kDa, increases metabolic syndrome and is associated with atherosclerosis in both men and women (Hordern et al., 2012; Choi et al., 2011). It is secreted from adipose tissue and linked to insulin resistance (Ouchi et al., 2011; Hida et al., 2005). In this context, the high correlation between serum vaspin concentration and visceral adipose has been shown (Chang et al., 2010). In addition, the serum vaspin concentration reported in obese and overweight women is higher than that of healthy women. Insulin sensitizers reduce serum vaspin (26). However, it has been shown that improved insulin resistance following aerobic exercise training can be achieved without any significant changes in vaspin levels (Kim et al., 2011). Therefore, it seems that mechanisms other than vaspin are involved in insulin sensitivity induced by exercise training (Lee et al., 2010). In this context, it has been reported that changes in plasma levels of vaspin are related to the weight loss. Since nutritional interventions were not incorporated in the present study, the observed reduction in vaspin concentration may be due to reduced body weight and BFM caused by endurance and resistance exercise trainings. Waist circumference is acknowledged as a useful index of changes in the central obesity (Wang et al., 2010). Thus, reduction in plasma levels of vaspin in present study can be partly attributed to reduced abdominal or central obesity. In one study, it was shown that modifying behavior and lifestyle could lead to weight loss, but no significant changes were observed in vaspin levels (Martos-Moreno et al., 2011). Thus, it can be stated that any change in the plasma level of vaspin is only achieved if weight loss is induced by exercise training rather than other interventions. As such, it has been revealed that weight loss induced by nutritional approaches does not have any significant effect on plasma levels of vaspin (Vink et al., 2017). An inverse correlation has been reported between cardiorespiratory endurance and vaspin levels (Klötting et al., 2006). In the present study, decreased plasma level of vaspin and hs-CRP and improved  $\text{VO}_{2\text{max}}$  were observed in both exercise training groups, which indicates the increased use of stored fats during the metabolism process.

Nesfatin-1, as a protein with 9.7 kDa, is synthesized in hypothalamus and crosses the brain-blood barrier in both directions (Pan et al., 2007). It can act as an anti-inflammatory agent and generate an anti-hyperglycemic effect under impaired glucose metabolism conditions (Azamar-Llamas et al., 2017). Nesfatin-1 has also been reported to increase insulin sensitivity in brain and raise insulin release in beta cells in response to hyperglycemia (Yang et al., 2012). The results of the present study showed that plasma levels of nesfatin-1 were increased following endurance and resistance exercise trainings. Our findings are consistent with another study that showed increased plasma nesfatin-1 levels in boxing and taekwondo athletes after each match (Yazici, 2015). Athletes engaged in anaerobic exercises often attempt to lose weight in the process of preparation for tournaments to reach optimal weight, which can explain the elevated nesfatin-1 (Yazici, 2015). In addition, it has been reported that eight weeks of moderate-intensity cycle ergometer in obese women diminished BMI and insulin and raised plasma levels of nesfatin-1 (Nabil and El Sayyad, 2015). As noted in the literature, nesfatin-1 is highly sensitive to long-term exercises and significant loss of weight and fat percentage (Yazici, 2015; Nabil and El Sayyad, 2015). In this context, it has been shown that only 5–10% reduction of visceral and subcutaneous fat induced by exercise can serve as stimuli to improve the levels of adipocytokine (Sakurai et al., 2017). According to the results of this study, weight change is associated with modification of BFM, BMI and nesfatin-1 levels. As such, a negative correlation has been reported between BMI and nesfatin-1 (Tsuchiya et al., 2010). Nesfatin-1 as a neuropeptide is involved in regulating metabolism and nutrition patterns (Azamar-Llamas et al., 2017). It also

induces a sense of satiety, which acts as a strong inhibitor of water and food intake, and therefore directly linked to the weight loss (Nabil and El Sayyad, 2015). In addition, it has been shown that fasting conditions decrease the level of serum nesfatin-1 up to 18% (Stengel et al., 2008). Therefore, the observed increased in plasma levels of nesfatin-1 in present study could be due to the fasting during sampling. On the other hand, it has been reported that the level of nesfatin-1 reverts to the baseline level 12 h after re-feeding (Gao et al., 2013).

hs-CRP, is a sensitive marker of systemic low-grade inflammation (Lakka et al., 2005). Elevated plasma hs-CRP levels are associated with obesity, insulin resistance, and metabolic syndrome (Festa et al., 2000). Results of this study demonstrated that both endurance and resistance exercise trainings reduced plasma hs-CRP levels in overweight and obese women. In line with our findings, it has been reported that both Pilates and resistance exercise trainings diminish hs-CRP levels in elderly men and women, but this decrease is more significant in the Pilates group (Silva Pestan et al., 2016). In addition, a recent study reveals reduced inflammatory marker of hs-CRP following aerobic, resistance and combined exercise training in diabetic patients (Jorge et al., 2011b). Moreover, a significant reduction in hs-CRP levels induced by long-term exercise intervention with moderate intensity has been reported in obese postmenopausal women (Campbell et al., 2009). It is believed that resistance training reduces hs-CRP levels by increasing muscle mass alleviating systemic inflammation and diminishing adipose tissue (Libardi et al., 2012; Nicklas and Brinkley, 2009). Furthermore, it has been revealed that chronic exercise training strengthens adaptation in skeletal muscle and immune cells, which is associated with lower levels of inflammatory factors of hs-CRP in active subjects (Nicklas and Brinkley, 2009). Also, another mechanism that may be involved in hs-CRP reduction following current exercise trainings is reduced body fat mass especially abdominal obesity (Silva Pestan et al., 2016; Jorge et al., 2011b). In contrast, other studies have not reported any significant change in the plasma levels of hs-CRP following combined exercise training (Libardi et al., 2012) and low, moderate and high intensity exercise trainings (Huffman et al., 2006) in sedentary overweight to mildly obese men and women. The discrepancy with the results of this study could be attributed to differences in subjects and type of exercise training.

In order to avoid entering another variable into the training protocol, no changes were made in the diet regimens of the participants. Although subjects were asked to maintain their normal eating habits, caloric intake and nutrient type of participant were not controlled during protocol study. In reality, the amount of consumed calories and the type of nutrition can affect the obesity-related factors (Mogharnasi et al., 2019). Based on the results of the current study, we cannot conclude that observed improvement in vaspin, hs-CRP, and nesfatin-1 are simply due to endurance and resistance exercise training. Therefore, one of the major limitations of this study is lack of a controlled diet. Hence, it is recommended that investigators focus on examining concurrent effects of nutrition and exercise training in future studies.

## 5. Conclusion

In conclusion, both endurance and resistance exercise training protocols can improve the cardiovascular health of overweight and obese women by improving obesity-related factors.

## Acknowledgments

We thank the participant for their valuable assistance with us in carrying out the protocols.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.obmed.2018.12.006>.

## Author contributions statement

All authors conceived the study and its design and coordination. MM, HT and NA-D were involved in the data collection, data analysis, and drafting of the manuscript. Finally, all authors read and approved the final version of the manuscript, and agreed with the order of presentation of the authors.

## Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

## Conflicts of interest

The authors declare that they have no conflict of interest.

## References

- Azamar-Llamas, D., Hernández-Molina, G., Ramos-Ávalos, B., Furuzawa-Carballeda, J., 2017. Adipokine contribution to the pathogenesis of osteoarthritis. *Mediat. Inflamm.* 2017, 1–26 Article ID 5468023.
- Bastien, M., Poirier, P., Lemieux, I., Després, J.P., 2014. Overview of epidemiology and contribution of obesity to cardiovascular disease. *Prog. Cardiovasc. Dis.* 56 (4), 369–381.
- Campbell, P.T., Campbell, K.L., Wener, M.H., Wood, B., Potter, J.D., McTIERNAN, A.N., Ulrich, C.M., 2009. A yearlong exercise intervention decreases CRP among obese postmenopausal women. *Med. Sci. Sports Exerc.* 41 (8).
- Chang, H.M., Park, H.S., Park, C.Y., Song, Y.S., Jang, Y.J., 2010. Association between serum vaspin concentrations and visceral adipose tissue in Korean subjects. *Metab. Clin. Exp.* 59 (9), 1276–1281.
- Chaolu, H., Asakawa, A., Ushikai, M., Li, Y.X., Cheng, K.C., Li, J.B., Zoshiki, T., Terashi, M., Tanaka, C., Atsuchi, K., Sakoguchi, T., 2011. Effect of exercise and high-fat diet on plasma adiponectin and nesfatin levels in mice. *Exp. Ther. Med.* 2 (2), 369–373.
- Choi, S.H., Kwak, S.H., Lee, Y., Moon, M.K., Lim, S., Park, Y.J., Jang, H.C., Kim, M.S., 2011. Plasma vaspin concentrations are elevated in metabolic syndrome in men and are correlated with coronary atherosclerosis in women. *Clin. Endocrinol.* 75 (5), 628–635.
- Donges, C.E., Duffield, R., Drinkwater, E.J., 2010. Effects of resistance or aerobic exercise training on interleukin-6, C-reactive protein, and body composition. *Med. Sci. Sports Exerc.* 42 (2), 304–313.
- Fayh, A.P., Lopes, A.L., da Silva, A.M., Reischak-Oliveira, A., Friedman, R., 2013. Effects of 5% weight loss through diet or diet plus exercise on cardiovascular parameters of obese: a randomized clinical trial. *Eur. J. Nutr.* 52 (5), 1443–1450.
- Feijóo-Bandín, S., Rodríguez-Penas, D., García-Rúa, V., Mosquera-Leal, A., González-Juanatey, J.R., Lago, F., 2016. Adipokines at the cardiovascular system: role in health and disease. *SM J. Endocrinol. Metab.* 2 (1), 1009.
- Festa, A., D'agostino, R., Howard, G., Mykkanen, L., Tracy, R.P., Haffner, S.M., 2000. Chronic subclinical inflammation as part of the insulin resistance syndrome: the Insulin Resistance Atherosclerosis Study (IRAS). *Circulation* 102 (1), 42–47.
- Gao, F.F., Liu, G.L., Zheng, R.X., Jiang, L.H., Bao, P.L., 2013. Correlation between vaspin concentration and insulin sensitivity in the visceral adipose tissue of young obese rats. *Zhongguo dang dai er ke za zhi Chin. J. Contemp. Pediatr.* 15 (1), 71–74.
- Hida, K., Wada, J., Eguchi, J., Zhang, H., Baba, M., Seida, A., Hashimoto, I., Okada, T., Yasuhara, A., Nakatsuka, A., Shikata, K., 2005. Visceral adipose tissue-derived serine protease inhibitor: a unique insulin-sensitizing adipocytokine in obesity. *Proc. Natl. Acad. Sci. U. S. A.* 102 (30), 10610–10615.
- Hill, A.M., Coates, A.M., Buckley, J.D., Ross, R., Thielecke, F., Howe, P.R., 2007. Can ECG reduce abdominal fat in obese subjects? *J. Am. Coll. Nutr.* 26 (4), 396S–402S.
- Hordern, M.D., Dunstan, D.W., Prins, J.B., Baker, M.K., Singh, M.A., Coombes, J.S., 2012. Exercise prescription for patients with type 2 diabetes and pre-diabetes: a position statement from exercise and sport science Australia. *J. Sci. Med. Sport* 15 (1), 25–31.
- Huffman, K.M.I., Samsa, G.P., Slentz, C.A., Duscha, B.D., Johnson, J.L., Bales, C.W., Tanner, C.J., Houmard, J.A., Kraus, W.E., 2006. Response of high-sensitivity C-reactive protein to exercise training in an at-risk population. *Am. Heart J.* 152 (4), 793–800.
- Jackson, A.S., Pollock, M.L., Ward, A.N., 1980. Generalized equations for predicting body density of women. *Med. Sci. Sports Exerc.* 12 (3), 175–181.
- Jakicic, J.M., Clark, K.R., Coleman, E., Donnelly, J.E., Foreyt, J.O., Melanson, E.D., Volek, J., Volpe, S.L., 2001. Appropriate intervention strategies for weight loss and prevention of weight regain for adults. *Med. Sci. Sports Exerc.* 33 (12), 2145–2156.
- Jorge, M.L., de Oliveira, V.N., Resende, N.M., Paraiso, L.F., Calixto, A., Diniz, A.L., Resende, E.S., Ropelle, E.R., Carvalheira, J.B., Espindola, F.S., Jorge, P.T., 2011a. The effects of aerobic, resistance, and combined exercise on metabolic control, inflammatory markers, adipocytokines, and muscle insulin signaling in patients with type 2 diabetes mellitus. *Metabolism* 60 (9), 1244–1252.
- Jorge, M.L., de Oliveira, V.N., Resende, N.M., Paraiso, L.F., Calixto, A., Diniz, A.L., Resende, E.S., Ropelle, E.R., Carvalheira, J.B., Espindola, F.S., Jorge, P.T., 2011b. The effects of aerobic, resistance, and combined exercise on metabolic control, inflammatory markers, adipocytokines, and muscle insulin signaling in patients with type 2 diabetes mellitus. *Metabolism* 60 (9), 1244–1252.
- Kamal, N.N., Ragy, M.M., 2012. The effects of exercise on C-reactive protein, insulin, leptin and some cardiometabolic risk factors in Egyptian children with or without metabolic syndrome. *Diabetol. Metab. Syndrome* 4 (1), 27.
- Kang, J., Rashti, S.L., Tranchina, C.P., Ratamess, N.A., Faigenbaum, A.D., Hoffman, J.R., 2009. Effect of preceding resistance exercise on metabolism during subsequent aerobic session. *Eur. J. Appl. Physiol.* 107 (1), 43–50.
- Kim, J.Y., Kim, E.S., Jeon, J.Y., Jekal, Y., 2011. Improved insulin resistance, adiponectin and liver enzymes without change in plasma vaspin level after 12 weeks of exercise training among obese male adolescents. *Kor. J. Obes.* 20 (3), 138–146.
- Klötting, N., Berndt, J., Kralisch, S., Kovacs, P., Fasshauer, M., Schön, M.R., Stumvoll, M., Blüher, M., 2006. Vaspin gene expression in human adipose tissue: association with obesity and type 2 diabetes. *Biochem. Biophys. Res. Commun.* 339 (1), 430–436.
- Koenig, W., 2013. High-sensitivity C-reactive protein and atherosclerotic disease: from improved risk prediction to risk-guided therapy. *Int. J. Cardiol.* 168 (6), 5126–5134.
- Kubota, Y., Moriyama, Y., Yamagishi, K., Tanigawa, T., Noda, H., Yokota, K., Harada, M., Inagawa, M., Oshima, M., Sato, S., Iso, H., 2010. Serum vitamin C concentration and hs-CRP level in middle-aged Japanese men and women. *Atherosclerosis* 208 (2), 496–500.
- Lakka, T.A., Lakka, H.M., Rankinen, T., Leon, A.S., Rao, D.C., Skinner, J.S., Wilmore, J.H., Bouchard, C., 2005. Effect of exercise training on plasma levels of C-reactive protein in healthy adults: the HERITAGE Family Study. *Eur. Heart J.* 26 (19), 2018–2025.
- Lee, M.K., Jekal, Y., Im, J.A., Kim, E., Lee, S.H., Park, J.H., Chu, S.H., Chung, K.M., Lee, H.C., Oh, E.G., Kim, S.H., 2010. Reduced serum vaspin concentrations in obese children following short-term intensive lifestyle modification. *Clin. Chim. Acta* 411 (5), 381–385.
- Li, Q.C., Wang, H.Y., Chen, X., Guan, H.Z., Jiang, Z.Y., 2010. Fasting plasma levels of nesfatin-1 in patients with type 1 and type 2 diabetes mellitus and the nutrient-related fluctuation of nesfatin-1 level in normal humans. *Regul. Pept.* 159 (1–3), 72–77.
- Libardi, C.A., De, G.S., Cavaglieri, C.R., Madruga, V.A., Chacon-Mikahil, M.P., 2012. Effect of resistance, endurance, and concurrent training on TNF- $\alpha$ , IL-6, and CRP. *Med. Sci. Sports Exerc.* 44 (1), 50–56.
- Martins, R.A., Neves, A.P., Coelho-Silva, M.J., Verissimo, M.T., Teixeira, A.M., 2010. The effect of aerobic versus strength-based training on high-sensitivity C-reactive protein in older adults. *Eur. J. Appl. Physiol.* 110 (1), 161–169.
- Martos-Moreno, G.Á., Kratzsch, J., Körner, A., Barrios, V., Hawkins, F., Kiess, W., Argente, J., 2011. Serum visfatin and vaspin levels in prepubertal children: effect of obesity and weight loss after behavior modifications on their secretion and relationship with glucose metabolism. *Int. J. Obes.* 35 (10), 1355–1362.
- Mogharnasi, M., Cheragh-Birjandi, K., Cheragh-Birjandi, S., TaheriChadorneshin, H., 2017. The effects of resistance and endurance training on risk factors of vascular inflammation and atherogenesis in non-athlete men. *Int. Med. Appl. Sci.* 9 (4), 185–190.
- Mogharnasi, M., TaheriChadorneshin, H., Papoli-Baravati, S.A., Teymuri, A., 2019. Effects of upper-body resistance exercise training on serum nesfatin-1 level, insulin resistance, and body composition in obese paraplegic men. *Disabil. Health J.* 12 (1), 29–34.
- Nabil, N., El Sayyad, M., 2015. Moderate exercise training has anorexigenic effect associated with improved oxidative stress in obese women. *Int. J. Nutr. Metab.* 7 (4), 52–61.
- Nicklas, B.J., Brinkley, T.E., 2009. Exercise training as a treatment for chronic inflammation in the elderly. *Exerc. Sport Sci. Rev.* 37 (4), 165.
- Oberbach, A., Kirsch, K., Lehmann, S., Schlichting, N., Fasshauer, M., Zarse, K., Stumvoll, M., Ristow, M., Blüher, M., Kovacs, P., 2010. Serum vaspin concentrations are decreased after exercise-induced oxidative stress. *Obes. Facts* 3 (5), 328–331.
- Ouchi, N., Parker, J.L., Lugus, J.J., Walsh, K., 2011. Adipokines in inflammation and metabolic disease. *Nat. Rev. Immunol.* 11 (2), 85.
- Pan, W., Hsueh, H., Kastin, A.J., 2007. Nesfatin-1 crosses the blood-brain barrier without saturation. *Peptides* 28 (11), 2223–2228.
- Parrinello, C.M., Lutsey, P.L., Ballantyne, C.M., Folsom, A.R., Pankow, J.S., Selvin, E., 2015. Six-year change in high-sensitivity C-reactive protein and risk of diabetes, cardiovascular disease, and mortality. *Am. Heart J.* 170 (2), 380–389.
- Sakurai, T., Ogasawara, J., Shirato, K., Izawa, T., Oh-ishi, S., Ishibashi, Y., Radák, Z., Ohno, H., Kizaki, T., 2017. Exercise training attenuates the dysregulated expression of adipokines and oxidative stress in white adipose tissue. *Oxid. Med. Cell. Longev.* 2017, 1–12 Article ID 9410954.
- Silva Pestan, M.D., Martins Netto, E., Castro Silva Pestana, M., Silva Pestana, V., Schinoni, M.I., 2016. Pilates versus resistance exercise on the serum levels of hs-CRP, in the abdominal circumference and body mass index (BMI) in elderly individuals. *Motricidade* 12 (1).
- Stengel, A., Goebel, M., Yakubov, I., Wang, L., Witcher, D., Coskun, T., Taché, Y., Sachs, G., Lambrecht, N.W., 2008. Identification and characterization of nesfatin-1 immunoreactivity in endocrine cell types of the rat gastric oxyntic mucosa. *Endocrinology* 150 (1), 232–238.
- Tsuchiya, T., Shimizu, H., Yamada, M., Osaki, A., Oh-I, S., Ariyama, Y., Takahashi, H., Okada, S., Hashimoto, K., Satoh, T., Kojima, M., 2010. Fasting concentrations of nesfatin-1 are negatively correlated with body mass index in non-obese males. *Clin. Endocrinol.* 73 (4), 484–490.
- Vink, R.G., Roumans, N.J., Mariman, E.C., van Baak, M.A., 2017. Dietary weight loss-induced changes in RBP4, FFA, and ACE predict weight regain in people with overweight and obesity. *Physiol. Rep.* 5 (21), e13450.
- Wang, Y.M., Wang, W.P., Wang, L.P., Lü, Q.H., Zhou, X.H., 2010. Calorie control increased vaspin levels of serum and periepididymal adipose tissue in diet-induced obese rats in association with serum free fatty acid and tumor necrosis factor alpha. *Chin. Med. J.* 123 (7), 936–941.
- Yang, M., Zhang, Z., Wang, C., Li, K., Li, S., Boden, G., Li, L., Yang, G., 2012. Nesfatin-1 action in the brain increases insulin sensitivity through Akt/AMPK/TORC2 pathway in diet-induced insulin resistance. *Diabetes* 61 (8), 1959–1968.
- Yazici, A.G., 2015. Relationship and interaction between anaerobic sports branches and serum nesfatin-1. *Turkiye Fiziksel Tip Ve Rehabilitasyon Dergisi-Turk. J. Phys. Med. Rehabil.* 61 (3), 234–240.