Orginal Article

Open Access

Iranian Journal of Diabetes and Obesity (IJDO)

Effect of Interval Resistance Training with Different Intensities on Some Selected Adipokines in Obese Men

Ensieh Yazdkhasti[1], Farnaz Seifi Skishahr[2*], Reza Farzizadeh[3]

[1]Ph.D. Students in Sports Physiology, Department of Physical Education and Sport Science, Faculty of Educational Sciences and Psychology, University of Mohagheghe Ardabili, Ardabil, Iran.
[2]Associate Professor Department of Physical Education and Sport Science, Faculty of Educational Sciences and Psychology, University of Mohagheghe Ardabili, Ardabil, Iran.
[3]Assistant Professor Department of Physical Education and Sport Science, Faculty of Educational Sciences and Psychology, University of Mohagheghe Ardabili, Ardabil, Iran.

Abstract

Objective: Adipose tissue-derived adipokines affect cardiometabolic health. This study aimed to evaluate the effect of interval resistance training with different intensities on leptin, adiponectin, and acrosin levels in obese males.

Materials and Methods: Fourty-four obese males (27.70± 3.02 years) were randomly allocated into four groups: high intensity (HI), moderate intensity (MI), low intensity (LI), and control (C). Subjects of the HI, MI, and LI groups implemented the training program for 12 weeks, three sessions a week with intensities corresponding 80% 1RM, 60% 1RM, and 40% 1RM,, respectively. Body composition and plasma levels of leptin, adiponectin, and asprosin were evaluated before and after interval resistance training at different intensities.

Results: Following exercise, serum levels of leptin and asporin were significantly reduced in all groups ($P<$ 0.01). The greatest reduction of leptin and asporin levels was recorded in the HI group ($P=$ 0.001, $P=$ 0.01) for leptin and asporsin levels, respectively. The level of adiponectin significantly decreased after exercise in all groups ($P<$ 0.001, F= 12.44). The greatest reduction was observed in the HI group compared with the MI and UI group ($P=$ 0.001).

Conclusion: Interval resistance training with different intensities can cause significant changes in leptin, asprosin, and adiponectin levels in obese men, and this improvement was better at high intensities.

Keywords: Obesity, Leptin, Adiponectin, Asprosin, Interval resistance training

QR Code:

Citation: Yazdkhasti E, Seifi Skishahr F, Farzizadeh R. Effect of Interval Resistance Training with Different Intensities on Some Selected Adipokines in Obese Men. IJDO 2023; 15 (4) :208-217

URL: http://ijdo.ssu.ac.ir/article-1-832-en.html

10.18502/ijdo.v15i4.14554

Article info:

Received: 25 July 2023
Accepted: 12 October 2023
Published in December 2023

This is an open access article under the (CC BY 4.0)

Corresponding Author:

Farnaz Seifi-SkiShahr, Department of Physical Education and Sport Science, Faculty of Education and Psychology, University of Mohagheghe Ardabili, Ardabil, Iran.

Tel: (98) 914 353 0227
Email: f.seify@yahoo.com
Orcid ID: 0000-0002-4065-379X

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

Introduction

Obesity is a significant public health concern affecting more than half a billion people worldwide. Generally, the lack of balance between calorie intake and consumption leads to obesity; it is not only limited to developed countries but also to developing nations Changes in diet styles in terms of more fat, more meat, added sugars, bigger portion sizes, and reduced physical activity level are known as the main factors for increasing overweight and cardiovascular diseases (1).

Current scientific evidence indicates that adipokines affect cardiometabolic health (2). Adipose tissue is a type of connective tissue that is used throughout the body but is mainly found in visceral and subcutaneous sources. (3). Fat tissue function is important for health. It is not only an energy-conserving tissue but can release many substances that act in paracrine, autocrine, endocrine, and apocrine ways to maintain metabolic homeostasis. These substances include immune-modulating proteins collectively known as adipokines oradipocytokines (4). Chronic low-grade inflammation in obesity induces changes in adipokine expression the effects of adipokines on the inflammatory system can be an effective factor in the pathogenesis of obesity-related diseases (5). Adipokines include leptin, adiponectin, asporsin, and many others (2).

Adiponectin is an anti-inflammatory protein beneficial for cardiovascular health that originates from adipose tissue found in plasma (4). Plasma adiponectin levels were reduced in obese patients, particularly those with visceral obesity. The association between obesity and low adiponectin levels suggested the possibility of adiponectin acting as a biomarker for metabolic syndrome (6).With its insulin-like effect, adiponectin increases glucose uptake and fatty acid oxidation, which prevents the spread of insulin resistance and leads to the improvement of metabolic disorders (7). In addition, it can inhibit the production of inflammatory cytokines such as IL-6 and TNF-α in macrophages through the activation of ERK1/2 and NF-kβ messengers and suppresses the expression of TNF-α mRNA in adipocytes, and produce anti-inflammatory cytokines such as IL-1r and IL-1 in monocytes. Metabolic syndrome is associated with mild chronic inflammation (8).

Leptin is a cytokine that is produced by white adipose tissue, and its secretion increases according to the volume of adipose tissue or fat cells. Leptin is known as a pro-inflammatory factor and is harmful to heart health. (9). Despite its anorexigenic effects, high leptin levels and leptin resistance caused by its low transport through the blood–brain barrier have been reported in patients (10). Higher leptin levels were associated with an increased incidence of metabolic syndrome both in an observational study with a 14-year follow-up period (11). Furthermore, high levels of circulating leptin in obese individuals are a relatively common phenomenon known as leptin resistance. Leptin has a pro-inflammatory effect and increases the production of pro-inflammatory factors such as tumor alpha necrosis factor (TNF-α) and interleukin-1 (IL-1) (12).

Asprosin is a fasting-induced protein hormone secreted by fat tissue and absorbed by the liver. It is a result of the C-terminal cleavage of profibrillin (encoded by FBN1) and is supposed to be a glucogenic protein hormone. At the circulating level, asprosin is mainly secreted by white adipose tissue, which increases with obesity. As white adipose tissue increases, an increase in acrosin levels occurs. This in turn activates agouti-related peptide neurons (AgRP) via cAMP to increase appetite and food intake (13). The fasting state (basal glucose) increases the circulating acrosin level, and this level is reduced after eating (high glucose). Blood glucose plays an inhibitory role in the negative feedback loop on plasma acrosin levels (14); however, the effects of other adipophines, such as acrosin, on heart health have not yet been determined.

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

Regular physical activity is an effective solution to fight obesity with the aim of improving cardiovascular and metabolic health. Physical activity has many effects in reducing the accumulation of visceral fat, thus reducing insulin resistance, metabolic syndrome, systemic inflammation, and cardiovascular diseases (15). Interval resistance training is a mode of resistance training with short rest intervals that involves different parts of the body separately comprehending repeated movements. Physical activity is an easy way and a good solution to lose weight and improve cardiovascular health. The benefits of this training include higher heart rate, metabolism rate, and energy consumption and less local muscle fatigue compared with traditional resistance training (16). Previous studies did not show changes in adiponectin and leptin levels following short-term resistance training (17). In addition, 12 weeks of circuit resistance training did not change serum adiponectin concentration in obese men (18). Ghadiri et al. (2014) In their research, they concluded that performing resistance training for 3 months causes a significant decrease in leptin concentration (19). Akbarpour (2013) showed that regular resistance training increases the plasma level of adiponectin in obese men (20). Exercise activity can be considered an effective factor in improving obesity. However, information about the effect of physical activity on the levels of leptin, adiponectin, and inflammatory indicators is contradictory (21). In addition, Ko et al. (2019) stated that aerobic exercise is associated with a decrease in hepatic acrosin levels in diabetic rats and suppresses blood glucose levels (22). Wichek et al. (2018) showed a significant decrease in women's asprosin levels 3 min after anaerobic exercise (23). Few studies have investigated the effect of interval resistance training on the levels of leptin, adiponectin, and asprosin and reported different results of the effect of interval resistance training on these levels.

Previous studies on modulation of adipokine secretion and signaling, i.e., reduction of inflammatory adipokines and hypersecretion of anti-inflammatory adipokines, generally focused on another types of resistance exercises (24). In the present study, we examined the effect of different intensities of interval resistance training on three adipose tissue-derived adipokines (i.e., leptin, adiponectin, and asporsin) in obese males for possible consequences for the treatment of obesity and improvement of cardiometabolic health.

Material and methods

In this semi-experimental research, obese men with a body mass index $30 \leq x \leq 35$ kg/m2 and an age range of 23-32 years willingly participated in the research and provided written informed consent. They were selected at convenience by fully complying with all health protocols and having vaccination cards.

The inclusion criteria for this research include not being addicted to drugs and alcohol, not having a history of regular exercise activity for at least 6 months, no history of various diseases, and no physical injury. Exclusion criteria were the occurrence of neurological, muscular, and skeletal problems during the research, contracting Covid-19 disease, and not participating in exercise activity for more than 3 consecutive sessions or 4 sessions.

The sample size considering $\beta = 0.1$ and $\alpha = 0.05$ using the following formula with 80 % power and 5 % significance level and assuming variance heterogeneity (higher sample size compared to homogeneity Variance results) and Assuming that the standardized value of the effect size is 0.75 and the ratio of the variances of the two groups is equal to Z=1.5 and the same considering the number of members of the control and case groups (φ=1).

Finally, 44 subjects consented to participate in the study and were selected into the High Intensity HI (n= 11), Moderate Intensity MI (n= 11), Low Intensity LI (n= 11), and Control C (n= 11) groups. It is noteworthy that during the implementation of the research, all

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

participants were under the supervision of a specialist doctor to avoid any possible risks. Moreover, the researchers assured the participants that their personal information would be confidential and that the researchers would report the sessions generally at the end. They also had the option to withdraw from the exercise if they did not want to continue cooperation. The standard method was used to evaluate the height and weight of the subjects. Body mass index was obtained using the formula, weight divided by the square of height (25).

Exercise protocol

The exercise program included resistance interval training with different intensities and 3 training days per week for 3 months. Each training session comprised a 10-min warm-up, main stage, and 10-min cool-down stage. The warm-up consisted of (continuous jogging at moderate intensity and stretching) and The main stage consisted of 50–60 min of resistance training including front leg, back leg, abdominal (lying and sitting), chest press, underarm cable pull from the back, back (loin filet), hog leg, Swedish swimming, military press, front arm pull-up, and back arm pull-up. After familiarizing the participants with the correct way of performing the movements by the trainer, the researchers took from the participants a test of one maximum repetition of 1RM by the Berzyski method for all the movements of the resistance program to apply training intensity. The researchers then divided the groups into 3 training groups at different intensities according to Table 1 (26).

The exercise volume was calculated according to the formula provided by Bichel et al. (2008) (27). The rest between the sets is active with an intensity of 20% and several 15 repetitions. Finally, participants were cooled down by running at low intensity and performing static stretching for 10 min.

Biochemical analysis

The participants' blood samples were collected in two stages to evaluate the biochemical variables: pre-test and post-test (after 12 weeks of training). Thus, in the pre-test phase, i.e., one day before the start of the training program and after 12 h of fasting, a blood sample (10 cc) was taken from the brachial vein of the subjects' right hand between 8 and 9 in the morning.

In the post-test, for preventing the acute effect of training on study variables, it was analyzed after 48 h from the last training session, similar to the pre-test stage after 12 h of fasting in the same interval of 8 to 9 in the morning, blood sampling was done. The blood samples were then quickly stored in packages containing dry ice at a temperature of 4 degrees Celsius and transferred to one of the accredited laboratories for biochemical measurement and analysis. The blood samples were centrifuged at 3000 rpm for 20 min to separate the serum.

In this study, leptin (sensitivity of the measurement method 0.2 ng/ml and intra-CV= 5.9%, inter-CV= 5.6%) and adiponectin (sensitivity of the measurement method 26 ng/ml and intra-CV= 4.9%, inter-CV= 6.7%) were measured by the ELISA method and through a kit (BioVendor, Brno, Czech Republic). Asprosin (the sensitivity of the measurement method 1 nM/L and intra-CV = 6 - 8%, inter-CV = 8 - 12%) was measured by the ELISA method and through a high-sensitivity kit (Aviscera Bioscience, United States). All steps of the ELISA measurement were performed according to the instructions of the kit.

Table 1. Study group of exercises

Training groups	Set	Repetition	Training intensity
High-intensity interval training group	3	10	**80%** 1RM
Moderate intensity interval training group	3	13	**60%** 1RM
Low-intensity interval training group	3	20	**40%** 1RM

1-RM = moved weight (kg) / 1.0278 – (number of repetitions until fatigue * 0.0278)

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

Statistical methods

The data of this research were analyzed using SPSS version 22 software. The Shapiro–Wilk test was used for the normality of the data, paired t-test was used to compare pre-test and post-test changes in each group, and one-way analysis of variance and Tukey's post hoc test were used to compare between groups. The statistical significance was determines as <0.05.

Ethical considerations

All ethical considerations for the participants have been considered research with Iranian clinical trial registration number IRCT 20191203045588N2. The study was approved by the Ethical Committee of Mohaghegh Ardabili University with ID IR.UMA.REC. 2021.024.

Results

The descriptive findings of this study are presented in Table 2. Table 3 shows the mean values of serum leptin concentration. It decreased significantly after training in the HI (P< 0.001), p MI (P< 0.001), and LI (P< 0.001) groups compared with before training. There was no significant between-group difference in either pre-training or post-training values (F= 27.53, P= 0.001).

The mean values of serum adiponectin concentration was increased in the HI (P< 0.001) and MI (P< 0.001) groups, whereas the subjects of the LI group did not show any significant change (P= 0.051). The greatest value was observed in the HI group compared with the MI and LI groups (F= 12.44, P< 0.001).

Table 2. Pre-test and post-test values of research indicators in the studied groups (standard deviation)

Variable	Groups	Pre-test (mean ± standard deviation)	Post-test (mean ± standard deviation)
Weight (kg)	Control	97.73 (±3.81)	97.41 (± 2.96)
	LI	97.92 (± 2.85)	95.19 (± 2.99)
	MI	97.92 (± 2.85)	95.19 (± 2.99)
	HI	98.87 (± 1.91)	95.31 (± 2.42)
Body fat percentage	Control	26.03 (± 2.90)	25.90 (± 3.80)
	LI	25.60 (± 2.20)	25.20 (± 3.10)
	MI	27.70 (± 3.60)	22.70 (± 2.80)
	HI	26.60 (± 3.50)	23.50 (± 3.30)
Fat-free body mass	Control	74.40 (± 8.70)	74.80 (± 9.30)
	LI	67.40 (± 3.60)	68.30 (± 3.30)
	MI	71.40 (± 8.70)	74.80 (± 9.30)
	HI	74.50 (± 8.40)	76.20 (± 6.60)
BMI (kg/m2)	Control	35.32 (± 2.52)	35.19 (±2.26)
	LI	35.47 (± 1.51)	34.49 (± 1.60)
	MI	35.47 (±1.51)	34.49 (±1.60)
	HI	36.18 (± 0.81)	34.92 (± 1.0)

Table 3. Measured indicators before and after the periodic resistance training test (mean ± standard deviation)

Variable	Groups	Pre-test (mean ± standard deviation)	Post-test (mean ± standard deviation)	*P* Intergrou	Analysis of variance *P*-value	F
Leptin (ng/ml)	Control	16.12 (± 1/00)	16.15 (± 0/73)	0.9	*0.001	27.53
	LI	16.03 (± 0/73)	13.21 (± 0/95)	*0.001		
	MI	16.10 (± 0/92)	12.23 (± 1/01)	*0.001		
	HI	16.49 (± 0/72)	11.13 (± 1/26)	*0.001		
Adiponectin (ng/ml)	Control	8.13(± 1/34)	8.05 (±1/14)	0.8	*0.001	12.44
	LI	8.42(± 0/90)	10.00 (± 1/14)	0.051		
	MI	8.48 (± 0/89)	10.84 (±1/04)	*0.001		
	HI	8.16 (± 0/78)	11.87 (± 0/87)	*0.001		
Asprosin (ng/ml)	Control	9.46 (± 0/82)	9.13 (± 0/54)	0.21	*0.001	29.90
	LI	9.52 (± 0/52)	8.21 (± 0/54)	*0.001		
	MI	9.27 (± 0/67)	6.26 (± 0/43)	*0.01		
	HI	9.5 (± 0/88)	5.87 (±0/59)	*0.01		

* sign of significant difference (P≥ 0.05) compared to before the intervention

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

The levels of asporin were significantly decreased after training in all groups ($P< 0.001$, F= 29.90). The biggest decrease was seen in the HI group compared with the MI and LI groups ($P< 0.01$).

Discussion

The results revealed that 12-week interval resistance training with different intensities decreased leptin and asporsin levels as offensive adipokines and increased adiponectin levels as defensive adipokines in obese males. This training program resulted in a significant decrease in leptin levels in all groups; however, HI resistance training had a greater effect on reducing serum leptin levels. These results are consistent with various findings (19,28-30). Haghigi et al. (2008). indicated that the reasons for the effect of resistance training on leptin are increased glucose uptake by peripheral tissues in the presence of lactate, acidosis, increased adrenal sympathetic stimulation and energy consumption, glycogen depletion, and inhibition of glycolysis (31). Fatouros et al. (2005) research, low, moderate and high-intensity resistance training decreased leptin levels inolder people. Due to the decrease in letin concentration and the increase in adiponectin induced to increase insulin sensitivity during exercise, Factors considered the increase in energy consumption and oxygen consumption in moderate- and high-intensity training (32). Moonikh et al. (2015) showed that plasma leptin and body composition did not change after 6 weeks of resistance training. It seems that the lack of change in serum leptin level is due to the lack of change in body fat percentage and lack of exercise, but these exercises increase muscle strength (33). Also, in other studies, no change of leptin in response to resistance exercise has been reported (34, 35).The difference in the (aerobic, resistance, combined) training program, the type of (patient, old, young, etc.) participants, and the training protocol (type of training, intensity, duration) can be a reason for the contradiction in these findings. One of the possible mechanisms for justifying the reduction of leptin due to resistance training is probably the reduction of body fat and its reserves following this type of training. Thus, it has become clear that resistance training stimulates muscle protein synthesis and consequently increases body muscle mass. This causes an increase in the total energy consumed during rest and a decrease in fat and its total storage in the body, thus preventing the release of leptin in obese and overweight people (32).

Another finding of this research showed a significant difference in the adiponectin variable between the resistance training groups with moderate and high intensity after twelve weeks of resistance training with different intensities; therefore, high-intensity resistance training has a greater increase in adiponectin levels. Ghorbani Ganjeh et al.(2020) state that, cellular adaptations such as increasing adiponectin levels and stimulating the AMPK signaling pathway resistance training with moderate training intensity can help to improve the lipid profile of people and improve their body composition (26). Fatouros et al.(2005) show that resistance training with high and moderate intensity can increase the adiponectin level (32). in return, Asad et al.(2012) shows that resistance training does not change the resting levels of adiponectin in overweight men who did not exercise (36). Haghighi (2015), Ahmadizad (2007), and Klimchakova (2006) also reported that adiponectin concentration did not change after strength training (18, 37, 38). This may be due to greater stimulation of the AMPK pathway that caused a greater increase in adiponectin in the group who trained with high intensity. Various studies have revealed that an increase in adiponectin is related to an increase in catabolism; therefore, the improvement of the lipid profile through moderate-intensity exercise is attributable to the increase caused by the catabolic pathway of AMPK stimulated by adiponectin. Given that high-intensity training can increase good adipokines such as adiponectin potential through the greater use

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

of anaerobic resources and accumulation of metabolites such as AMP, it increases mitochondrial biogenesis and improves the lipid profile (39). Adiponectin stimulates glucose consumption and fatty acid oxidation and improves insulin action by activating AMP kinase in the muscle. Exercise also improves glucose consumption and fat acid oxidation by activating AMP kinase in the muscle (40). Conflicting results in this study are probably due to differences in the intensity, duration, type of exercise activity, presence or absence of metabolic diseases such as diabetes and cardiovascular diseases, weight, age, gender, and the type of participants under study.

Another finding is that resistance training with high and moderate intensity causes a greater decrease in the serum level of asprosin. Wang et al.(2019) explained that an increase in acrosin levels in obese individuals might be due to its release from other organs (41). Duerrschmid et al.(2017) and Roemer et al.(2016) noted that asprosin is an appetite-stimulating hormone that causes appetite (13,42). Therefore, the high level of acrosin in obese people may cause hunger and more food consumption, weight gain, and obesity. Ko et al.(2019) disclosed that aerobic exercise by decreasing liver acrosin levels in diabetic rats suppresses blood glucose levels (22). Wiecek et al.(2018) showed a significant decrease in acrosin levels in females 3 min after anaerobic exercise. They also explained that the acrosin level decreased in the third minute after exercise when blood sugar was high and increased 30 min after exercise when blood sugar was low. Glucose is the dominant energy source during high-intensity exercise; therefore, glucose in the blood or muscles decreases after intense exercise, and the blood glucose level becomes stable by converting it to glucose in the liver. Thus, acrosin may play an active role in this process (23). In contrast, Shumann et al.(2017) reported that anaerobic exercise had no effect on acrosin levels in obese participants (43). The type, duration, and intensity of training, experimental design, and evaluated parameters can lead to different findings in studies on the effect of exercise on acrosin (22). While there is limited literature on the effect of aerobic and anaerobic exercise on the acrosin level, no researcher has studied the effect of resistance training on the acrosin level. The location of the central receptors of acrosin in the arcuate nucleus of the hypothalamus, which helps to increase appetite, may be one of the possible mechanisms for the reduction of acrosin. The hypothalamus, as a feeding control center, regulates appetite by relying on two types of neuronal populations, proopiomelanocortin (POMC) anorectic neurons and agouti-related protein (AgRP) orexigenic neurons (44). Asprosin increases the amplitude of AgRP neurons and changes their membrane potential, which increases the activity of AgRP neurons through a G-cAMP-PKA protein axis. Nevertheless, this signaling inhibits the activity of POMC neurons in a GABA-dependent manner, thereby stimulating food intake and regulating energy homeostasis (42). Rezaeinezhad et al.(2018) showed that agouti-related protein levels decreased more in high-intensity resistance training (80% of a maximum repetition); therefore, this training intensity can be useful in reducing appetite and preventing weight gain in young men. The following resistance training, plasma volume increases and decreases agouti-related protein levels (45). It can be concluded that the decrease in acrosin concentration is due to the decrease in appetite resulting from reduced AgRP.

This study was conducted to assess the effects of interval resistance training with different intensities on leptin, adiponectin, and acrosin levels in obese males. Taking participants in groups with different intensities permitted us to conclude that intensity influences adipokine response to interval resistance training. The study has limitations such as a small sample size and no control for dietary intake and energy expenditure. In addition, participants' usual physical activity was assessed only using a questionnaire.

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

Given the abovementioned methodological limitations, the study results should be considered with caution.

Conclusions

The results of the study show that interval resistance training decreased leptin and asporsin levels as inflammatory adipokines and increased adiponectin levels as anti-inflammatory adipokines, and it was effective for cardiovascular health. However, the study revealed that these effects related to exercise intensity and interval resistance training with high intensity have more effects on the serum levels of asprosin, leptin, and adiponectin. Further research with a large sample size and long-term training programs are needed to shed light on the effects of interval resistance training on adipokine production and uncover the underlying mechanisms.

Acknowledgments

This article is an extract of the doctoral thesis. The authors express their appreciation to the participants of this study.

Funding

The research and funding were supported by Mohagheghe Ardabili, Ardabil, Iran.

Conflict of Interest

The authors declare no conflicts of interest.

Authors' contributions

All authors have accepted responsibility for the entire content of this manuscript and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved and approved the version to be published.

References

1. Ahmadi M, Abbassi Daloii A, Shadmehri S, Agghaei Bahmanbeglu N. Compare the effect of eight weeks aerobic and resistance training on Oxidant, antioxidant status and lipid profile in obese girls. Journal of Sport and Exercise Physiology. 2019;11(1):139-52.(in Persian)
2. Ouerghi N, Fradj MK, Duclos M, Bouassida A, Feki M, Weiss K, et al. Effects of high-intensity interval training on selected adipokines and cardiometabolic risk markers in normal-weight and overweight/obese young males-a pre-post test trial. Biology (Basel). 2022; 11 (6): 853.
3. Murawska-Ciał owicz E. Adipose tissue–morphological and biochemical characteristic of different depots. Advances in Hygiene and Experimental Medicine. 2017;71:466-84.
4. Esler M, Straznicky N, Eikelis N, Masuo K, Lambert G, Lambert E. Mechanisms of sympathetic activation in obesity-related hypertension. Hypertension. 2006;48(5):787-96.
5. Ouerghi N, Fradj MK, Talbi E, Bezrati I, Feki M, Bouassida A. Association of selected adipokines with metabolic syndrome and cardio-metabolic risk factors in young males. Cytokine. 2020;133:155170.
6. Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. The Journal of clinical investigation. 2006;116(7):1784-92.
7. de Winter CD, Bastiaanse LP, Hilgenkamp TI, Evenhuis HM, Echteld MA. Overweight and obesity in older people with intellectual disability. Research in developmental disabilities. 2012;33(2):398-405.
8. Azali Alamdari K, Khalafi M. The Effect of Aerobic Exercise on Serum Levels of Adiponectin and CRP and Insulin Resistance in Women with Metabolic Syndrome. Sport Physiology & Management Investigations. 2019;11(4):53-63.(in Persian)
9. Esteghamati A, Khalilzadeh O, Anvari M, Rashidi A, Mokhtari M, Nakhjavani M. Association of serum leptin levels with homeostasis model assessment–estimated insulin resistance and metabolic syndrome: the key role of central obesity. Metabolic

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

syndrome and related disorders. 2009;7(5):447-52.(in Persian)
10. Ahima RS. Revisiting leptin's role in obesity and weight loss. The Journal of clinical investigation. 2008;118(7):2380-3.
11. Lee KW, Shin D. Prospective associations of serum adiponectin, leptin, and leptin-adiponectin ratio with incidence of metabolic syndrome: the Korean Genome and Epidemiology Study. International Journal of Environmental Research and Public Health. 2020;17(9):3287.
12. Lee SW, Rho JH, Lee SY, Kim JH, Cheong JH, Kim HY, et al. Leptin protects rat articular chondrocytes from cytotoxicity induced by TNF-α in the presence of cyclohexamide. Osteoarthritis and Cartilage. 2015;23(12):2269-78.
13. Romere C, Duerrschmid C, Bournat J, Constable P, Jain M, Xia F, et al. Asprosin, a fasting-induced glucogenic protein hormone. Cell. 2016;165(3):566-79.
14. Kajimura S. Advances in the understanding of adipose tissue biology. Nature reviews endocrinology. 2017;13(2):69-70.
15. Hayashino Y, Jackson JL, Hirata T, Fukumori N, Nakamura F, Fukuhara S, et al. Effects of exercise on C-reactive protein, inflammatory cytokine and adipokine in patients with type 2 diabetes: a meta-analysis of randomized controlled trials. Metabolism. 2014;63(3):431-40.
16. Sepehrirad M, Valipour Dehnou V, Fathi MO. Effects of hict on serum lipids and glucose levels in elderly women. Iran Journal of Nursing. 2018;31(115):20-8.(in Persian)
17. Ara I, Perez-Gomez J, Vicente-Rodríguez G, Chavarren J, Dorado C, Calbet JA. Serum free testosterone, leptin and soluble leptin receptor changes in a 6-week strength-training programme. British journal of nutrition. 2006;96(6):1053-9.
18. Ahmadizad S, Haghighi AH, Hamedinia MR. Effects of resistance versus endurance training on serum adiponectin and insulin resistance index. European journal of Endocrinology. 2007;157(5):625-31.
19. Ghaderi M, Azizbeigi K. Hormonal responses to acute resistance exercise after branched-chain amino acids supplementation. International Medical Journal. 2014;22(1):1-5.
20. Akbarpour M. The effect of aerobic training on serum adiponectin and leptin levels and inflammatory markers of coronary heart disease in obese men. Biology of sport. 2013;30(1):21-7.
21. Bouassida A, Lakhdar N, Benaissa N, Mejri S, Zaouali M, Zbidi A, et al. Adiponectin responses to acute moderate and heavy exercises in overweight middle aged subjects. The Journal of sports medicine and physical fitness. 2010;50(3):330-5.
22. Ko JR, Seo DY, Kim TN, Park SH, Kwak HB, Ko KS, et al. Aerobic exercise training decreases hepatic asprosin in diabetic rats. Journal of clinical medicine. 2019;8(5):666-73.
23. Wiecek M, Szymura J, Maciejczyk M, Kantorowicz M, Szygula Z. Acute anaerobic exercise affects the secretion of asprosin, irisin, and other cytokines–a comparison between sexes. Frontiers in physiology. 2018;9:1782.
24. Hazley L, Ingle LE, Tsakirides C, Carroll S, Nagi D. Impact of a short-term, moderate intensity, lower volume circuit resistance training programme on metabolic risk factors in overweight/obese type 2 diabetics. Research in Sports Medicine. 2010;18(4):251-62.
25. Ablove T, Binkley N, Leadley S, Shelton J, Ablove R. Body mass index continues to accurately predict percent body fat as women age despite changes in muscle mass and height. Menopause. 2015;22(7):727-30.
26. Qorbani Ganjeh Z, Gholami M, Nikbakht H. Effect of resistance training with different intensities on adiponectin and lipid profiles in overweight women. The Scientific Journal of Rehabilitation Medicine. 2019;8(4):47-55.(in Persian)
27. Baechle TR, Earle RW, editors. Essentials of strength training and conditioning. Human kinetics; 2008.
28. Burr JF, Rowan CP, Jamnik VK, Riddell MC. The role of physical activity in type 2 diabetes prevention: physiological and practical perspectives. The Physician and sportsmedicine. 2010;38(1):72-82.
29. Jürimäe J, Jürimäe T. Leptin responses to short term exercise in college level male rowers. British Journal of Sports Medicine. 2005;39(1):6-9.
30. Prestes J, Shiguemoto G, Botero JP, Frollini A, Dias R, Leite R,et al. Effects of resistance training on resistin, leptin, cytokines, and muscle force in elderly post-menopausal

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]

women. Journal of sports sciences. 2009;27(14):1607-15.
31. Haghighi A, Hamedinia M. The effect of resistance training on serum leptin in obese men. Research Sport Science. 2008;20:13-25.(in Persian)
32. Fatouros IG, Tournis S, Leontsini D, Jamurtas AZ, Sxina M, Thomakos P, e. Leptin and adiponectin responses in overweight inactive elderly following resistance training and detraining are intensity related. The Journal of Clinical Endocrinology & Metabolism. 2005;90(11):5970-7.
33. Monikh K, Kashef M, Azad A, Ghasemnian A. Effects of 6 weeks resistance training on Body Composition, serum Leptin and muscle strength in non-athletic men. Internal Medicine Today. 2015;21(2):135-40.(in Persian)
34. Hamedinia M, Haghighi A. Investigation of effect of one session moderate and heavy resistance exercise on acute and delayed responses of leptin, insulin, cortisol, testosterone and 24-hour energy expenditure in healthy men. Iranian Journal of Endocrinology and Metabolism. 2011;13(1):67-73.(in Persian)
35. Saremi A, Shavandi N, Bayat N. The effect of aerobic training on ghrelin and leptin serum levels and sleep quality in obese and overweight men. Journal of Arak University of Medical Sciences. 2012;15(1):52-60.(in Persian)
36. Asad MR, Ferdosi MH, Yoosefi Z. The effects of three training methods endurance, resistance and concurrent on adiponectin resting levels in overweighed untrained men. Procedia-Social and Behavioral Sciences. 2012;46:440-4.(in Persian)
37. Haghighi AH, Yarahmadi H, IldarAbadi A. The effect of green tea and aerobic exercise on serum adiponectin and ghrelin. medical journal of mashhad university of medical sciences. 2015;57(8):904-12.(in Persian)
38. Klimcakova E, Polak J, Moro C, Hejnova J, Majercik M, Viguerie N, et al. Dynamic strength training improves insulin sensitivity without altering plasma levels and gene expression of adipokines in subcutaneous adipose tissue in obese men. The Journal of Clinical Endocrinology & Metabolism. 2006;91(12):5107-12.
39. Mager U, Kolehmainen M, de Mello VD, Schwab U, Laaksonen DE, Rauramaa R, et al. Expression of ghrelin gene in peripheral blood mononuclear cells and plasma ghrelin concentrations in patients with metabolic syndrome. European journal of endocrinology. 2008;158(4):499-510.
40. Zhaosheng T, Li Y, Chengying G, Yun L, Lian Z. Effect of exercise on the expression of adiponectin mRNA and GLUT4 mRNA in type 2 diabetic rats. Journal of Huazhong University of Science and Technology [Medical Sciences]. 2005;25:191-3.
41. Wang CY, Lin TA, Liu KH, Liao CH, Liu YY, Wu VC, et al. Serum asprosin levels and bariatric surgery outcomes in obese adults. International journal of obesity. 2019;43(5):1019-25.
42. Duerrschmid C, He Y, Wang C, Li C, Bournat JC, Romere C, et al. Asprosin is a centrally acting orexigenic hormone. Nature medicine. 2017;23(12):1444-53.
43. Schumann U, Qiu S, Enders K, Bosnyák E, Laszlo R, Machus K, et al. Asprosin, A Newly Identified Fasting-Induced Hormone Is Not Elevated In Obesity And Is Insensitive To Acute Exercise: 3592 Board# 39 June 3 8: 00 AM-9: 30 AM. Medicine & science in sports & exercise. 2017;49(5S):1023:23-30.
44. Sohn JW. Network of hypothalamic neurons that control appetite. BMB reports. 2015;48(4):229.
45. Rezaeinezhad N, Ghanbari-Niaki A, Alizadeh R. The effect of circuit resistance training with different intensities on the agouti-related protein, insulin and glucose plasma levels in young men.Feyz Medical Science Journal. 2018;22(5):478-86.(in Persian)

[Downloaded from ijdo.ssu.ac.ir on 2026-11-25]

[DOI: 10.18502/ijdo.v15i4.14554]